List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
EPA Index Number	**Agency/Organization**	**First Name**	**Last Name**	**Division**	**Title/Role**	**Date submitted**
Federal and State Government (1-9)						
1	United States Senate	Kirsten	Gillibrand		United States Senator	6/7/2017
2	United States Senate	Charles	Schumer		United States Senator	7/18/2017
		Kristen	Gillibrand		United States Senator	
3	New York State Assembly	Didi	Barrett	106th District	Assemblymember	8/28/2017
4	New York State Assembly	Didi	Barrett	106th District	Assemblymember	6/28/2017
5	New York State Assembly	Ellen	Jaffee	97th District	Assemblymember	6/7/2017
6	New York State Senate	David	Carlucci	38th District	State Senator	8/30/2017
		Terrance	Murphy	40th District	State Senator	
		Martin J.	Golden	22nd District	State Senator	
		Marisol	Alcantara	31st District	State Senator	
		Jesse	Hamilton	20th District	State Senator	
7	New York State Senate	Brad	Hoylman	27th District	State Senator	8/9/2017
8	New York State Senate	Liz	Krueger	28th District	State Senator	7/19/2017
		Carrie	Woerner	113th District	Assemblymember	
		Joseph P.	Addabbo, Jr.	15th District	State Senator	
		Jamaal	Bailey	36th District	State Senator	
		Brian	Benjamin	30th District	State Senator	
		John E.	Brooks	8th District	State Senator	
		Leroy	Comrie	14th District	State Senator	
		Martin Malavé	Dilan	18th District	State Senator	
		George	Latimer	37th District	State Senator	
		Kevin S.	Parke	21st District	State Senator	
		José	Peralta	13th District	State Senator	
		Gustavo	Rivera	33rd District	State Senator	
		James	Sanders, Jr.	10th District	State Senator	

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
EPA Index Number	**Agency/Organization**	**First Name**	**Last Name**	**Division**	**Title/Role**	**Date submitted**
		José M.	Serrano	29th District	State Senator	
		Thomas J.	Abinanti	92nd District	Assemblymember	
		Didi	Barrett	106th District	Assemblymember	
		Kevin A.	Cahill	103rd District	Assemblymember	
		Jeffrey	Dinowitz	81st District	Assemblymember	
		Anthony	D'Urso	16th District	Assemblymember	
		Patricia	Fahy	109th District	Assemblymember	
		Sandra R.	Galef	95th District	Assemblymember	
		Deborah J.	Glick	66th District	Assemblymember	
		Richard N.	Gottfried	75th District	Assemblymember	
		Pamela J.	Hunter	128th District	Assemblymember	
		Ellen	Jaffee	97th District	Assemblymember	
		Brian P.	Kavanagh	74th District	Assemblymember	
		William	Magee	121st District	Assemblymember	
		Shelley	Mayer	90th District	Assemblymember	
		John T.	McDonald, III	108th District	Assemblymember	
		Yuh-Line	Niou	65th District	Assemblymember	
		Daniel	O'Donnell	69th District	Assemblymember	
		J. Gary	Pretlow	89th District	Assemblymember	
		Linda B.	Rosenthal	67th District	Assemblymember	
		Nily	Rozic	25th District	Assemblymember	
		Rebecca A	Seawright	76th District	Assemblymember	
		Jo Anne	Simon	52nd District	Assemblymember	
		Dan	Stec	114th District	Assemblymember	
		Fred W.	Thiele	1st District	Assemblymember	
		Mary Beth	Walsh	112th District	Assemblymember	
		Jaime R.	Williams	59th District	Assemblymember	

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
EPA Index Number	**Agency/Organization**	**First Name**	**Last Name**	**Division**	**Title/Role**	**Date submitted**
		Kenneth P.	Sebrowski	96th District	Assemblymember	
9	New York State Senate	José	Peralta	13th District	State Senator	8/31/2017
Agencies (10-16)						
10	National Oceanic and Atmospheric Administration	Thomas	Brosnan		Hudson River Case Manager	9/1/2017
11	National Oceanic and Atmospheric Administration	Jay	Field			9/1/2017
		Lisa	Rosman			
12	New York State Bridge Authority	Joseph	Ruggiero		Executive Director	6/28/2017
13	New York State Department of Environmental Conservation	Kevin	Farrar			9/1/2017
14	New York State Department of Environmental Conservation	Basil	Seggos		Commissioner	6/7/2017
15	New York State Department of Environmental Conservation	Basil	Seggos		Commissioner	8/30/2017
16	New York State Office of the Attorney General	Maureen	Leary		Assistant Attorney General	9/1/2017
		James	Wood		Assistant Attorney General	
		Brittany	Haner		Assistant Attorney General	
		John D.	Davis		Environmental Scientist	

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
EPA Index Number	**Agency/Organization**	**First Name**	**Last Name**	**Division**	**Title/Role**	**Date submitted**
Local Government (17-28)						
17	Albany County	Daniel	McCoy		County Executive	9/1/2017
	Rockland County	Edwin J.	Day		County Executive	
	Dutchess County	Marcus J.	Molinaro		County Executive	
	Ulster County	Michael P.	Hein		County Executive	
	Orange County	Steven M.	Neuhaus		County Executive	
	Westchester County	Robert P.	Astorino		County Executive	
18	Columbia County Environmental Management Council	Edwin	Simonsen		Chair	8/31/2017
19	Dutchess County	Marcus	Molinaro		Dutchess County Executive	6/28/2017
20	Dutchess County Regional Chamber of Commerce	Frank	Castella Jr.		President and CEO	8/16/2017
21	Kingston Conservation Advisory Council	Julie	Noble		Chair	8/31/2017
		Elizabeth	Broad			
		Lorraine	Farina			
		Emilie	Hauser			
		Lynn	Johnson			
		Kevin	McEvoy			
		Casey	Schwarz			
22	Town of Saratoga	Thomas N.	Wood III		Supervisor	8/21/2017
23	Town of Saugerties	Greg	Helsmoortel		Supervisor	8/29/2017

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
EPA Index Number	**Agency/Organization**	**First Name**	**Last Name**	**Division**	**Title/Role**	**Date submitted**
24	Town of Stuyvesant Town Board	Melissa	Naegeli		Town Clerk	8/10/2017
		Ed	Scott		Councilman	
		Tom	Burrall		Councilman	
		Brian	Chittenden		Councilman	
		Kelley	Williams		Councilwoman	
		Ron	Knott		Supervisor	
25	Ulster County Environmental Management Council	Dave	Haldeman		Chair	8/31/2017
26	Village of Schuylerville	Dan	Carpenter		Mayor	9/1/2017
27	Village of Schuylerville	Dan	Carpenter		Mayor	9/1/2017
28	Westchester County	Robert	Astorino		County Executive	8/28/2017
Organizations (29-43)						
29	Catskill Mountainkeeper	Kathleen	Nolan		Senior Research Director	9/1/2017
30	The Chamber of Southern Saratoga County **Signatories include organizations and businesses*					9/1/2017
	Mechanicville-Stillwater Chamber of Commerce	Barbara A.	Corsale		President	
	NYS Building and Construction Trades Council	James	Cahill		President	
	Dutchess County Regional Chamber of Commerce	Frank M.	Castella, Jr.		President & CEO	
	R. L. Baxter Building Corporation	Robert	Baxter		Owner	

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
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	Schuylerville Area Chamber of Commerce	Marla	Hodge		President	
	McDonald's REAAL, Inc.	Roger E.	Grout		President	
	The Chamber of Southern Saratoga	Pete	Bardunias		President & CEO	
	Elyse Harney Real Estate	Elyse D.	Harney		Principal Broker/Owner	
	Local Union 21	Ron	Diaz		Business Agent	
	Plumbers and Steamfitters HVACR	Thomas	Carey		Business Agent	
	Walkway Over the Hudson	Elizabeth	Waldstein-Hart		Executive Director	
	Poughkeepsie Alliance	Paul	Calogerakis		Chairman	
	Hudson Development Corporation	Sheena	Salvino		Executive Director	
	American Towns	Ted	Buerger		Chairman	
	Dutchess Community College	Pamela	Edington, Ed.D		President	
	The Business of Your Business	Wiley	Harrison		Owner	
	Rbeach & Bartolo Realtors	Victor	Mendolia		Associate Real Estate Broker	
	Finance& Corporate Development Omnicom Group	John	Hamilton		Vice President	
	Bryant Rabbino LLP	Kim	Taylor		Of Counsel	
	Saugerties Lighthouse	Patrick	Landewe		Keeper	
	Bonura Hospitality Group	Joe	Bonura		Principal	
	IKOR - Life Care Management Solutions	James	Sullivan		President & Managing Director	
	Consigli Construction, NY	Gregory	Burns		President	

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
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	Meyer Contracting Corporation	Christian W.	Meyer		President	
	Putnam Market	Catherine	Hamilton		President	
	Obercreek Farm LLC	Alex	Reese		Owner	
	Ugly Rooster Café	Ariel	Pagan		Owner	
	Northshire Bookstore	Chris	Morrow		Co-Owner	
	Five Porch Farms	Dan	Lundtquist		Owner	
	Healthy Living	Eli	Lesser-Goldsmith		Co-owner and General Manager	
	H H Hill Realty Services, Inc.	Harry	Hill		Principal Broker	
	National Resources, Inc.	Joseph	Cotter		CEO	
	Green Conscience Home & Garden	Karen	Totino		Licensed Real Estate Salesperson	
	Peak Magazine	Kellie	McGuire		Owner	
	Hudson River Cruises	Kevin	Buckel		General Manager	
	Spath Counseling Services	Kevin	Spath		Owner	
	Landscape Architects, P.C.	Kim	Mathews, RLA, FASLA		Principal	
	Kit Burke-Smith Jewelry	Kit	Burke-Smith		Owner	
	Storm King Adventure Tour	Kris	Seiz		Owner	
	Mohawk Maiden Cruises, LLC	Mara Hodge &	Maria Saavedra		Owners	
	Dutchess Tourism Inc.	Mary Kay	Verba		President & CEO	
	Bellefield Development Partners, LLC	Michael	Oates		Managing Partner	
	Fusion Lab, Inc.	Alon	Koppel		Partner	
	Jeffrey Russell Werner, LLC	Jeffrey Russel	Werner, Esq		Attorney	

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
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	Spatial Dynamics	Jaime	McMillian		Founder	
	Arts Center on Hudson	Jaime	McMillian		Founder	
	Growler and Grill	Mike	Fitzgerald		Owner	
	Saratoga Apple, Inc.	Nathan	Darrow		Owner	
	Gardening Angels	Peggy	Fusco		Owner	
	Alisson Spears AIA	Alison	Spears			
		Chip	Lowenson			
		Daniel	Kramer			
	Mary W. Harriman Foundation	David H.	Mortimer		President	
	David Redden, LLC.	David	Redden		Director	
	Deco Works Ltd.	Evan Mason and	Garrard Beeney		Principals	
		Gary	Glynn			
		Hoke	Slaughter			
		Jay	Saunders			
		James	Goodfellow			
		Julia	Widowson			
		Kristin	Flood			
		Leigh	Seippel			
	United Catalyst, LLC.	Marjorie Hart	Acting CEO			
	Land Trust Alliance	Michael P.	Dowling		Immediate Past Chair	
	Dillion, Ready & Co, Inc.	Ned	Whitney		Retired Managing Director	
		Richard	Klapper			
	Pierpoint Capital	Richard	Krupp		Managing Partner	

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
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	Debevoise & Plimpton LLP	Sara A. Q.	Fitts			
31	Hudson River Fishermen's Association	Gil	Hawkins		Vice President	6/15/2017
32	Riverkeeper, Inc.	Jeremy	Cherson		Campaign Advocacy Coordinator	7/7/17
33	Riverkeeper, Inc.	Jeremy	Cherson		Campaign Advocacy Coordinator	7/19/17
34	Riverkeeper, Inc.	Richard	Webster, Esq			6/5/2017
	Hudson Fishermen's Association	Gil	Hawkins			
	Natural Resources Defense Council	Daniel	Raichel, Esq			
	Scenic Hudson, Inc.	Althea	Mullarkey			
	Hudson River Sloop Clearwater, Inc.	Manna Jo	Greene			
35	Riverkeeper, Inc.	Richard	Webster		Legal Director	6/16/2017
36	Saratoga Unites Environmental Action Committee	Julie	Wash			8/31/2017
37	Scenic Hudson, Inc.	Hayley	Carlock		Director of Environmental Advocacy	9/1/2017
	Hudson River Fishermen's Association	Gil	Hawkins			
	Riverkeeper, Inc.	Richard	Webster, Esq			
	Hudson River Sloop Clearwater, Inc.	Manna Jo	Green			
	Sierra Club, Atlantic Chapter	Roger	Downs			

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
EPA Index Number	**Agency/Organization**	**First Name**	**Last Name**	**Division**	**Title/Role**	**Date submitted**
	Natural Resources Defense Council	Mark	Izeman			
38	Scenic Hudson, Inc.	Hayley	Carlock		Director of Environmental Advocacy	9/1/2017
	Hudson River Fishermen's Association	Gil	Hawkins			
	Riverkeeper, Inc.	Richard	Webster, Esq			
	Hudson River Sloop Clearwater, Inc.	Manna Jo	Green			
	Sierra Club, Atlantic Chapter	Roger	Downs			
	Natural Resources Defense Council	Mark	Izeman			
39	Society of Saint Ursula	Kathleen	Donnelly			8/15/2017
40	The Historic Hudson - Hoosic Rivers Partnership	Tom	Richardson		Partnership Chairperson	8/31/2017
41	Walkway Over the Hudson	Elizabeth	Waldstein-Hart		Executive Director	8/31/2017
42	Hudson River Sloop Clearwater, Inc. Petition * Petition with 503 signatures					8/22/2017
43	Hudson River Sloop Clearwater, Inc. Petition *Petition with 150 signatures					8/28/2017

List of Commenters on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site: Government, Agencies, Organizations and Businesses/Corporations						
EPA Index Number	**Agency/Organization**	**First Name**	**Last Name**	**Division**	**Title/Role**	**Date submitted**
Businesses/Corporations (44-51)						
44	Bonura Hospitality Group	Joseph	Bonura Jr.		Owner	8/1/2017
45	ecoSPEARS	Ian	Doromal		Vice President	9/1/2017
46	General Electric	John	Haggard	Global Remediation; Global Operations, Environmental, Health & Safety	Leader	9/1/2017
47	Hudson Development Corporation	Sheena	Salvino		Executive Director	8/31/2017
48	Mohawk Maiden Cruises	Marla	Hodge		Master Captain, Owner	8/30/2017
49	Seaweed Yacht Club; Hudson River Boat & Yacht Club Association	Janice	Anderson		Commodore; Director	8/28/2017
50	The Business of your Business	Wiley	Harrison		Owner	8/7/2017
51	United Campus Holdings Company, LLC	Wayne	Senecal		President and CEO Emeritus	7/19/2017

KIRSTEN GILLIBRAND
NEW YORK
SENATOR

RUSSELL SENATE OFFICE BUILDING
SUITE 478
WASHINGTON, DC 20510–3205
202–224–4451

COMMITTEES:
ARMED SERVICES
ENVIRONMENT AND PUBLIC WORKS
AGRICULTURE
SPECIAL COMMITTEE ON AGING

United States Senate

WASHINGTON, DC 20510–3205

June 7, 2017

The Honorable Scott Pruitt
Secretary
Environmental Protection Agency
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Dear Administrator Pruitt:

I write to request a 90 day extension to the public comment period regarding EPA's second five year review of the Hudson River PCBs Superfund Site. The report is more than 1000 pages, and includes detailed technical data and assessments. Its findings have ramifications for stakeholders along a 200-mile stretch of the Hudson River from Hudson Falls, New York to New York City. Therefore, I do not believe that EPA's 30 day comment period is sufficient. It is vital that local residents, community and environmental organizations, business leaders, state and federal agencies, as well as the Hudson River Natural Resource Trustees, and representatives from the Community Advisory Group have the opportunity to review and evaluate the results and make their voices heard.

The five year review presents an opportunity to realize goals that you have articulated, including the importance of cleaning up the Hudson River pollution, ensuring the Superfund program succeeds in achieving both environmental outcomes and creating jobs. When EPA announced the Hudson River cleanup 15 years ago, it was a promise to New Yorkers that the long-damaged river would finally be on the path to a rapid recovery. However, after the cleanup plan was established, EPA discovered that at least 2-3 times more PCB contamination existed in Hudson River sediments than had been assumed; yet EPA did not modify the scope of the cleanup. As a result, the Hudson River remains contaminated at levels far beyond the cleanup targets EPA established. Economic development on the Upper Hudson River has long been stifled by the dark cloud of toxic pollution; communities cannot wait decades longer for a clean and usable river. Long-term "natural attenuation" of PCBs is not a solution to this problem. The PCBs in the Upper Hudson River are continuing to be transported down-river as far as New York Harbor and beyond. PCB levels in fish in the lower Hudson River are not declining as expected, pointing to the need for investigation of downriver contamination and appropriate remedial action.

New York State has a long and proud history of environmental protection in conjunction with economic development, and the Hudson River is a national symbol as an American Heritage River. New Yorkers live, work and play along the Hudson River. I strongly believe that additional cleanup is needed. A credible five year review is crucial to ensure the integrity of the federal Superfund program, given that the Hudson is one of the largest and most visible sites in the country.

It is essential that those who are most directly impacted have a sufficient opportunity to review and respond to the EPA report. I urge you to extend the public comment period for this important purpose.

Sincerely,

Kirsten Gillibrand

Kirsten Gillibrand
United States Senator

United States Senate
WASHINGTON, DC 20510

July 18, 2017

Administrator Scott Pruitt
Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Dear Administrator Pruitt:

We write to urge the Environmental Protection Agency (EPA) to hold a public information meeting in New York City regarding the Proposed Second Five-Year Review Report for the Hudson River Superfund Site. It is crucial that the local community, including those along the Lower Hudson River, have the opportunity to hear directly from the EPA on this proposed report and to have their own voices be heard.

We appreciate that the EPA has already held a public information meeting in Poughkeepsie, and has another meeting planned in Saratoga Springs on July 19. However, we believe it is important that the EPA hold an additional meeting in New York City, as well, to give the maximum number of New Yorkers who live adjacent to the Hudson River an opportunity to participate in the public comment process. There is a significant population of residents in New York City who may have an interest in the status of the cleanup of the Hudson River PCBs Superfund Site and they should have maximum ability to learn about the status of this endeavor and to offer their views on current and future plans in this regard. With Poughkeepsie located more than 80 miles away from New York City, and Saratoga Springs more than 180 miles away, EPA should do more to ensure that potentially interested communities have the opportunity to hear directly from the agency and participate in the public comment process, particularly since some elderly, disabled, low-income, and other residents who have hectic schedules, yet who wish to engage in the process, may have a difficult time attending a meeting so far from home. It is for this reason that we urge the EPA to hold a public information meeting in New York City on this Proposed Second Five-Year Review Report as soon as possible.

Thank you in advance for your consideration of this request. Should you have any additional questions, please do not hesitate to contact our offices.

Sincerely,

Charles E. Schumer
United States Senator

Kirsten Gillibrand
United States Senator

Public Comment for Submission: Hudson River PCBs Superfund Site Second Five-Year Review

Richard Stein <steinr@nyassembly.gov>

Mon 8/28/2017 4:45 PM

To: epahrfo@outlook.com <epahrfo@outlook.com>;

1 attachments (118 KB)

EPA Five-Year Review of PCBs Public Comment.pdf;

Dear Mr. Gary Klawinksi:

Please see the attached letter to be submitted for public comment from State Assemblymember Didi Barrett regarding the Hudson River PCB Superfund Site Second Five-Year Review. Thank you.

Kind Regards,
Rich Stein
--

Rich Stein
Chief of Staff
NYS Assemblymember Didi Barrett
12 Raymond Ave, Ste. 105
Arlington N.Y. 12603
(o) 845.454.1703
(c) 914.384.9680
(f)845.454.2408

Didi Barrett
Assemblymember 106th District
Columbia County
Dutchess County

THE ASSEMBLY
STATE OF NEW YORK
ALBANY

CHAIR
Task Force on People with Disabilities

COMMITTEES
Agriculture
Economic Development, Job Creation, Commerce and Industry
Environmental Conservation
Mental Health
Tourism, Parks, Arts and Sports Development
Veterans' Affairs

August 23, 2017

Gary Klawinski, Director
EPA Region 2, Hudson River Office
187 Wolf Road, Suite 303
Albany, NY 12205
epahrfo@outlook.com

Re: Hudson River PCBs Superfund Site Second Five-Year Review

Dear Director Klawinski,

On behalf of the people of the 106th Assembly District of New York, and as a resident of the beautiful Hudson River Valley, I write to urge the EPA to continue active remediation efforts in the Upper Hudson as well to investigate PCBs in the Mid and Lower Hudson. The Hudson River, often called America's River, is a vital economic, recreational, and cultural resource for millions of people. It is the historic spine of our region and a critical source of water for communities and businesses on its shores. The EPA must do more to ensure the remediation will protect human health and the environment, and meet the goals from its original Record of Decision (ROD) in 2002.

Analysis from National Oceanic and the Atmospheric Administration (NOAA) and the US Fish and Wildlife Service (USFWS) suggests pre-remedial PCB concentrations in areas targeted for dredging were 2 to 3 times higher than EPA anticipated. Higher than anticipated levels of surface sediment contamination was found in portions of River Sections 2 & 3, areas not designated for dredging. Data also suggests the lower 150 miles of the river is not responding to the dredging as anticipated, and PCB concentrations in fish have not declined. According to the EPA's own analysis, it will be, at minimum, over 50 years before New Yorkers can safely eat one half-pound fish meal from the Hudson River once a week without negative health effects. Our communities should not have to wait generations to safely eat locally caught fish.

General Electric fought for decades and spent millions to avoid cleaning the over 1.3 million pounds of PCBs it dumped into the Hudson River. We are asking the EPA to stand up for the people that live by, work on, and visit the majestic Hudson River, and tell GE to take responsibility for the tremendous damage it has done to the river and the local economy. The

EPA must require GE to achieve “protective” status for the entire 200-mile stretch of the Hudson River ensuring that PCB levels are truly safe for humans and the environment.

I hope the EPA will consider the people of New York when makings its decision about our River’s vitality.

Sincerely,

Didi Barrett
Assemblymember, 106th District

cc:
Administrator Scott Pruitt, U.S. Environmental Protection Agency
Commissioner Basil Seggos, NYS Department of Environmental Conservation

Didi Barrett
Assemblymember 106th District
Columbia County
Dutchess County

THE ASSEMBLY
STATE OF NEW YORK
ALBANY

CHAIR
Task Force on People with Disabilities

COMMITTEES
Agriculture
Economic Development, Job Creation, Commerce and Industry
Environmental Conservation
Mental Health
Tourism, Parks, Arts and Sports Development
Veterans' Affair

June 28, 2017

Gary Klawinski
Director
US EPA Region 2
290 Broadway
New York, New York 10007-1866

RE: Hudson River PCB Dredging Five Year Review

Dear Director Klawinski,

I have the privilege of representing the beautiful 106th Assembly District, which includes twenty municipalities in Columbia and Dutchess Counties; seven of these are riverfront communities.

The Hudson River is the lifeline of this region: It connects us economically, culturally, and historically. I believe the EPA's decision to discontinue the dredging of PCBs is premature and will leave the region with a contaminated river for decades to come. The fact is the river is still contaminated. Data presented by the National Oceanic and Atmospheric Administration, the New York State Department of Health and the New York State Environmental Protection Agency all say the river is not yet safe for the health of humans, wildlife and the environment.

The Hudson River Valley is home to a stunning array of natural treasures, a robust tourism industry, countless historic landmarks, the landscapes of America's first artistic movement and thousands of residents and businesses that depend upon the iconic Hudson, America's River. A safe and healthy river is within our reach. I urge you not to stop only part way to that goal. Please keep dredging the river until all of the PCBs are removed.

Thank you for your consideration. Please feel free to reach out to my office if you have any questions or concerns.

Sincerely,

Didi Barrett

Didi Barrett
Member of Assembly, 106th District

DISTRICT OFFICES: 12 Raymond Avenue, Suite 105, Poughkeepsie, New York 12603 • 845-454-1703, FAX: 845-454-2408
751 Warren Street, Hudson, New York 12534 • 518-828-1961, FAX: 518-828-5329
ALBANY OFFICE: Room 553, Legislative Office Building, Albany, NY 12248 • 518-455-5177, FAX: 518-455-5118
EMAIL: barretd@nyassembly.gov

Protect people and wildlife, not GE

Ellen Jaffee <info@Riverkeeper.org>

Fri 9/1/2017 11:47 AM

To: Gary Klawinski, Project Director, EPA <epahrfo@outlook.com>;

Sep 1, 2017

Mr. Gary Klawinski, Project Director, EPA
US EPA Hudson River Field Office, Region 2, 187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski, Project Director, EPA,

September 1, 2017

Administrator E. Scott 'Scott' Pruitt
Mr. Gary Klawinski, Project Director, EPA

RE: U.S. Environmental Protection Agency's (EPA's) Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site.

Dear Administrator Pruitt and Mr. Klawinski:

As a member of the New York State Assembly and both the Assembly Environmental Conservation and Health Committees, I am writing on behalf of the residents of Rockland County, NY, my constituents in the 97th Assembly District, and the communities I represent, including the Hudson River Villages of Nyack, South Nyack, Piermont, Grandview-on-Hudson, Upper Grandview, and the Hamlet of Palisades, to express my serious concerns regarding the U.S. Environmental Protection Agency's (EPA's) Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site.

The Hudson River is a critical resource vital to the quality of life in our communities. The PCB cleanup is not performing as planned and thus the remedy is neither protective of human health nor the environment. Consequently, I strongly urge you to order further dredging in the Upper Hudson River, and to order a full remedial investigation and feasibility study in the Lower Hudson River as soon as possible.

EPA's review must clearly state "the remedy is not protective." In the report you admit that General Electric's (GE's) cleanup of toxic PCBs it dumped into the Hudson River does not currently protect the health of the public or the river. That should be the only finding of the report. And you must remove the phrase "the remedy will be protective." Such a statement conflicts with your agency's admissions that the cleanup is not protective now, that at least eight more years of data are needed to predict future trends with any confidence, that the short-term five-year fish tissue goal will not be met, and that more investigation is needed in the

lower 150 miles of the Hudson River.

The economic, recreational, cultural and scenic value of the Hudson River form the bedrock of past development as well as the future vitality of the Hudson Valley and New York City. General Electric's negligent dumping of more than a million pounds of toxic PCBs into the Hudson River for more than 30 years resulted in a once vibrant commercial fishing industry being shut down. Today, the Hudson River is one of the nation's largest Superfund sites, and the consumption of fish from the River has been significantly restricted. As demonstrated by the public outcry at EPA's information meetings on its Five-Year Review Report, and at a time when many of our communities have long-term waterfront revitalization goals that will increase tourism, create jobs, and boost our local economies, New Yorkers want a healthy Hudson River as soon as possible.

The goals set forth by EPA to clean up the Hudson River are already weak. In the Upper Hudson River--the 40 miles north of the Federal Dam in Troy, NY--EPA expected that within five years of the completion of dredging, it would only be safe to eat one fish meal every two months, and that within 16 years, it would only be safe to eat one fish meal per month. Under the cleanup plan, EPA did not expect people to be able to eat one fish meal per week for over 55 years. Because the timelines for the cleanup are so long, I expect EPA to hold GE accountable for meeting not moving the goal posts. In the meantime, I am concerned about the many people who eat fish from the Hudson River, and I urge EPA to do better outreach to subsistence and recreational fishing communities about the health risks involved in doing so.

EPA's determination that the cleanup "will be protective" of human health and the environment of the Upper Hudson River is unacceptable. This determination is inconsistent with the agency's admission that the cleanup is currently not protective and with EPA's repeated statements that at least eight more years of data are needed to predict future trends with any confidence.

EPA's determination is further undercut by the agency's reluctance to provide specific timeframes for reaching the short- and long-term goals. In addition, the National Oceanic and Atmospheric Administration (NOAA) recently published a peer-reviewed study suggesting that hazardous levels of PCBs will remain in fish in the Lower Hudson River for much longer than the EPA predicts. The New York State Department of Environmental Conservation (NYSDEC) has also expressed its concerns with the findings in the report, stating that the significant amount of contamination left in the river threatens both public health and the environment. Therefore, EPA should revise its determination and recognize that the cleanup is not protective of human health and the environment.

The data show that the Lower Hudson River--the 150 miles south of the Federal Dam--is not responding as anticipated. EPA essentially admits that the cleanup is not working in the Lower Hudson River by failing to make a protectiveness determination that covers this stretch. From Poughkeepsie and continuing downstream, the decay rates (or rate of decrease in PCB concentration) in fish are not statistically different from zero. NYSDEC and the Hudson River Foundation do not expect the dredging to result in additional improvement in the Lower Hudson River.

While EPA agrees that more investigation is needed, the agency has made no definite plans on how this will be done. Therefore, I urge EPA to require GE to do a full remedial investigation and feasibility study of the Lower Hudson River.

EPA should be transparent regarding the facts in its Five-Year Review Report. For instance, during Phase 1 of dredging, EPA discovered that it had underestimated both the depth of the PCB contamination and the concentration of PCBs in the surface sediment. Despite acknowledging that there were more PCBs present, EPA did not change the goals for the cleanup. Instead, EPA focused on removing a certain percentage of contaminated sediment, leaving behind two to three times more PCBs than anticipated. NOAA has stated that this means that cleanup goal targets will be met up to 60 years later than expected. The public has a right to know how much PCB contamination remains in the Hudson River today, and I urge EPA to make that information clear and accessible in its final report.

In short, for the Upper Hudson River, EPA has failed to evaluate all of the signs that the cleanup will not meet its goals, and instead has made a determination that is not grounded in science. For the Lower Hudson River, EPA has recognized that the cleanup is not working as anticipated, but it has failed to provide a plan for a prompt investigation and cleanup. If Administrator Pruitt's words about implementing Superfund better and faster mean anything at all, they should cause EPA to make a "not protective" finding for the entire Hudson River Superfund Site, order GE to take more PCBs out of the Upper Hudson River, and compel GE to devise a real cleanup of the Lower Hudson River.

Respectfully submitted,

Ellen C. Jaffee
Member of Assembly,
97 (AD)

Sincerely,

Ms. Ellen Jaffee
1 Blue Hill Plz
Pearl River, NY 10965-3104
(845) 624-4601
jaffeee@nyassembly.gov

Fwd: Letter Re Hudson River PCBs Superfund Site

Romanowski, Larisa <Romanowski.Larisa@epa.gov>

Wed 8/30/2017 3:16 PM

To: Klawinski, Gary J <Klawinski.Gary@epa.gov>; epahrfo@outlook.com <epahrfo@outlook.com>;

2 attachments (202 KB)

Letter to EPA Re Hudson River PCB Superfund Site.pdf; ATT00001.htm;

Sent from my iPhone

Begin forwarded message:

From: Oscar Dunham <odunham@nysenate.gov>
Date: August 30, 2017 at 1:26:29 PM EDT
To: Romanowski Larisa <Romanowski.Larisa@epa.gov>
Cc: <kevin.farrar@dec.ny.gov>, <wendy.rosenbach@dec.ny.gov>
Subject: Letter Re Hudson River PCBs Superfund Site

Hey Larisa,

In response to the release of Hudson River Superfund site: Second Five Year Review Report, Senator Carlucci would like to submit the attached letter to the EPA signed by other senators whose districts border the Lower Region of the Hudson River Superfund site.

Oscar Dunham
Director of Operations
The Office of New York State Senator David Carlucci (SD-38)
(Rockland Office) 845-623-3627
(Albany Office) 518-455-2991

THE SENATE
STATE OF NEW YORK
ALBANY, NY 12247

August 28, 2017

The Honorable Scott Pruitt, Administrator
U.S. Environmental Protection Agency
Ariel Rios Building
1200 Pennsylvania Avenue NW
Washington, DC 20460

Re: Hudson River PCBs Superfund Site Second Five-Year Review Report

Dear Administrator Pruitt,

As members of the New York State Senate delegation whose districts border the shoreline of the Hudson River PCBs Superfund site, we are writing you today to express our concerns over the findings stated in the U.S. Environmental Protection Agency's (EPA) Hudson River Superfund site: Second Five Year Review report released on June 1, 2017.

The Hudson River, specifically the Lower Region, is home to millions of New Yorkers who reside along the shoreline and is a keystone of the Hudson Valley's $5.2-billion tourism economy responsible for more than tens of thousands of jobs in the region as well. Moreover, plans for future economic development along the Hudson River and its shoreline have been developed by state and local agencies – including the renewal of the once vibrant commercial fishing industry.

While the Second Five Year Review report does acknowledge the need for more investigation in the Lower Hudson River, the conclusion of the report clearly ignores the failure of the cleanup to meet health and ecological targets established by the EPA when it selected the dredging project for the Upper Hudson River. The report states that PCB levels in the Lower Hudson River, between the Troy Dam and the tip of Manhattan, have not demonstrated expected declines resulting from Upper Hudson River dredging. Consequently, it is abundantly clear this Superfund project has not gone far enough – empirical evidence demonstrates this.

Furthermore, two other federal agencies, the National Oceanic and Atmospheric Administration (NOAA) and the U.S. Fish and Wildlife Service (USFWS), recently published a peer-reviewed study suggesting hazardous levels of PCBs will remain in fish in the Lower Hudson River for much longer than the EPA predicts. The New York State Department of Environmental Conservation (NYSDEC) has expressed their concerns with the findings in the report, stating that significant amount of contamination left in the river threatens both the public health and the environment. Even more disconcerting, other EPA officials have confirmed that not until fifty-three years from now, people would be able to eat a fish-meal from the Hudson River without facing serious health concerns.

Without the EPA taking additional remediation steps in the Lower Hudson River, restoring the health and well-being of people subsisting on the river's polluted fish and developing job-creating riverfront revitalization projects will be suspended well into the 22nd century. If the EPA fails to take action, New York taxpayers will be forced to foot the bill to clean up a mess they did not create. Neither option is acceptable.

Therefore, we respectfully request for EPA to direct General Electric (GE) to develop an adequate plan of action for the additional removal of PCB-contaminated sediment in the Lower Hudson River to meet human health and ecological satisfactory levels in a timely manner. Additionally, we further request for EPA, in coordination with other agencies, to provide more effective outreach regarding fish consumption advisories along the Lower Hudson River shoreline.

Thank you in advance for your time and attention to this matter.

Sincerely,

Senator David Carlucci
38th Senate District

Senator Marisol Alcantara
31st Senate District

Senator Terrence Murphy
40th Senate District

Senator Jesse Hamilton
20th Senate District

Senator Martin J. Golden
22nd Senate District

Cc:

Gary Klawinski, Director
EPA Region 2, Hudson River Office

Kevin L. Farrar
Section Chief, Section A/Bureau D, Division of Environmental Remediation
New York State Department of Environmental Conservation (NYSDEC)

Comments from Senator Hoylman on Hudson River PCBs

Tara Klein <tara@bradhoylman.com>

Thu 8/10/2017 10:50 AM

To:epahrfo@outlook.com <epahrfo@outlook.com>;

1 attachments (98 KB)

Comments to EPA on Second Five Year Review on Hudson River PCB Cleanup.pdf;

Hello,

Attached are comments on the EPA's Second Five-Year Review Report for the Hudson River PCBs Superfund site from New York State Senators Brad Hoylman and Daniel Squadron. A hard copy was also submitted at last night's public hearing at John Jay College.

Thank you.

--
Tara Klein
Deputy Policy Director
New York State Senator Brad Hoylman
322 Eighth Avenue, Suite 1700
New York, NY 10001
(212) 633-8052
tara@bradhoylman.com
NY Senate | Twitter | Facebook

August 9, 2017

Gary Klawinski, Director
EPA Region 2, Hudson River Office
187 Wolf Road, Suite 303
Albany, NY 12205

Re: Comments on EPA's Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site

Dear Director Klawinski:

Thank you for the opportunity to submit comments on the Environmental Protection Agency's (EPA) Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund site. As representatives of Senate Districts that abut the Hudson River in Manhattan, we write to express our deep concerns that the preliminary review does not recommend more cleanup of the river. We urge the EPA to revisit the terms of its 2005 settlement agreement with General Electric (GE) in light of widespread evidence that calls into question the efficacy of the agreement's remedy.

For more than three decades, General Electric knowingly dumped millions of pounds of toxic polychlorinated biphenyls, or PCBs, into the Hudson River. As a direct result of PCB contamination – which has been linked to cancer, low birth weight, thyroid disease, and learning, memory, and immune system disorders – the EPA classified parts of the Hudson as a Superfund site and mandated that GE remediate the river through dredging. After GE ostensibly met the terms of the settlement agreement, EPA allowed the company to begin dismantling its dredging equipment in November 2015. However, subsequent reports indicate that GE removed only 65% to 72% of PCBs.[1]

Unfortunately, the Hudson River is far from remediated, and recent studies suggest the extent of the damage may be worse than originally thought. The three trustees of the Hudson River – including the National Oceanic and Atmospheric Administration (NOAA), the U.S. Fish and Wildlife Service (FWS), and the New York State Department of Environmental Conservation (DEC) – as well as numerous environmental watchdog organizations and even the EPA's own analysis have all found evidence that the cleanup is not complete.

[1] Nearing, Brian, "EPA says Hudson PCB cleanup shows improvement, more study still needed." Times Union, June 1, 2017: http://www.timesunion.com/allwcm/article/EPA-says-Hudson-PCB-cleanup-shows-improvement-11188727.php

In November 2015, FWS and NOAA released a statement on the Hudson River Demobilization Plan to dismantle dredging equipment, stating: "Although the dredging has removed some of the contamination, it has not addressed nor compensated the public for injuries to natural resources. Trustees have determined that GE's PCBs have injured groundwater, fish, waterfowl, surface water, and navigational services of the Hudson River for decades, and these injuries will continue well into the future."[2]

In May of 2015, NOAA issued a report[3] examining the model projections used as the basis of the 2005 agreement between EPA and GE, finding that the "original models used were overly optimistic" and overestimated the rate of natural recovery in the Hudson River. As a result, achieving the EPA's remedial objectives "will take longer than predicted." Ultimately, NOAA concluded, "[a]dditional removal of PCB-contaminated sediment in the Upper Hudson River [is] needed to achieve [the] reductions in Lower Hudson River fish PCBs" that were initially anticipated by the EPA.

The same report found that the amount of PCBs remaining in the Upper Hudson River after GE leaves is likely to be three to five times higher than EPA initially forecast, and the rate of recovery of the river is expected to take generations longer than predicted — an additional 40 to 50 years. In other words, because of the flawed modeling used by the EPA over a decade ago, if GE's federally-mandated dredging is allowed to conclude the EPA will have fallen far short in achieving its intended remediation.

As the state agency that serves as a trustee to New York's Hudson River, DEC is uniquely positioned to understand the local impact and effectiveness of the cleanup. In December 2016, DEC released an independent review of PCB contamination in the Hudson River, informed by EPA guidelines and criteria, and shared its findings with the EPA. DEC's recommendations found that "despite the substantial remedial work done in constructing the dredging remedy between 2009 and 2015, the risks to human health and the environment are well above the EPA acceptable risk range, and … unacceptable exposures are still occurring." The report recommended the EPA conduct additional studies on the effectiveness of the remedy, update its data and scope to optimize the remedy, and expand its site investigation to the Lower Hudson between the Federal Dam at Troy and the Battery in Manhattan.[4]

The EPA itself has said it will take 55 years before it is safe to eat Hudson River fish once per week.[5] While certainly the remedy will not be immediate, this time frame is

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[2] Statement on Hudson River Demobilization Plan by U.S. Fish & Wildlife Service and NOAA: https://www.fws.gov/northeast/ecologicalservices/HudsonRiver/docs/Statement%2011_12_15.pdf
[3] NOAA report: http://www.hudsonriver.org/download/seminars/HRF_Field.pdf
[4] DEC report: http://www.dec.ny.gov/docs/fish_marine_pdf/hudsondredging5yr.pdf
[5] Hill, Michael, "Q&A: Controversy lingers after $1.7B cleanup of Hudson River." AP, June 27, 2017. http://poststar.com/news/state-and-regional/q-a-controversy-lingers-after-b-cleanup-of-hudson-river/article_548495e4-73d5-597f-ba3e-2efdccfb223c.html

concerning, especially because the EPA's Second Five-Year Review Fact Sheet from June 2017 states, "The EPA's five-year review acknowledges that more years of post-dredging data are needed to identify, with a higher degree of confidence, long-term trends in the river's recovery."[6] The lack of clear data about trends impacting the river's long-term recovery, paired with warnings being raised from the river's trustees, should be sufficient justification to revisit the terms of the settlement agreement.

Environmental advocacy organizations, including Riverkeeper, Scenic Hudson, NRDC, Sierra Club Atlantic Chapter, Hudson River Sloop Clearwater and many others have highlighted these widespread expert opinions on the cleanup and concur that more work needs to be done.

On the basis of the foregoing, we once again strongly urge the EPA to revisit the terms of the 2005 agreement to ensure that GE completes a thorough and effective cleanup of Hudson River PCBs.

Sincerely,

Brad Hoylman
New York State Senator
27th District
322 8th Avenue, Suite 1700
New York, NY 10001

Daniel Squadron
New York State Senator
26th District
250 Broadway, Suite 2011
New York, NY 10007

[6] Hudson River PCBs Superfund Site Second Five-Year Review Fact Sheet: https://www.epa.gov/sites/production/files/2017-06/documents/final_hudsonriver_2ndfyr_factsheet_june2017.pdf

FW: Letter from NYS Legislators RE Hudson Superfund Site Five-Year Review

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Tue 8/15/2017 2:57 PM

To: Public Comment Hudson 2nd FYR (epahrfo@outlook.com) <epahrfo@outlook.com>;

Cc: Romanowski, Larisa <Romanowski.Larisa@epa.gov>;

1 attachments (484 KB)

NYS Legislators Letter RE Hudson PCB Superfund.pdf;

From: Justin Flagg [mailto:flagg@nysenate.gov]
Sent: Wednesday, July 19, 2017 10:28 AM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>
Cc: Pruitt, Scott <Pruitt.Scott@epa.gov>
Subject: Letter from NYS Legislators RE Hudson Superfund Site Five-Year Review

Dear Director Klawinski,

Please accept the attached letter as comments from 41 New York State legislators regarding EPA's recently released Hudson River PCBs Superfund Site Second Five-Year Review. If you have any questions or concerns you can reach me at this email address or at 212-490-9535,

Sincerely,

Justin Flagg
--
State Senator Liz Krueger
212-490-9535

July 19th, 2017

Gary Klawinski, Director
EPA Region 2, Hudson River Office
187 Wolf Road, Suite 303
Albany, NY 12205

Re: Hudson River PCBs Superfund Site Second Five-Year Review

Dear Director Klawinski,

We write to you as the elected representatives of millions of New Yorkers living along the banks of the Hudson River and beyond. Our constituents look to the Hudson as a vital economic, recreational, and cultural resource, part of the heritage of all New Yorkers and all Americans. As stewards of that heritage, we urge you to revise the second Five-Year Review of the Hudson River PCBs Superfund Site to declare that cleanup efforts completed thus far are **not protective of human health and additional remediation and evaluation is required** to restore the river and meet the goals set out in the Record of Decision.

Although dredging of PCB-laced sediment in the Upper Hudson was effective at removing PCBs from the riverbed, data has shown that two to three times as many PCBs remain in the river as expected. Additionally, PCB levels in the Lower Hudson, between the Troy Dam and the tip of Manhattan, have not demonstrated expected declines resulting from Upper Hudson dredging. According to EPA's own analysis, it will be over 50 years before it will be safe for New Yorkers and visitors to eat fish from the Hudson *once a week* without negative health effects. Two of EPA's sister agencies, NOAA and USFWS, have issued a study suggesting natural attenuation will take even longer.

It is simply unacceptable that New Yorkers may have to wait decades, and possibly several generations, before the Hudson River can be considered safe as a result of natural attenuation. Unless further action is taken, many of us, and even some of our children, will not live to see that time. General Electric alone is responsible for causing this

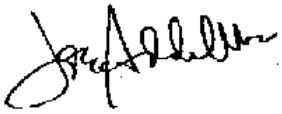
extensive damage to the river, its people, and its economic productivity. Now it is up to the EPA to require GE to do whatever is necessary to make the river whole. The first step is for the Five-Year Review to reflect the reality that the cleanup has not achieved its objectives, and require further dredging of the Upper Hudson and further investigation of the Lower Hudson.

Thank you for the opportunity to comment, and for your serious consideration of our concerns.

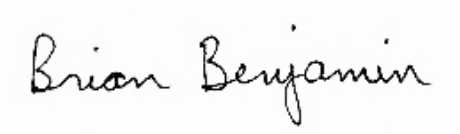
Sincerely,

Liz Krueger
State Senator

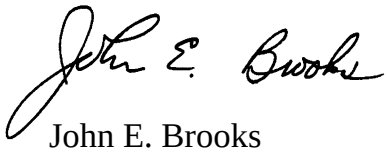
Carrie Woerner
Assembly Member



Joseph P. Addabbo, Jr.
State Senator



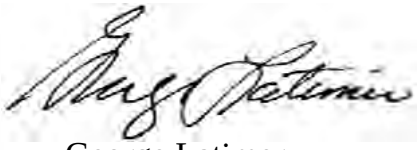
Brian Benjamin
State Senator



John E. Brooks
State Senator

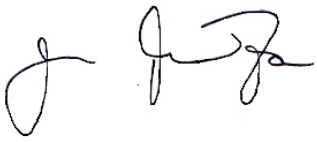
Leroy Comrie
State Senator

Martin Malavé Dilan
State Senator



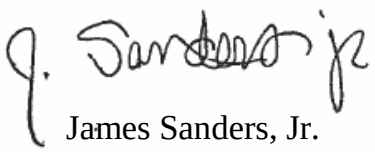
George Latimer
State Senator

Kevin S. Parker
State Senator

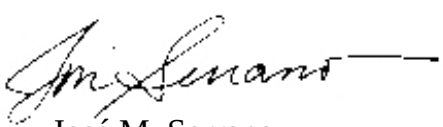


Jose Peralta
State Senator

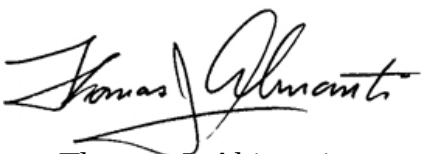
Gustavo Rivera
State Senator



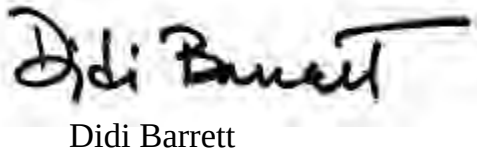
James Sanders, Jr.
State Senator



José M. Serrano
State Senator

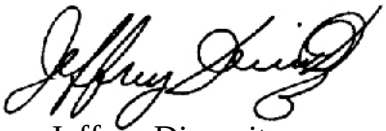


Thomas J. Abinanti
Assemblymember

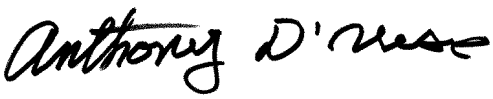


Didi Barrett
Assemblymember

Kevin A. Cahill
Assemblymember

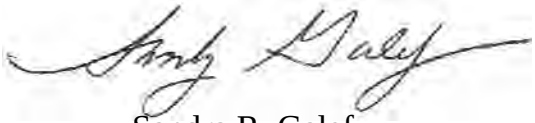


Jeffrey Dinowitz
Assemblymember

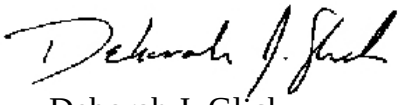


Anthony D'Urso
Assemblymember

Patricia Fahy
Assemblymember



Sandra R. Galef
Assemblymember



Deborah J. Glick
Assemblymember

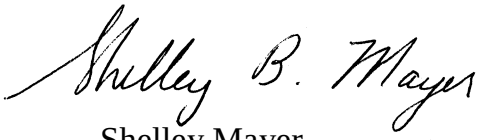
Richard N. Gottfried
Assemblymember

Pamela J. Hunter
Assemblymember

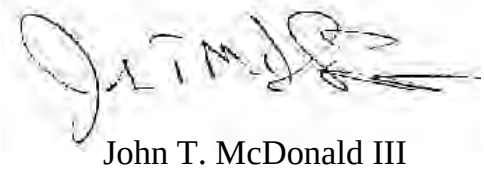
Ellen Jaffee
Assemblymember

Brian P. Kavanagh
Assemblymember

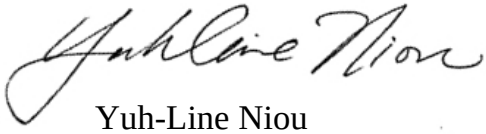
William Magee
Assemblymember



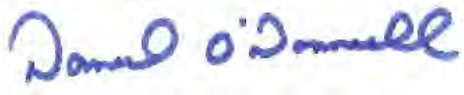
Shelley Mayer
Assemblymember



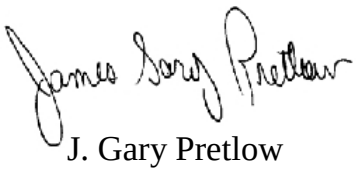
John T. McDonald III
Assemblymember



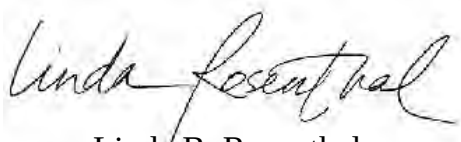
Yuh-Line Niou
Assemblymember



Daniel O'Donnell
Assemblymember



J. Gary Pretlow
Assemblymember

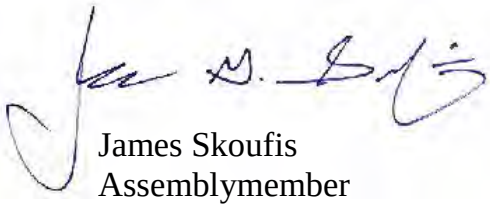


Linda B. Rosenthal
Assemblymember

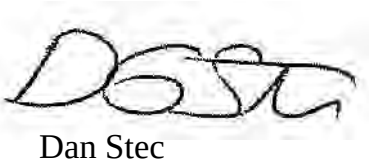
Nily Rozic
Assemblymember

Rebecca A. Seawright
Assemblymember

Jo Anne Simon
Assemblymember

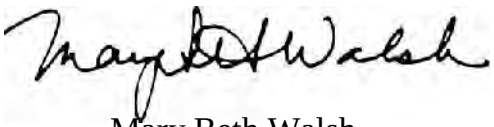


James Skoufis
Assemblymember

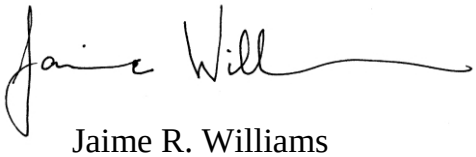


Dan Stec
Assemblymember

Fred W. Thiele
Assemblymember



Mary Beth Walsh
Assemblymember



Jaime R. Williams
Assemblymember

Kenneth P. Zebrowski
Assemblymember

cc:
Administrator Scott Pruitt, U.S. Environmental Protection Agency
Commissioner Basil Seggos, NYS Department of Environmental Conservation

Protect people and wildlife, NYS deserves a Healthy Hudson River

Jose Peralta <info@Riverkeeper.org>

Thu 8/31/2017 3:15 PM

To: Gary Klawinski, Project Director, EPA <epahrfo@outlook.com>;

Aug 31, 2017

Mr. Gary Klawinski, Project Director, EPA
US EPA Hudson River Field Office, Region 2, 187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski, Project Director, EPA,

Mr. Pruitt, Mr. Klawinski.

As a longtime advocate for the people of New York State, I have the following comments on the U.S. Environmental Protection Agency's (EPA's) Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site.

The Hudson is a critical resource. The PCB cleanup is not protective of human health and the environment because it is not performing as planned. We need additional dredging in the Upper Hudson River, as well as a remedial investigation/feasibility study in the Lower Hudson River.
The EPA's review must clearly state "the remedy is not protective." The report states that General Electric's (GE's) cleanup of toxic PCBs it dumped in the Hudson River does not currently protect the health of the public or the river. This indicates a necessity for more investigation in the lower 150 miles.
The economic, recreational, cultural, and scenic value of the Hudson River form the bedrock of past development and future vitality for the Hudson Valley and New York City. Because GE dumped over a million pounds of toxic PCBs into Hudson River from 1947 to 1977, a once vibrant commercial fishing industry has halted; New Yorkers want, a healthy Hudson River, our children deserve a healthy Hudson River.
The New York State Department of Environmental Conservation (NYSDEC) has also expressed its concerns with the findings in the report, stating that the significant amount of contamination left in the river threatens both the public health and the environment.
The EPA agrees that, there is a need for more investigation.
Therefore, I urge the agency to require GE to do a full remedial investigation and feasibility study of the Lower Hudson River.
The National Oceanic and Atmospheric Administration (NOAA) has stated meeting cleanup goals targets would be 60 years later than expected.
Where this to be the case, the EPA's Five-Year Review Report should make this information clear and accessible to the public, who has the right to know how much PCB contamination remains in the River today.

For the Upper Hudson River, the EPA has not yet evaluated all of the

signs that the cleanup will not meet its goals, and for the Lower Hudson River, the EPA has recognized that the cleanup is not working as anticipated, yet we do not have a plan for a prompt investigation or cleanup. It is imperative that GE takes more PCBs out of the Upper Hudson River, and in turn, devises a proper plan for the Lower Hudson River.

Thank your efforts in ensuring a healthy Hudson River for generations to come.

Sincerely,

Senator Jose Peralta
District 13, NYS
32-37 Junction Boulevard
East Elmhurst, New York 11369

Sincerely,

Mr. Jose Peralta
3237 Junction Blvd
East Elmhurst, NY 11369-2605
(718) 205-3881
peralta.socialmedia@gmail.com

FW: NOAA's Comments on EPA's Proposed Second Five Year Review

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Wed 9/6/2017 9:56 AM

To:'epahrfo@outlook.com' <epahrfo@outlook.com>;

1 attachments (105 KB)

NOAA's Trustee comments on FYR report 9-1-17.pdf;

From: Tom Brosnan - NOAA Federal [mailto:tom.brosnan@noaa.gov]
Sent: Friday, September 01, 2017 9:52 AM
To: Rosman, Lisa (NOAA) <Lisa.Rosman@noaa.gov>
Cc: Klawinski, Gary J <Klawinski.Gary@epa.gov>; Jay Field <jay.field@noaa.gov>; Kimberly Katzenbarger - NOAA FEDERAL <kimberly.katzenbarger@noaa.gov>
Subject: Re: NOAA's Comments on EPA's Proposed Second Five Year Review

Hi Gary: please also find attached NOAA's trustee comments on EPA's May 31, 2017 Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site. Please contact us if you have any questions.
Regards,
Tom

On Wed, Aug 30, 2017 at 7:06 PM, Lisa Rosman - NOAA Federal <lisa.rosman@noaa.gov> wrote:

> Gary
>
> Please accept NOAA's comments on EPA's May 31, 2017 Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site. Please contact us if you have any questions.
>
> Lisa and jay
>
> --
> Lisa Rosman
> NOAA/ORR/ARD
> 290 Broadway, 20th Fl
> NY, NY 10007
> 212-637-3259 voice
> 212-637-4206 fax
> 206-619-7965 cell

--
Tom Brosnan
Deputy, Assessment and Restoration Division

NOAA's Office of Response and Restoration
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OR&R's mission is to protect and restore ocean and coastal resources from the impacts of oil, chemicals, marine debris, and other hazards. We provide expert leadership, training, and time-critical services that benefit the environment, public, and economy.

By Electronic Mail

September 1, 2017

Mr. Gary Klawinski, Director
Hudson River Field Office
U.S. Environmental Protection Agency, Region 2
187 Wolf Road, Suite 303
Albany, NY 12205

Subject: Second Five-Year Review Report for the Hudson River PCBs Superfund Site

Dear Mr. Klawinski:

National Oceanic and Atmospheric Administration, in it s role as a Natural Resource Trustee for the Hudson River appreciates the opportunity to provide comments on EPA s second Five-Year Review report. We share EPA s goal of the successful recovery of the Hudson River—a nationally significant ecological, cultural, and economic resource—from PCB contamination. Our comments are provided to further that shared goal.

Under federal Superfund law, the General Electric Company (GE) is responsible for both the remediation -- cleanup -- of the PCB contamination, and the restoration of the natural resources harmed by PCBs. The State and Federal Hudson River Natural Resource Trustees are conducting a natural resource damage assessment (NRDA) and will seek to recover damages from GE to restore the natural resources of the Hudson River on behalf of the public.

PCBs released from GE facilities on the Upper Hudson River present a serious and long-term threat to the health of the entire Hudson River ecosystem. PCBs are highly toxic, cancer-causing compounds, and have contaminated the surface water, groundwater, sediments, and floodplain soils of the Hudson River. Living resources at every level of the Hudson s aquatic, terrestrial, and wetland based food chains are contaminated with PCBs.

GE s PCBs have significantly injured the public s natural resources for over 200 miles (from the Hudson Falls plant site to the Battery in New York City, and beyond). These injuries have occurred for decades, and may span the next half century or more following completion of the remedial dredging. The trustees are committed to the timely recovery and restoration of the Hudson River such that fish and wildlife can once again thrive and all people can fully enjoy the Hudson River and all that it offers.

The EPA s s assessment of the effectiveness and protectiveness of the remedy has a connection to the Hudson River Natural Resource Trustees damage assessment, particularly with respect to the amount of time it will take for the river to recover from decades of PCB contamination and the resulting determination of future injury to natural resources. As always, our comments on the remedy seek to maximize the effectiveness of the cleanup and habitat restoration and to reduce the time to full recovery.

We commend EPA for pursuing and implementing a remedy that included the removal of significant amounts of PCBs from the Hudson River. This active removal has reduced the amount of toxic PCBs in the river.

However, we continue to have overarching concerns, including:

1. Substantial quantities and very high levels of sediment PCB concentrations left behind will continue to contaminate and adversely impact natural resources, and the human use of those resources, resulting in ongoing injury and lost uses to the public.

2. Rates of recovery appear to be overestimated for PCBs in water, sediment, fish, and the PCB load traveling from the Upper Hudson River to the Lower Hudson River. These recovery rates and residual contamination drive EPA s determination of how long it will take for the public s natural resources to recover.

Given the highly contaminated residual sediments and the optimistic recovery rates, the remedy as implemented will likely not achieve the targeted reductions of PCB levels in sediments, water and fish tissue within the timeframes originally anticipated by EPA. Further, the magnitude of contamination remaining may limit the type and amount of in-river restoration options available to the trustees, particularly in the Upper Hudson River.

3. The extended timeline for recovery of the Hudson River highlights the importance of a robust and data-driven monitoring program. As we have commented previously, the Federal Trustees have concerns regarding the adequacy of the monitoring program to provide an appropriate baseline for evaluating recovery and how the public s natural resources, including the human use of those resources, will be adversely affected.

4. EPA notes in the Five Year Review Report that the remedial work in the Upper Hudson River will have little or no beneficial impact in the Lower Hudson River. This is in contrast to the ROD assumption that PCB loading from Upper Hudson to the Lower Hudson plays a major role in recovery of the Lower Hudson River. EPA appears to have rejected this major ROD assumption with little technical basis provided in the draft FYR report.

All four of these overarching concerns relate to the timing and extent of recovery of the river. Such recovery affects future injuries, and thus has a bearing on the trustees need to pursue restoration to compensate for such injuries. Attached are additional technical comments from our membership on EPA s Second Five Year Review Team. Our aim in sharing this information is to provide EPA our best available science to help inform your decision-making regarding the effectiveness and protectiveness of the remedy.

We will continue to work with EPA to achieve our shared goal of successful recovery of the Hudson River from PCB contamination.

Sincerely,

_____

Thomas Brosnan
Hudson River Case Manager
National Oceanic and Atmospheric Administration

By Electronic Mail

September 1, 2017

Gary Klawinski, Director
US Environmental Protection Agency
Region 2, Hudson River Field Office
187 Wolf Road, Suite 303
Albany, NY 12205

Subject: Technical Comments on EPA's Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site, May 31, 2017

In a letter to non-government organizations, the U.S. EPA (2012) committed to continue to consult with the Trustees including on the scoping, data collection, and preparation of the second Five Year Review. Subsequently, \in 2016, NOAA was invited as technical experts to participate in EPA s Second Five Year Review (FYR) Team to provide review and feedback on a variety of FYR topics. NOAA accepted this offer (Brosnan et al. 2016a) and throughout the process provided detailed analysis and feedback that was intended to improve EPA s technical analyses and transparency, so that EPA would have an informed basis for evaluating the effectiveness and protectiveness of the remedy, based on the best available science. NOAA s feedback was provided at several FYR meetings as comments, presentations and follow up letters (*e.g.*, Field et al, 2016; Field and Rosman 2016; Brosnan and Jahn 2016, Brosnan *et al*. 2016b)[1]. NOAA s technical comments on the FYR report follow.

The primary objective of EPA s Proposed Second FYR for the Hudson River PCBs Superfund Site is *to determine whether the remedial actions at the Hudson River PCBs Superfund Site (Site) are protective of public health and the environment and functioning as designed*. [2] Based on our review of the report and the underlying data NOAA believes that certain Record of Decision (ROD) assumptions (*e.g.* sediment surface PCB concentrations and mass, impact of remedy on lower Hudson, and PCB recovery rates in water, sediment and fish,) are not being met), and, as a consequence, the protectiveness expected in the ROD will be substantially delayed. A summary of NOAA s comments and recommendations follow:

[1] These submittals should be included in Appendix 12 to the FYR list of correspondence provided to EPA by NOAA or the Hudson River Natural Resource Trustees.
[2] Executive Summary pg. 1

1. **Significant amounts of elevated PCB contamination have been left in Upper Hudson River (UHR) sediments following remedy implementation which will further delay recovery of Trustee resources:**

    a. **The Upper Hudson in-river remedy leaves highly elevated PCBs in the sediment surface and at depth in the immediate vicinity of dredged areas in River Sections (RS) 2 and 3 (i.e., incomplete PCB sediment deposit removal)**

    b. **PCB mass remaining outside of dredged areas is underestimated in RS2 and RS3**

    c. **FYR estimates of monitored natural attenuation (MNA) recovery rates appear to be higher than supported by data and analyses for PCBs in water, sediment, fish, and PCB load to the LHR. Overestimation of the rate of recovery reduces the ability of EPA's models to discriminate among remedial alternatives.**

        i. **Assessment of remedy effectiveness and protectiveness should be based on measured post-dredging PCB concentrations per the ROD and not an overreliance on percent reduction in PCBs and PCB decay rates**

        ii. **The FYR reliance on retroactive data adjustment adds significant uncertainty to temporal projections of PCBs for fish and sediment**

    d. **Underestimation of Total and Tri+PCBs in sediment based on recent EPA Method 1668 split-sample analysis is not addressed in FYR**

2. **The ROD assumption that PCB loading from UHR to the LHR plays a major role in LHR recovery appears to be rejected with little technical basis provided**

3. **The 2016 surface sediment monitoring plan does not provide an appropriate baseline for evaluating sediment recovery**

NOAA's recommendations for improving the FYR are as follows:

- When calculating mass, Sediment Sampling and Analysis Program (SSAP) cores with an unclassified sediment texture should be treated as fine-grained sediments rather than gravel or bedrock as many of these cores most likely represent undredged PCB deposits.
- The post-source control period from 2005 to 2008 should be used as baseline when calculating HUDTOX-generated MNA PCB decay rates for water.
- Calculation of MNA decay rates for water should only use PCB monitoring data from the baseline monitoring sampling period because of major changes in sampling location and sample collection method.

- PCB load from the UHR to the LHR should continue to be measured in a consistent manner and a more robust analysis is required to assess the impact of the UHR on the LHR.
- The MNA period for fish should begin in 1997 rather than 1995, which is consistent with prior practice. (*i.e.*, use consistent data).
- For evaluating temporal trends in fish, use the long-term monitoring species (or species groups) and stations established by NYSDEC and restrict the size range and time of year to be consistent with NYSDEC monitoring and EPA s food web models.
- Assess the impact of using a single correction factor to adjust year(s) of fish data on the uncertainty of the temporal PCB trend analysis.
- Conduct rib-in vs rib-out comparative study for other fish species that were previously incorrectly processed using non-NYS Standard Fillet protocols.
- Increase sample size, sampling segmentation (0-2, 2-6, 6-12 inches) and spatial resolution of post-remediation sediment sampling design sufficient to create a surface weighted average concentration (SWAC) for cohesive sediment in each river pool in order to capture the highly contaminated unremediated cohesive sediment areas in RS2 and RS3 sampled in the SSAP, and treat these as a separate stratum from the non-cohesive sediments.
- Measured PCB concentrations should be the primary measure of remedy success as defined by the 2002 ROD rather than decay rates or percent reduction.
- For future PCB sample analyses, switch to EPA Method 1668 entirely (preferred option) or use a higher percentage (*i.e.*, at least 25%) of split-sample PCB congener Method 1668.
- Incorporate Hudson River Reference Material into future fish PCB analyses.
- For past data adjustments, analyze archived sediment and fish samples (or sample homogenates for fish) by PCB congener Method 1668 that had previously been analyzed using PCB Aroclor Method 8082 or the modified Green Bay Method (mGBM).

DETAILED TECHNICAL COMMENTS ON THESE POINTS FOLLOW:

Remedy leaves highly elevated PCBs in the surface and at depth in the immediate vicinity of the dredged areas in RS2 and RS3 that will significantly delay recovery of the river.

The extensive SSAP coring for the dredge area design demonstrated that surface sediment PCB concentrations were considerably higher, shallower, and more widespread than EPA expected, especially in RS2 and RS3 (Field *et al.* 2009; USEPA 2012). The majority of the highly elevated PCBs in surface sediment and PCB mass were found immediately adjacent to defined dredge areas (Field *et al.* 2011a; Field *et al.* 2011b). Approximately 175 acres surrounding the dredged areas in RS2 and RS3 exceeded the more stringent cleanup levels for RS1 for PCB mass (MPA) or surface (top 12 inches) PCB concentration (Field et al 2016). The FYR (USEPA 2017) confirmed that PCB mass within PCB contaminated sediment deposits was dramatically higher than the 2002 ROD expected. Because target cleanup levels for RS2 and RS3 were approximately 3X higher for PCB mass and surface concentrations than in RS1, the dredge areas for RS2 and RS3 surgically removed a portion of larger sediment PCB deposits, essentially removing the hole, but leaving the donut of contamination un-dredged.

FYR underestimates PCB mass outside of dredged areas.

> ***Recommendation: Treat SSAP cores with "unclassified" sediment types differently when calculating post-dredging mass, as these SSAP cores most likely consist of fine-grained sediments representative of PCB deposits.***

The FYR confirmed that PCB mass within PCB contaminated sediment deposits was dramatically higher than the ROD expected. According to Table A8-2 (USEPA 2017, Appendix 8), the ROD substantially underestimated the PCB mass in all three river sections. Overall, the observed total PCB mass removed under the Remedial Action was 223% greater than the ROD estimate from approximately the same number of acres. Total PCB mass was underestimated by 45% and 220% for RS2 and RS3, respectively. PCB mass per acre was underestimated by 26% and 349% for RS 2 and RS3, respectively. This implies that the PCB deposits had significantly more PCBs than the ROD expected. The FYR attributes the reason for these differences to *earlier estimates … based on cores that did not fully characterize the vertical extent of contamination* [3], but provides no documentation that this was the primary explanation for the differences from the ROD expectations in RS2 and RS3. According to NYSDEC, underestimation of the depth of contamination associated with inadequate core penetration was observed in RS1 but was limited in RS2 and RS3. Therefore, it is reasonable to assume that the un-dredged PCB deposits adjacent to the dredged areas in RS2 and RS3 also had higher PCB mass than the ROD expected. Further evidence is provided by our observation that sediment samples exceeding RS1 target cleanup levels in all three river sections had similar average surface (top 12 inches) PCB concentrations (19-25 mg/kg Tri+PCBs) and MPA (8-9 g/m^2 Tri+PCBs).

[3] Appendix 2, Section 4.5, pg. 4-8

The FYR generalized assumption that *unclassified areas within River Section 3… were predominately comprised of gravel and bedrock substrate* [4] increases the uncertainty of EPA s estimates of PCB mass remaining in the river. Most of the areas with multiple unclassified SSAP cores (without a sediment type classification) are located in shallow nearshore or backwater areas. These unclassified shallow nearshore and backwater areas are often adjacent to dredge areas and represent unremediated PCB sediment deposits. The 240 unclassified cores in RS3 had an average MPA of greater than 7 g/m^2 Tri+PCBs, and 148 (62%) of those cores exceeded the target cleanup levels for RS1. However, the FYR assigns the average MPA for gravel (2.5) or bedrock (0.00) as upper and lower bounds, respectively, in the calculation of PCB mass outside the dredged areas. Using a lower average MPA for unclassified cores than was actually measured leads to an underestimation of PCB mass. At a minimum, the unclassified areas represented by SSAP cores (~30 acres) should be treated as cohesive fine grain sediment in the calculation of mass remaining in un-dredged areas.

FYR estimates of MNA recovery rates appear to be higher than supported by data and analyses for PCBs in water, sediment, fish, and PCB load to the LHR.

> ***Recommendation: Consistent with prior practice, the MNA period should begin in 1997 rather than 1995.***

The FYR relies on estimated recovery rates for water, sediment, fish, and PCB load to LHR to confirm model estimates of approximately 8% per year. In most cases, as discussed below, the FYR uses data treatment approaches that result in elevated rates of recovery.

The FYR incorrectly defines 1995 as the beginning of the MNA period. Previously, EPA recognized that PCB releases from the failure of the Allen Mill gate structure and from the migration of PCB oil through the bedrock were not mostly controlled by remedial measures until 1997 (USEPA 2000a). For that reason, it has been customary to use 1997 as the starting point for pre-dredging temporal analyses. For example, for fish, the time period of 1997-2008 was used as the basis for development of pre-dredging temporal models (USEPA 2010b; Greenberg *et al.* 2010; Greenberg *et al.* 2011; Greenberg 2013).

Water

> ***Recommendation: Calculation of MNA decay rates for water from the HUDTOX model should use the post source control period from 2005 to 2008. Calculation of MNA decay rates from PCB monitoring data should only use data from the baseline monitoring sampling because of major changes in sampling location and sample collection method.***

The FYR reports that revamped HUDTOX model MNA predictions based on updated hydrologic conditions (but not updated surface sediment concentrations) forecast PCB decay rates between 9.9% and 11.7% for the four stations (Thompson Island Dam, Schuylerville,

[4] Appendix 2, Section 4.4, pg. 4-6

Stillwater, Waterford) considered (USEPA 2017, Appendix 1, Table A1-7). These estimated PCB decay rates are considerably higher than the reported data-based rates for Stillwater and Waterford for the same 1995-2008 time period. Unfortunately, neither the MNA predicted decay rates nor the data-based decay rates should be taken at face value. The HUDTOX model incorporated a 6-fold drop in upstream water concentration (from 0.16 kg PCB/day to 0.0256 kg PCB/day) occurring January 1, 2005 (USEPA 2002). Calculating a decay across that step-wise drop in PCB input concentration provides more information on source control at the two GE plant sites than on natural recovery of UHR sediments (Field and Rosman 2016). Between 2005 and 2008, a period of natural recovery, NOAA calculated PCB decay rates for the four stations using data from the revamped model (USEPA 2017, Table A1-7) that are considerably lower than reported in the FYR, ranging from 0.01% to 5.5%.

The FYR evaluation of the water column data-based decay rates does not account for the PCB releases from the failure of the Hudson Falls Allen Mill gate structure in 1991 and from the migration of PCB oil through the bedrock. Although not as marked as during the initial period of GE s Allen Mill release, the continuing impact to PCBs in the water column is evident from the Rogers Island water column monitoring data (see Attachment, Figure 1---plot of Rogers Island water data provided by NYSDEC with the period between 1995 and 1997 highlighted for emphasis). Additionally, the FYR analysis does not account for major changes in sampling location (*e.g.*, Thompson Island station moved from nearshore to mid-channel) and method (shift to automated samplers) beginning in June 2004 with the initiation of GE s baseline monitoring program. The high variability, compounded by differences in sampling location and methods, makes the currently available surface water data an unreliable measure of temporal change in PCBs.

PCB load to LHR

Recommendation: NOAA supports the recommendation of the Hudson River Foundation report (Farley et al 2017) to re-instate the USGS suspended sediment monitoring at Waterford and to collect additional high flow samples to improve evaluation of PCB loading to the LHR.

The measured PCB load to the LHR between 2004 and 2008 was 2 to 3 times greater than the original HUDTOX projections (USEPA 2010a; Hydroqual 2010). In the FYR, EPA updated the HUDTOX model projections with observed flows and estimated tributary flows and solids loads through 2008, but did not update the sediment concentrations with SSAP data. The updated HUDTOX model projections in the FYR (USEPA 2017, Appendix 1, Table A1-8) improved the model-data comparison, but still underestimates the measured 2004-2008 PCB loads by 8-41%, with the difference increasing with time. Based on data provided in the FYR (USEPA 2017, Appendix 1, Table A1-8), the updated HUDTOX model predicts PCB load to the LHR between 2004-2008 will decrease at a rate of 9.2%, while the estimated decay rate from the measured PCB load has a decay rate of 2.8% [Attachment 1, Figure 2]. This shows that, prior to dredging, PCB loading to the LHR was declining at a much slower rate than the updated HUDTOX model predicted.

Sediment

Recommendation: The comprehensive sediment sampling data from the SSAP should be treated as the baseline for evaluating recovery of PCB-contaminated cohesive sediment in un-dredged areas. Studies conducted since the SSAP (Downstream Deposition study 2011-3 and 2016 post-dredging sediment study)* mostly do not address the *highly PCB-contaminated cohesive sediment areas adjacent to dredged areas in RS2 and RS3 and should not be used as a measure of sediment recovery without significant caveats. Sediment grain size data should be used to reduce the uncertainty in defining cohesive sediment areas.

EPA s 2002 ROD assumed that implementation of the selected remedial alternative REM 3/10/S would result in post-dredging surface sediment concentrations in RS2 and RS3 less than or equal to 1 ppm Tri+PCBs in cohesive sediment, comparable to post-dredging surface sediment concentrations in RS1. EPA (USEPA 2010b) confirmed the finding of Field *et al.* (2009) that pre-dredging surface sediment concentrations were *much higher than model predictions* [5] and *exceed the upper bound of model predictions*. [6] As discussed earlier, the highly elevated PCBs in surface sediment in the SSAP samples were mostly immediately adjacent to dredge areas. Unfortunately, the surface sediment surveys conducted since the SSAP data collection (Downstream Deposition Study (DDS) 2011-3 and 2016 post-dredging sediment study) provide data that are not directly comparable to the SSAP data (very limited data collected from the highly contaminated cohesive sediments surrounding the dredge areas and only sampled the top 2 inches and not the top 12 inches of surface sediment used to define dredge areas). In addition, the analysis of split sediment samples in 2016 using the current EPA standard method for PCB congener analysis (Method 1668) indicates that the PCB Aroclor analysis (Method 8082) for those studies significantly underestimated Tri+ and Total PCBs.

The surface sediment PCB concentrations for cohesive sediment in RS2 and RS3 estimated from the DDS sediment survey and 2016 sediment monitoring survey should be considered to be biased low. The DDS survey used a biased sampling design to specifically focus on the downstream edge of dredge prisms. According to EPA (USEPA 2016), *If assessing changes in conditions for the entire river section were the DQO, then care would have been taken to ensure that the distribution of the PCB concentrations targeted by the DDS program would have been a representative subset of the SSAP program*. This was clearly not the case for RS2 and RS3. The sample locations selected for comparison to nearby (within 20ft) SSAP locations outside of dredge areas represented locations that had PCB concentrations that were significantly higher than the results for all SSAP locations for RS2, and the median PCB concentration was higher than the 95% UCL for all SSAP locations in RS3. Attempting to re-sample high concentration

[5] USEPA (2010b), pg. I-53
[6] Ibid.

samples from a lognormal distribution has a high statistical probability that the re-sample result will be lower than the original sample (Field et al. 2015).

The FYR uses the side-scan sonar results from 1992 to classify the UHR bottom sediment type, rather than using the GE results from the SSAP. Because the Reassessment bottom type mapping did not cover RS3, EPA chose to create a model to predict sediment type in RS3 from the GE data. The 2016 samples are classified into cohesive and non-cohesive samples based on this predictive model, which adds considerable uncertainty to the classification, in spite of the fact that sediment grain size analysis on the 2016 samples was available for classification. In the baseline modeling report (USEPA 2000b), samples with at least 25% fines (silt + clay) were classified as cohesive sediment. Only about 1/3 of the samples identified as cohesive by the predictive model for RS3 had at least 25% fines and more than 20% had sediment texture classified by the field samplers as coarse or rock. Including samples with a much lower percentage of fines likely underestimates the PCB concentration in cohesive sediments. For example, identifying cohesive sediment based on grain size analysis in RS3, the arithmetic mean Tri+ PCB concentration is 1.3 (mg/kg) compared to 0.8 as reported in the FYR (USEPA 2017, Appendix 4, Table A4-3). These concentrations do not take into account the underestimation of PCB concentration by the Aroclor Method (discussed elsewhere in this document). The adjusted mean cohesive sediment Tri+ PCB concentration in RS3 is 4.2 (mg/kg), based on the correction factor from the split-sample Method 1668 analysis.

Fish

> ***Recommendation: For evaluating temporal trends, use the long-term monitoring species (or species groups) and stations established by NYSDEC and restrict the size range and time of year to be consistent with NYSDEC monitoring and EPA's food web models. Use only lipid-normalized data to evaluate temporal trends and for comparison to food web model projections use wet weight values adjusted to the standard lipid content for each fish species used in the modeling. Assess the impact of using a single correction factor to adjust year(s) of PCB data on the uncertainty of the temporal fish trend analysis. Conduct rib-in vs rib-out comparative study for other fish species incorrectly processed using non-NYS Standard Fillet protocols. Collect sufficient spatial data to analyze fish concentrations on a pool by pool basis, rather than river section basis.***

Evaluation of temporal trends in fish PCBs requires consistent sampling for fish species from specific sampling locations over time. Because PCBs in fish are strongly associated with lipid content and lipid content has decreased in spring-collected resident species, analyzing temporal trends should take into account lipid content and not rely on wet weight concentrations. Prior to the GE s implementation of the baseline monitoring plan (BMP) in 2004, all fish data were collected by NYSDEC from 2 regular monitoring stations in the UHR Thompson Island Pool (RS1) and Stillwater (RS3) for spring-collected resident species (bullhead, black bass, yellow perch) and fall-collected forage fish (yearling pumpkinseed). In the LHR, 3 regular monitoring locations (Albany/Troy, Catskill, and Poughkeepsie) for the same species with the addition of white perch. These species/locations represent the most robust and consistent dataset to evaluate

temporal trends. In contrast, the BMP sampled multiple locations in each river section in the UHR. By including all BMP sampling locations from RS1 and RS3, the FYR overweighs the data from the BMP (2004-8) and adds significant uncertainty by including the additional stations. The variability among stations within river sections is clearly evident in Greenberg *et al.* (2010, 2011). The other species used in the FYR fish trend analysis were inconsistently sampled throughout the time period and not suitable for long-term PCB temporal trend analysis.

The FYR temporal analysis does not account for rib-on /rib-off difference for lipid-normalized PCBs. Based on an unpublished special study for black bass (largemouth and smallmouth bass), the FYR minimizes the effects of GE s change in fillet processing protocol by excluding the 2007-8 fillet samples from temporal trend analysis for wet weight PCBs. Lipid-normalized results were assumed to be unaffected by this change, in spite of the fact that average lipid-normalized concentrations for rib-on fillets in black bass were ~22% higher (13-31% 95% confidence interval) than the rib-off samples and would require a correction factor analogous to the FYR homologue adjustment factors. Additionally, the FYR assumes, with no supporting data, that the special study results for black bass apply equally to all other fillet species (*e.g.*, bullhead, yellow perch, white perch, catfish, and striped bass) (Brosnan *et al.* 2015; Brosnan and Jahn 2015; Brosnan *et al.* 2016). Including the 2007-8 lipid-normalized fillet data, which is biased low by an unknown degree, results in an inflated temporal decay rate for those species.

The homologue correction factor used in the FYR for NYSDEC data from 1999-2011 uses the wet weight adjustment factor for the lipid-normalized results, in spite of the fact that both NY and GE labs analyzed lipid along with PCBs and the correction factors for the lipid-normalized concentrations were different. NYSDEC data during the MNA (pre-dredging) period from 1999-2003, before GE began sampling in 2004 for the baseline monitoring program, was inflated by this approach, because the wet weight correction factor used by EPA was 1.17 compared to a lipid-normalized correction factor of <1 (0.96). This inflates the NYSDEC data during the period from 1999-2003 before GE began sampling in 2004 for the baseline monitoring program and effectively increases the estimated temporal decay rate.

Using only the lipid-normalized PCB data from the principal monitoring stations, species or species groups, and MNA time-period from 1997-2006 (excluding data from 2007-8 when GE incorrectly processed fillet samples), NOAA calculated exponential decay rates using the original (unadjusted) data, the FYR-adjusted data, and modifications to the FYR adjustment factors for NYSDEC fish data that incorporated the lipid-normalized adjustment factors (discussed above) (Table 1). The PCB decay rates vary somewhat among the 3 data approaches, but the overall conclusions are much the same. In the UHR, only black bass and yellow perch from the Thompson Island Pool monitoring station show PCB decay rates greater than 8%. Bullhead (the species most closely associated with the sediment) and yearling pumpkinseed from that same location have PCB decay rates of less than 5% and 0%, respectively. At the other UHR long-term monitoring locations in the Stillwater Pool, all species had PCB decay rates less than 5%.

At the Albany/Troy location all species except pumpkinseed had PCB decay rates of 4% or less. Pumpkinseed showed a very high PCB decay rate at Albany/Troy, but the sampling location was changed several times during the time period. Because pumpkinseed are known to show high site fidelity, the changes in sampling location makes those results for pumpkinseed highly unreliable. Other locations in the LHR (Catskill and Poughkeepsie) showed similarly low PCB decay rates. Overall those results were very consistent with findings of Field et al (2016) based on emulation of the HUDTOX-FISHRAND models for the LHR applying updated surface sediment concentrations.

The sampling program was designed to determine PCB concentrations in fish by river section rather than each river pool. The river pool sampling approach for fish (sediment and water) is essential to establishing a post-dredging baseline for evaluations of fish exposure in the UHR and LHR. Resident fish tend to remain within a river pool, which means they integrate their exposure within pools or smaller areas, and not over much larger river sections (Field and Kern 2009b).

FYR appears to reject ROD assumption that PCB loading from UHR to the LHR plays a major role in LHR recovery

Recommendation: Need a more robust analysis of impact of UHR on LHR.

The FYR appears to disregard prior conclusions and modeling results in the ROD (USEPA 2002) that the UHR PCB load to the LHR is the primary factor in the recovery of LHR fish. The FYR cites slower recovery of LHR fish as evidence that the UHR does not play an important role in LHR and speculates about other sources . Based on high-resolution core sampling data and modeling (Thomann *et al.* 1989, Farley *et al.* 1999, USEPA 2000a, Hydroqual 2007, Rodenburg and Ralston 2017), the primary source of PCBs to the LHR is the result of past and continued loading of PCBs originating from the Hudson Falls and Fort Edward plant sites and sediments within the UHR.

EPA concluded in their Phase 1 report (p. I-4) that,

> *The observed baseline loads to the Lower Hudson prior to dredging were substantially greater than the model forecast of Monitored Natural Attenuation (MNA) and show very little decline. The loads to the Lower Hudson River under MNA will be substantially greater than those forecast by the model by approximately 6,000 kg over 25 years. Also the surface sediment concentrations in the Upper Hudson River remain elevated despite the passage of time and continue to provide a greater reservoir of contaminated sediments for transport to the Lower Hudson than was envisioned when the remedy was selected*.

Post-dredging, as pointed out previously, most of the remaining sediment PCB contamination is found in RS2 and RS3. Based on GE s modeling, Connolly *et al.* 2000 pointed out the importance of sediment remediation in RS2 and RS3 in reducing PCB loading to the LHR:

Sediment remediation in the TIP would be less effective at reducing PCB flux to the Lower Hudson River in the short term than would remediation of sediments downstream of the TIP.

There appears to be little basis to reject the ROD s assumption that UHR sediment PCBs are a major factor in the recovery of LHR fish, given the higher than expected PCBs in surface sediment and the much slower decline of PCB loading to the LHR.

2016 surface sediment monitoring plan provides inadequate baseline of PCBs for evaluating sediment recovery.

> ***Recommendation: The sediment monitoring program should be modified to adequately address the highly contaminated sediments in RS2 and RS3, which will remain a major source of PCBs to Upper Hudson food webs and provide continued PCB loading to the LHR. The highly contaminated cohesive sediment areas sampled during the SSAP should be treated as a separate stratum from the non-cohesive sediments and more samples are needed within those cohesive areas per river pool to establish a surface sediment baseline for evaluations of fish exposure, PCB loading to the Lower Hudson River, and the rate of recovery of the system. Core samples should be collected and analyzed from 0-2, 2-6, and 6-12 inch intervals consistent with the definition of "surface" as the top 12 inches in the ROD (USEPA 2002) and confirmed in the Final Dispute Resolution (July 26, 2004).***

EPA s 2016 sediment sampling plan is intended to provide a baseline for future monitoring to determine the rate of recovery in surface sediment. The underlying premise assumes that a SWAC for each entire river section is the best metric for evaluating recovery. The modeling done by EPA to support the ROD (and by GE) is based on the understanding that cohesive (fine-grained) sediment provides the foundation for the food web (NOAA 2016). However, the 2016 sediment sampling, by virtue of the design, provides only minimal information on the known highly contaminated unremediated areas (mostly cohesive sediments) surrounding the RS2 and RS3 dredge areas that were identified in the SSAP. Therefore, the 2016 sediment sampling provides an inadequate post-dredging baseline.

Cohesive sediments represent the primary source of exposure to the benthic food web and fish species, but most of the 2016 samples were collected from non-cohesive sediment areas. The highly contaminated cohesive sediment areas sampled during the SSAP should be treated as a separate stratum from the non-cohesive sediments and more samples are needed within those cohesive areas to properly characterize them.

The sampling program was designed to determine the PCB SWAC by entire river sections rather than the smaller river pools (=river reach). The river pool sampling approach is essential to establishing a surface sediment baseline for evaluations of fish exposure, PCB loading to the Lower Hudson River, and the rate of recovery of the system. Resident fish tend to remain within a river pool, which means they integrate their exposure within pools or smaller areas, and not over much larger river sections (Field and Kern 2009b).

The 2016 sediment sampling only collected the top 2 inches of surface sediment, but it is important to measure PCBs in the top 12 inches of sediment. Surface sediment was defined in the 2002 ROD and in the Final Dispute Resolution (July 26, 2004) as 0-12 inches. The PCBs in

the top 12 inches represent a more complete accounting of the PCBs biota are or may be exposed to in the future, as well as the mass that may be transported to the lower river. In the dispute resolution EPA acknowledged that *The Agency selected a remedy that targeted dredge areas based on, among other things, PCB concentrations representing the top 12 inches of sediment in order to account for all processes – whether physical, chemical or biological - that can make PCBs bioavailable*.

According to the FYR: *One year of post-dredging data indicate a reduction in exposures consistent with EPA's expectations at the time of the ROD.* [7] EPA s 2016 sampling plan, which is proposed to serve as the baseline for future sediment sampling to assess temporal change, only minimally addresses areas of known highly contaminated sediments in cohesive sediments adjacent to dredge certification units (*see* NOAA 2016 comments on plan). Consequently, the sediment monitoring program will not adequately address these highly contaminated sediments in RS2 and RS3, which will remain a major source of PCBs to Upper Hudson food webs and provide continued PCB loading to the LHR. The 2016 sediment monitoring plan also provides an inadequate basis to *indicate a reduction in exposures consistent with EPA's expectations at the time of the ROD.*

NOAA s 2016 comments on the Operation, Maintenance, and Monitoring Plan (OMM) 2016 summarize the concern:

> Both EPA s and GE s bioaccumulation modeling recognized that fine-grained (cohesive) sediments were a major source of exposure for PCBs entering the food web. For example, the EPA bioaccumulation modeling assumed that fish were primarily exposed to cohesive sediment (75%) while in GE s bioaccumulation models [QEA 1999], PCB concentrations in the food web were based entirely on exposure to cohesive sediments. Most vegetation in the Upper Hudson is found associated with fine-grained sediments.
>
> The OMM Plan is not comparable to any historical data, and does not provide sufficient sampling power to address individual reaches in RS2 & RS3, which will provide necessary spatial resolution to measure recovery. Using statistical analysis of DDS data as the basis for sample density underestimates number of samples required for RS2 & RS3 because it doesn t take into account composite sampling in the DDS in RS2 and RS3.
>
> Focusing exclusively on the top 5 cm instead of the bioactive zone of 30 cm (as defined in the EPA-GE Dispute Resolution) provides limited information on the PCBs in the surface that will be available to biota and at potential immediate risk of recontamination of dredged areas and transport to the LHR.

[7] Pg. 67.

FYR assessment of remedy effectiveness over-relies on percent reduction and decay rates rather than actual PCB concentrations

> ***Recommendation: Actual PCB concentrations should be the primary measure of remedy success as defined by the ROD rather than decay rates or percent reduction. The FYR should acknowledge that the highly contaminated areas adjacent to the dredged areas in the SSAP have not been re-sampled sufficiently to determine post-dredging PCB concentrations, percent reduction, or decay rates.***

The FYR emphasizes the percent reduction in PCB mass in the river. The success of the remedy does not depend on the percentage or amount removed, but the magnitude and spatial extent of PCBs left behind, which greatly exceeded expectations in the 2002 ROD. The FYR underestimate of ~60,000 kg PCB mass left behind outside of dredged areas is almost equivalent to the 70,000 kg PCBs the 2002 ROD estimated would be removed from the river by the dredging remedy.

The FYR compares SSAP and 2016 surface sediment data from the top 2 inches to estimate reduction in surface area PCB concentration. This is an apples to oranges comparison, as the 2016 data only minimally included samples in RS2 & RS3 from the highly elevated fine-grained PCB sediment deposit areas adjacent to dredged areas. Additionally, 2016 PCB Aroclor analysis significantly underestimated PCB concentrations based on split-sample analyses (see EPA Method 1668A discussion, below).

Important implications of split-sample PCB analysis are not addressed in FYR.

> ***Recommendation: In future PCB analyses switch to Method 1668 entirely (preferred) or use a much higher percent of split-sample EPA Method 1668 (Method 1668). Incorporate Hudson River Reference Material into future fish PCB analyses. For past data adjustments, analyze archived sediment and fish samples (or sample homogenates for fish) that had previously been analyzed using Aroclor and/or mGBM methods.***

The 2016 sediment sampling included analysis of a subset of the samples by a highly qualified independent laboratory using the current standard for PCB congener analysis (EPA Method 1668A) (Anchor QEA 2017). However, the FYR report does not contain an analysis of these results. NOAA s evaluation indicates that Total PCBs (and Tri+ PCBs) measured by EPA Method 1668A congener analysis were more than twice as high as the Arcolor Method 8082 (geometric mean ratio of 2.4) previously used by Pace laboratory (formerly NEA, GE s contractor for laboratory analysis). This implies that recent previous sediment analysis conducted by GE s Pace laboratory (*e.g.*, DDS 2011-3 analyses) (and possibly earlier analyses conducted by NEA such as SSAP) underestimated PCBs in the sediment. The modified Green Bay Method (mGBM) split-extract analysis conducted by NEA during the last 3 years of the SSAP sampling indicated that the Aroclor total PCB concentrations in sediment were higher than reported from the mGBM peak analysis (USEPA 2017). The 2016 results suggest that the mGBM peak analysis may have also underestimated PCB concentrations in sediment.

Although comparable split-sample data from Method 1668 for fish do not exist, it is reasonable to hypothesize that total PCB concentrations for fish may also have been underestimated. This uncertainty could have been avoided if GE had used the Hudson River Reference Material (HRM) prepared by NYSDEC, as required by the Consent Decree. NYSDEC contract labs have been using the HRM routinely since 2009.

To evaluate the potential implications of this major uncertainty, NOAA recommends that EPA send previously analyzed and archived frozen fish samples to the same laboratory that EPA used to analyze the 2016 sediment-samples using Method 1668 (Axys Laboratory) for PCB congener analysis using Method 1668 that have prior PCB analyses by both Aroclor and mGBM methods and include both the Hudson River Reference Material[8] and Standard Reference Material available from the National Institute of Standards and Technology (NIST) or a commercial vendor.

Homologue adjustment of fish PCB data and estimating sediment type for RS3 adds major uncertainty to the FYR evaluations

> ***Recommendation: Assess the impact of using a single correction factor to adjust multiple years of fish PCB data on the uncertainty of the temporal PCB trend analysis. Confirm that Total PCBs in fish from mGBM are comparable to Total PCBs from Method 1668.***

The FYR s homologue adjustment of NYSDEC and GE fish data uses a single factor based on a geometric mean of the ratio of Aroclor PCBs to mGBM Total PCBs. In the case of the NYSDEC data, the adjustment factor from 1999-2000 is applied to all subsequent years without any data to document applicability. The NYSDEC adjustment factor applies the factor from wet weight analysis to the lipid-normalized concentrations, instead of more appropriately using the factor from lipid-normalized analyses, which are substantially different for some years. Also, for the 1997 NYSDEC fish data, the FYR relies on a model-estimated factor from Butcher *et al.* .(1998), ignoring the data from the split-sample approach used for subsequent years. Using a single factor ignores the uncertainty/variability of the relationship for different subgroups (e.g., species, location, year) and may not represent the pattern in the underlying data. Additionally, the 2016 split-sample analysis for sediment suggests that both the Aroclor and mGBM PCB analyses may significantly underestimate the total PCB concentration from full congener analysis (Method 1668).

> ***Recommendation: Use direct measurement (i.e., sediment grain size analysis) to determine cohesive/non-cohesive sediment type in monitoring sediment.***

The FYR relies on side scan sonar surveys conducted in 1992 to determine sediment type rather than the side scan sonar surveys conducted by GE in 2004 to design the remedy. Because the

[8] "Performance evaluation (PE) samples for fish tissue, in the form of the Hudson River Reference Material (HRM) developed by New York State, shall be incorporated into the program. EPA will consider removing the MS/MSD samples if the HRM material is incorporated." From Appendix B to the Consent Decree, Hudson River PCBs Site, Statement of Work (SOW) for Remedial Action and Operations, Maintenance and Monitoring, December 2010, Section 2.7.5 Measurements.

1992 sediment type analysis did not include RS3, the FYR developed a predictive model of sediment type to determine cohesive/non-cohesive areas in RS3. The model was estimated to accurately predict cohesive/non-cohesive locations 76% of the time. As discussed earlier, a number of samples from the 2016 sediment sampling were incorrectly classified as cohesive based on grain size analysis or sediment texture observations, which results in an underestimation of the PCB concentration in cohesive sediment. Given the importance of the cohesive sediment in estimating sediment recovery and PCB mass, using a model rather than direct measurement increases the uncertainty of the estimates.

Conclusion

In conclusion, NOAA appreciated the opportunity to serve on EPA's FYR panel and to provide technical comments on the draft FYR report. NOAA shares the goal of full recovery of the river from PCB contamination in as short a time as is practicable and commend EPA for the significant dredging that has been achieved. Our technical comments are intended to improve EPA's ability to more accurately assess remedy protectiveness and effectiveness, including the time for the river to fully recover, based on the best available science. Our comments include observations that major ROD assumptions are not being met (*e.g.*, levels and amount of PCBs left behind in sediment and other environmental media, rate of recovery, and impact of the remedy on the LHR), that recovery will be significantly delayed from what was anticipated in the ROD, recommendations on how to improve data analysis, and the need for a much more robust monitoring program to assess protectiveness and effectiveness in future. Please do not hesitate to contact us with any questions or comments or if you would like to discuss these recommendations further.

Sincerely,

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Lisa Rosman

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USEPA 2017. Proposed Second Five-Year Review For Hudson River PCBs Superfund Site, U.S. Environmental Protection Agency, Region 2, New York, NY, May 31, 2017.

ATTACHMENTS

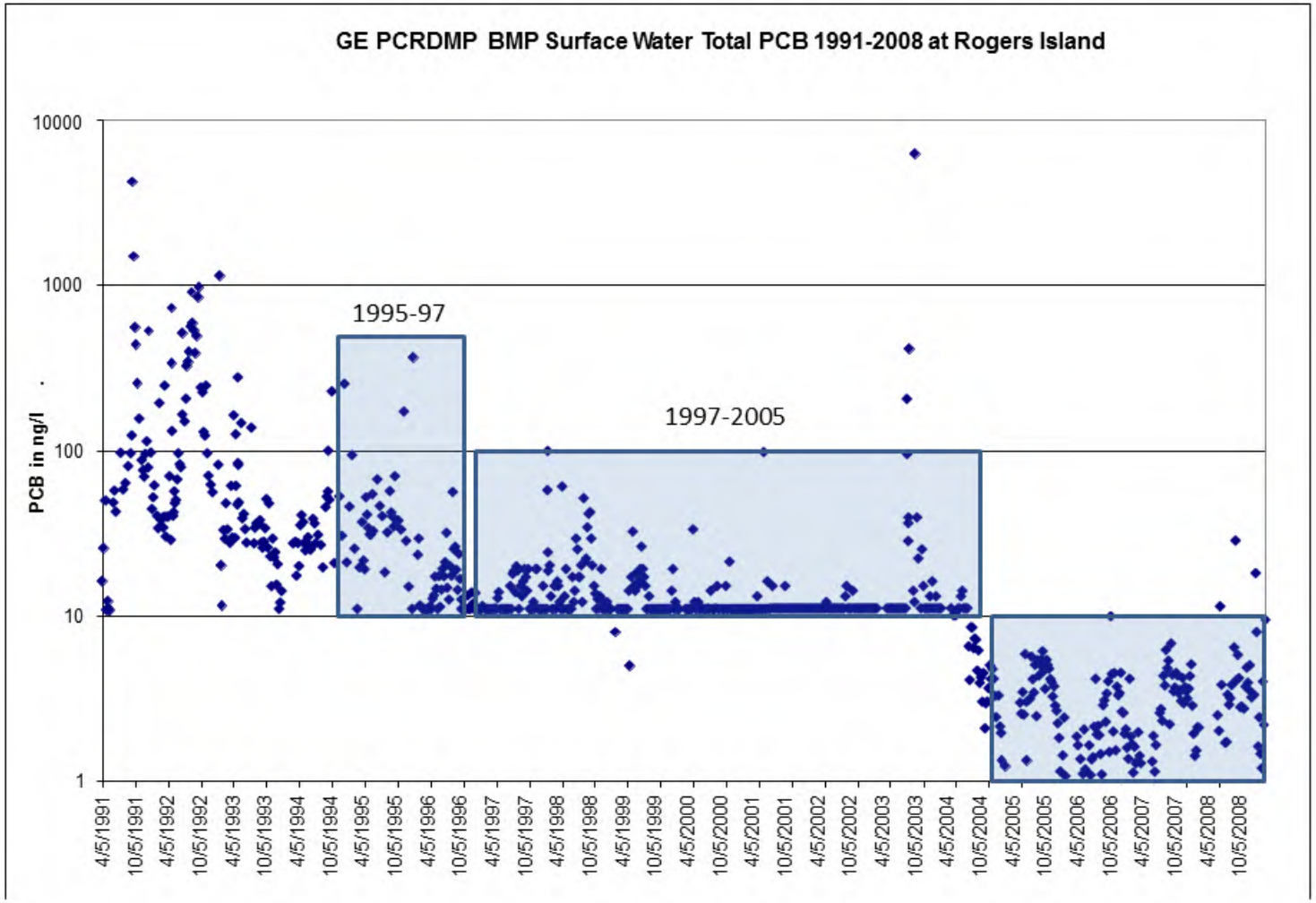

Figure 1. Total PCBs (ng/L) at Rogers Island monitoring station from 1991 to 2008. Data provided by NYSDEC. Emphasis added.

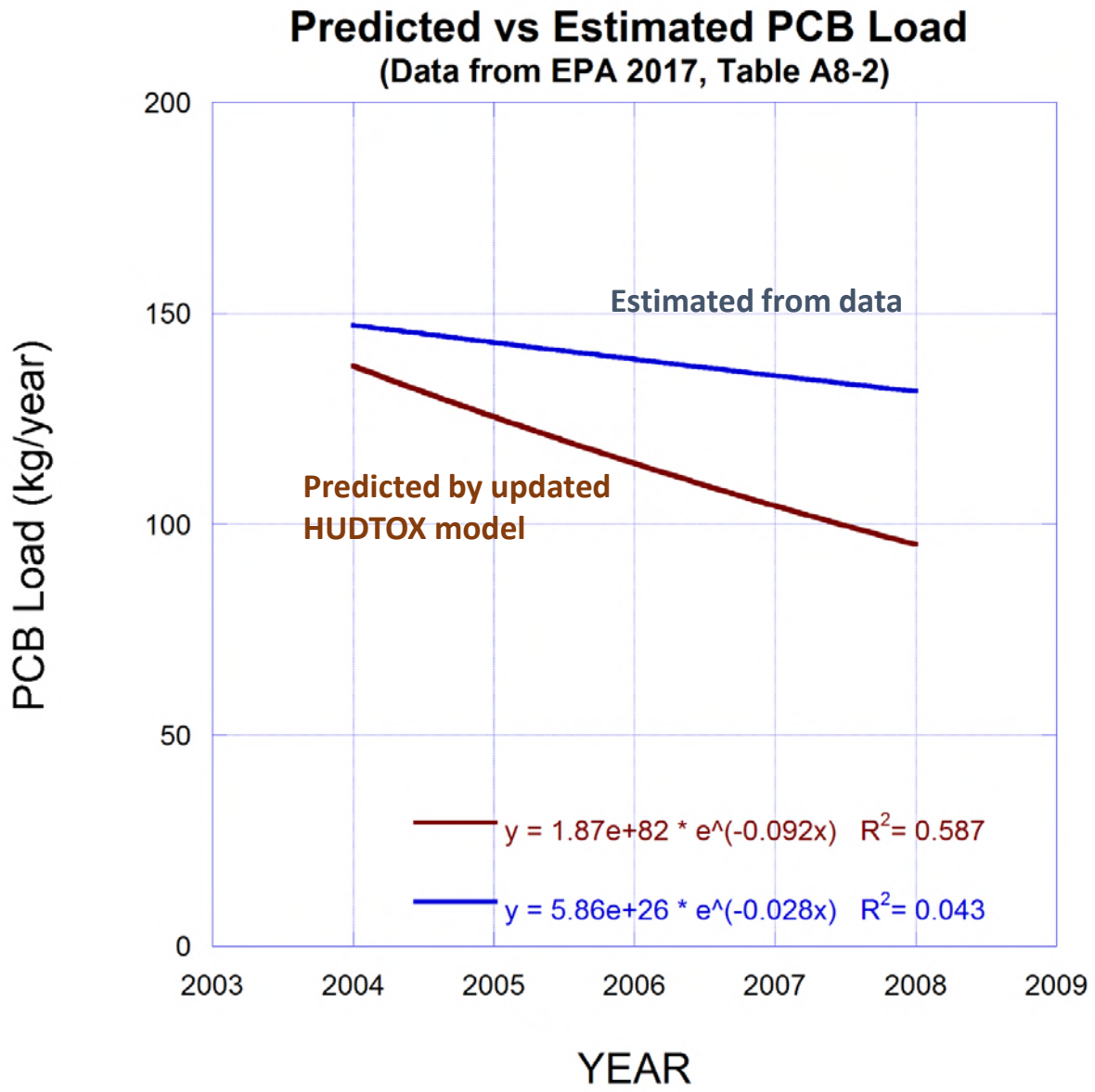

Figure 2. Predicted vs estimated PCB load (kg/year) exponential decay using data from USEPA 2017, Appendix Table A1-8.

Table 1. Exponential decay rate for standard long-term monitoring species and locations between 1997 and 2006.

		1997-2006			
SP_GROUP	RMILE	Unadjusted Data	FYR Adjusted Data	NOAA modified adjusted data	Average
Black Bass	RM189	-0.102	-0.102	-0.089	-0.098
Black Bass	RM176	-0.049	-0.051	-0.039	-0.046
Bullhead	RM189	-0.045	-0.031	-0.038	-0.038
Bullhead	RM176	-0.056	-0.036	-0.048	-0.047
Yellow Perch	RM189	-0.133	-0.131	-0.118	-0.127
Yellow Perch	RM176	-0.007	-0.025	0.001	-0.011
Pumpkinseed	RM189	0.048	0.044	0.052	0.048
Pumpkinseed	RM168	-0.057	-0.046	-0.041	-0.048
Black Bass	RM152	-0.047	-0.042	-0.035	-0.041
Bullhead	RM152	0.017	0.035	0.040	0.031
Yellow Perch	RM152	-0.041	-0.030	-0.017	-0.029
White Perch	RM152	-0.040	-0.027	-0.028	-0.032
Pumpkinseed	RM152	-0.174	-0.153	-0.150	-0.159
Black Bass	RM113	-0.078	-0.041	-0.075	-0.065
Black Bass	RM076	-0.067	-0.016	-0.043	-0.042
Bullhead	RM113	-0.043	-0.001	-0.023	-0.023
Bullhead	RM076	-0.015	0.019	0.000	0.001
White Perch	RM113	0.053	0.094	0.062	0.070
White Perch	RM076	-0.007	0.012	0.002	0.002
Yellow Perch	RM113	-0.007	0.031	0.010	0.011
Yellow Perch	RM076	0.020	0.018	0.019	0.019
Pumpkinseed	RM113	0.056	0.056	0.056	0.056
Pumpkinseed	RM076	-0.050	-0.050	-0.051	-0.051

< 5% decay
> 8% decay

Black Bass includes Largemouth and Smallmouth bass > 250 mm
Bullhead includes Brown Bullhead and Yellow Bullhead >= 175 mm
Yellow Perch > 150 mm

Yearling Pumpkinseed (< 120 mm)

FYR adjusted data provided to NOAA by EPA

NOAA modified adjusted data: uses lipid-normalized adjustment factors for 1997-2011 NYDEC data based on geometric mean ratio

Pumpkinseed Albany/Troy (RM152) sampling location changed over time, which makes temporal evaluation unreliable

Bullhead from Albany/Troy had small sample size

NEW YORK STATE OF OPPORTUNITY. | Bridge Authority

ANDREW M. CUOMO
Governor

JOSEPH RUGGIERO
Executive Director

RICHARD A. GERENTINE
Chairman

June 28, 2017

RECEIVED
JUN 28 2017
Hand delivered @ poughkeepsie

Mr. Gary Klawinski, Director
EPA Region 2 Hudson River Office
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski,

I am submitting my comments on the Hudson River Superfund Draft Second Five Year Review on behalf of the New York State Bridge Authority.

The Bridge Authority has responsibility for five spans that link the two shores of the Hudson River. These beautiful and historic structures stretch from the Bear Mountain Bridge in the Hudson Highlands to the Rip Van Winkle Bridge at the foothills of the Catskill Mountains, and include the dramatic Mid-Hudson Bridge, just down the hill from here. All of the bridges link the communities of the region and contribute to its cohesive economy.

But the river that flows beneath these bridges—and back and forth with the tides in the 66 miles between them—is part of one of the nation's largest Superfund sites, contaminated by toxic PCBs that imperil public health, the environment, people's enjoyment of the river and economic opportunity.

It is my understanding that your draft Five-Year Review acknowledges that GE's cleanup hasn't achieved the goals you set to restore the river's health, but states it will ultimately achieve the goals of those goals in 53 years, a claim that is challenged by other federal and state agencies. I understand that a large quantity of PCBs remains in the upper Hudson that will continue to pollute the river north of Troy and contaminate the lower river. I urge you to issue a Final Five Year Review that deletes the claim that the remedy will be protective and that lays the groundwork for additional action to ensure -- beyond doubt -- that the entire river will be healthy and safe, so the economy of the region can be restored.

I also urge you to evaluate the success of the cleanup by strict adherence to the timeframes in the cleanup plan within which fish consumption advisories were expected to be relaxed. I have a young son, and it pains me that I am not able to tell him with confidence that – at least when he is my age – he and all his family will be able to safely eat fish from the Hudson. It is unacceptable that he may not be able to safely eat fish from our river within his lifetime. Your Five Year Review can and must honestly address this problem.

FW: Supplemental Comment on the Hudson River Five Year Review Report

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Wed 9/6/2017 9:57 AM

To: 'epahrfo@outlook.com' <epahrfo@outlook.com>;

1 attachments (63 KB)

Summary of decline rate calculations 9.1.17.xlsx;

From: Farrar, Kevin (DEC) [mailto:kevin.farrar@dec.ny.gov]
Sent: Friday, September 01, 2017 2:14 PM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>
Cc: Edwards, Susan L (DEC) <susan.edwards@dec.ny.gov>
Subject: Supplemental Comment on the Hudson River Five Year Review Report

Hello, Gary;

I am sending this email and the attached Excel spreadsheet to supplement the comments provided earlier this week by letter from Commissioner Seggos to Administrator Pruitt.

The attached spreadsheet contains calculations of fish PCB concentrations over time in the upper Hudson River.

I chose the period from 1995 to 2003 for a few reasons: (1) By 1995, the primary IRMs had been in place at the two GE plant sites and the major releases were under control, and (2) the data are all from the DEC monitoring program, which had consistent sampling, processing, and analytical protocols. Due to the change in sampling with the start of the baseline monitoring program in 2004, with new sample locations, new labs, and new sample preparation team, and the lack of a quantified basis (such as use of the Hudson Reference Material or similar standard material) to compare the DEC and GE data to see if they were comparable, I believe a trend analysis using these years was best.

(Perch data were not available for 1995 and 1996.)

Only two locations are available for the DEC monitoring program over this period. The data were looked at from Coveville (now called SW3) and Griffen Island (now called TD5). At each location, four species were evaluated – Pumpkinseed (PKSD), Black Bass, Perch, and Ictalurids (primarily Brown Bullhead). At each location and for each species, a separate calculation was done using the annual mean original DEC Lipid Based PCB concentration, and the annual mean EPA Homologue Equivalent (called here EPA-Transformed).

The data were also looked at by using the simple linear regression tool in excel to generate an equation for each set of data, and rates of decline were generated using that equation. This was done to avoid any possible bias or “luck” in selecting high or low starting or ending data points among the data set.

All in all, 32 rates of decline were generated in the calculation. There was very little difference between the average rates of decline between the rates generated from directly from the data and from the simple regression; there was some difference

between the rates generated using the original vs. the transformed data, with the transformed data typically showing lower rates of decline.

Overall, the data can be summarized as follows:

Mean annual rate of decline in fish LPCB concentrations (data) – 2.45% (4.01% DEC data, 0.88% EPA transformed data)

Mean annual rate of decline in fish LPCB concentrations (regressions) – 2.53% (4.08% DEC data, 0.97% EPA transformed data)

The annual rates of decline ranged from a high of 8.40% (PKSD at Coveville, DEC data), to seven results showing increases in fish LPCB (negative declines).

These results indicate that EPA should take the following into account in finalizing the Five Year Review Report:

1) The observation by EPA that the rates of decline prior to remediation in fish PCB concentrations in the upper Hudson were robust, and were between 12% and 20% is not supported by the DEC fish samples collected prior to remediation.
2) The overall rate of decline in fish PCB concentrations in the upper Hudson prior to remediation was very likely less than 5%, and typically 2-3%.
3) The overall rate of decline in fish PCB concentrations in the upper Hudson prior to remediation do not support the hypothesis that there will be a robust post remedial decline in fish PCB concentrations, or that the rates of decline after remediation will allow for the targeted reductions in fish PCB concentrations to be met.
4) The rates of decline in fish PCB concentrations were properly portrayed in the ROD (see ROD figures 6-2 and 6-3, and the text on page 25, 3rd paragraph).

You can contact me if you have any questions or wish to discuss theses comments or the attached spread sheet. Please include this email and the attached spreadsheet in the administrative record for this site and in the compilation of public comments on the Five Year Review Report.

Thanks,
Kevin

Kevin L. Farrar

Section Chief, Section A/Bureau D, Division of Environmental Remediation

New York State Department of Environmental Conservation

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www.dec.ny.gov |  | 

LPCB in Ictalurids at TD 5 (Griffen Island)

Year	DEC	EPA Transformed
1995	389.98	341.38
1996	449.68	393.65
1997	398.28	441.25
1998	438.89	297.82
1999	348.29	400.18
2000	304.97	474.42
2001	265.83	312.15
2002	278.50	327.04
2003	250.72	294.41
% decline 95-03	35.71%	13.76%
Annual	4.46%	1.72%

LPCB in Ictalurids at SW3 (Coveville)

Year	DEC	EPA Transformed
1995	184.64	161.64
1996	136.15	119.18
1997	154.09	134.89
1998	281.97	235.48
1999	162.01	181.79
2000	203.15	232.68
2001	183.33	215.28
2002	174.49	204.90
2003	96.60	113.44
% decline	47.68%	29.82%
Annual	5.96%	3.73%

LPCB in PKSD at TD 5 (Griffen Island)

Year	DEC	EPA Transformed
1995	176.99	154.91
1996	352.79	308.83
1997		
1998	302.39	230.11
1999	215.35	252.88
2000	223.25	262.15
2001	125.16	146.97
2002	87.77	103.06
2003	231.57	271.92
% decline 9	-30.84%	-75.54%
Annual	-3.86%	-9.44%

LPCB in PKSD at SW3 (Coveville)

Year	DEC	EPA Transformed
1995	273.70	239.60
1996	187.31	163.97
1997		
1998	142.81	105.78
1999	119.16	139.92
2000	129.88	152.52
2001	83.82	98.43
2002	79.15	92.94
2003	128.49	150.89
% decline 9	53.05%	37.03%
Annual	6.63%	4.63%

LPCB in Perch at TD5 (Griffen Island)

Year	DEC	EPA Transformed
1995	x	x
1996	x	x
1997	334.50	297.93
1998	416.95	308.83
1999	344.93	405.03
2000	306.15	359.50
2001	302.11	354.75
2002	265.90	312.24
2003	219.24	257.44
% decline 9	34.46%	13.59%
Annual	5.74%	2.27%

LPCB in Perch at SW3 (Coveville)

Year	DEC	EPA Transformed
1995	x	x
1996	x	x
1997	95.96	91.30
1998	116.31	86.15
1999	164.73	200.42
2000	120.76	141.80
2001	122.16	143.44
2002	135.24	158.80
2003	88.82	104.30
% decline 9	7.44%	-14.25%
Annual	1.24%	-2.37%

LPCB in Black Bass at TD 5 (Griffen Island)

Year	DEC	EPA Transformed
1995	1230.59	1037.08
1996	888.77	778.03
1997	868.88	635.19
1998	1211.43	1147.76
1999	965.56	1082.54
2000	714.24	906.61
2001	875.69	1028.29
2002	881.51	1035.12
2003	682.49	801.42
% decline 9	44.54%	22.72%
Annual	5.57%	2.84%

LPCB in Black Bass at SW3 (Coveville)

Year	DEC	EPA Transformed
1995	636.81	522.85
1996	396.79	347.35
1997	382.03	337.81
1998	575.97	443.45
1999	415.98	618.64
2000	397.81	502.08
2001	503.54	591.29
2002	335.81	394.33
2003	314.19	368.94
% decline 9	50.66%	29.44%
Annual	6.33%	3.68%

LPCB in Ictalurids at TD 5 (Griffen Island)

Year	DEC	EPA Transformed
1995	389.98	341.38
1996	449.68	393.65
1997	398.28	441.25
1998	438.89	297.82
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1998	142.81	105.78
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2002	79.15	92.94
2003	128.49	150.89
% decline 9	53.05%	37.03%
Annual	6.63%	4.63%

LPCB in Perch at TD5 (Griffen Island)

Year	DEC	EPA Transformed
1995	x	x
1996	x	x
1997	334.50	297.93
1998	416.95	308.83
1999	344.93	405.03
2000	306.15	359.50
2001	302.11	354.75
2002	265.90	312.24
2003	219.24	257.44
% decline 9	34.46%	13.59%
Annual	5.74%	2.27%

LPCB in Perch at SW3 (Coveville)

Year	DEC	EPA Transformed
1995	x	x
1996	x	x
1997	95.96	91.30
1998	116.31	86.15
1999	164.73	200.42
2000	120.76	141.80
2001	122.16	143.44
2002	135.24	158.80
2003	88.82	104.30
% decline 9	7.44%	-14.25%
Annual	1.24%	-2.37%

LPCB in Black Bass at TD 5 (Griffen Island)

Year	DEC	EPA Transformed
1995	1230.59	1037.08
1996	888.77	778.03
1997	868.88	635.19
1998	1211.43	1147.76
1999	965.56	1082.54
2000	714.24	906.61
2001	875.69	1028.29
2002	881.51	1035.12
2003	682.49	801.42
% decline 9	44.54%	22.72%
Annual	5.57%	2.84%

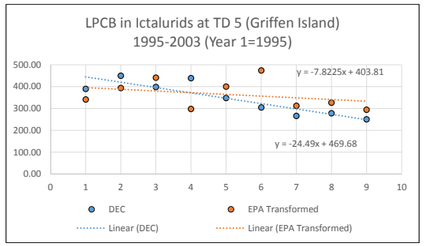
LPCB in Black Bass at SW3 (Coveville)

Year	DEC	EPA Transformed
1995	636.81	522.85
1996	396.79	347.35
1997	382.03	337.81
1998	575.97	443.45
1999	415.98	618.64
2000	397.81	502.08
2001	503.54	591.29
2002	335.81	394.33
2003	314.19	368.94
% decline 9	50.66%	29.44%
Annual	6.33%	3.68%

Regression

LPCB in Ictalurids at TD 5 (Griffen Island)

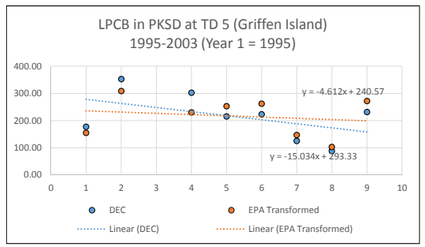
Year		DEC	EPA Transformed	DEC	EPA
	1	389.98	341.38	395.9875	445.19
	2	449.68	393.65		
	3	398.28	441.25		
	4	438.89	297.82		
	5	348.29	400.18		
	6	304.97	474.42		
	7	265.83	312.15		
	8	278.50	327.04		
	9	250.72	294.41	333.4075	249.27
% decline		35.71%	13.76% Linear	15.80%	44.01%
Annual		4.46%	1.72% Annual	1.98%	5.50%



Regression

LPCB in PKSD at TD 5 (Griffen Island)

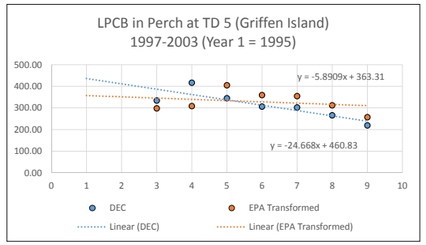
Year		DEC	EPA Transformed	DEC	EPA
	1	176.99	154.91	278.296	235.958
	2	352.79	308.83		
	3				
	4	302.39	230.11		
	5	215.35	252.88		
	6	223.25	262.15		
	7	125.16	146.97		
	8	87.77	103.06		
	9	231.57	271.92	158.024	199.062
% decline		-30.84%	-75.54%	43.22%	15.64%
Annual		-3.86%	-9.44%	5.40%	1.95%



Regression

LPCB in Perch at TD5 (Griffen Island)

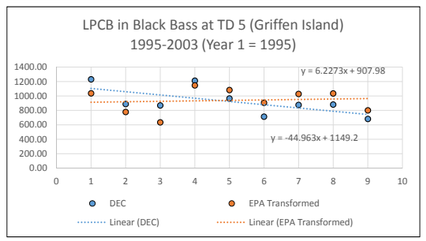
Year		DEC	EPA Transformed	DEC	EPA
	1			436.162	357.4191
	2				
	3	334.50	297.93		
	4	416.95	308.83		
	5	344.93	405.03		
	6	306.15	359.50		
	7	302.11	354.75		
	8	265.90	312.24		
	9	219.24	257.44	238.818	310.2919
% decline		34.46%	13.59%	45.25%	13.19%
Annual		5.74%	2.27%	5.66%	1.65%



Regression

LPCB in Black Bass at TD 5 (Griffen Island)

Year		DEC	EPA Transformed	DEC	EPA
	1	1230.59	1037.08	1104.237	914.2073
	2	888.77	778.03		
	3	868.88	635.19		
	4	1211.43	1147.76		
	5	965.56	1082.54		
	6	714.24	906.61		
	7	875.69	1028.29		
	8	881.51	1035.12		
	9	682.49	801.42	744.533	964.0257
% decline		44.54%	22.72%	32.57%	-5.45%
Annual		5.57%	2.84%	4.07%	-0.68%

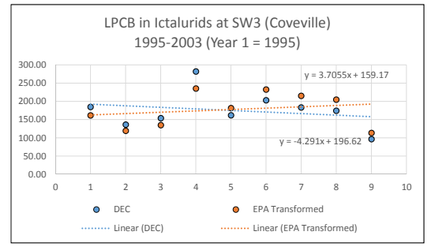


(Year 1 = 1995)

Regression

LPCB in Ictalurids at SW3 (Coveville)

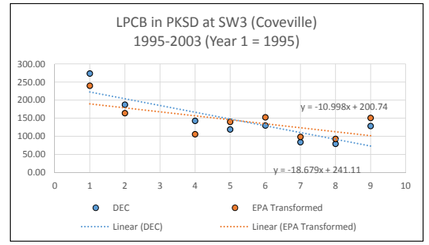
Year		DEC	EPA Transformed	DEC	EPA
	1	184.64	161.64	188.04	166.58
	2	136.15	119.18		
	3	154.09	134.89		
	4	281.97	235.48		
	5	162.01	181.79		
	6	203.15	232.68		
	7	183.33	215.28		
	8	174.49	204.90		
	9	96.60	113.44	158.00	192.52
% decline		47.68%	29.82%	15.97%	-15.57%
Annual		5.96%	3.73%	2.00%	-1.95%



Regression

LPCB in PKSD at SW3 (Coveville)

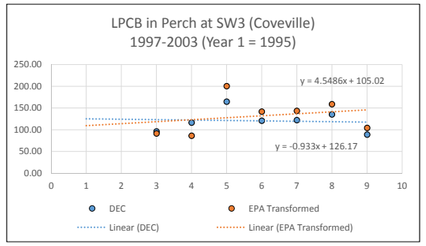
Year		DEC	EPA Transformed	DEC	EPA
	1	273.70	239.60	222.43	189.74
	2	187.31	163.97		
	3				
	4	142.81	105.78		
	5	119.16	139.92		
	6	129.88	152.52		
	7	83.82	98.43		
	8	79.15	92.94		
	9	128.49	150.89	73.00	101.76
% decline		53.05%	37.03%	67.18%	46.37%
Annual		6.63%	4.63%	8.40%	5.80%



Regression

LPCB in Perch at SW3 (Coveville)

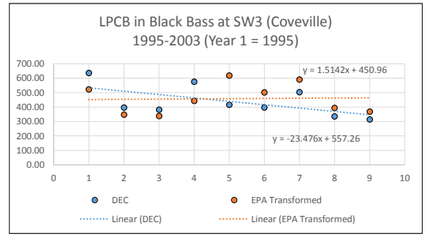
Year		DEC	EPA Transformed	DEC	EPA
	1			125.237	109.5686
	2				
	3	95.96	91.30		
	4	116.31	86.15		
	5	164.73	200.42		
	6	120.76	141.80		
	7	122.16	143.44		
	8	135.24	158.80		
	9	88.82	104.30	117.773	145.9574
% decline		7.44%	-14.25%	5.96%	-33.21%
Annual		1.24%	-2.37%	0.74%	-4.15%



Regression

LPCB in Black Bass at SW3 (Coveville)

Year		DEC	EPA Transformed	DEC	EPA
	1	636.81	522.85	533.784	452.4742
	2	396.79	347.35		
	3	382.03	337.81		
	4	575.97	443.45		
	5	415.98	618.64		
	6	397.81	502.08		
	7	503.54	591.29		
	8	335.81	394.33		
	9	314.19	368.94	345.976	464.5878
% decline		50.66%	29.44%	35.18%	-2.68%
Annual		6.33%	3.68%	4.40%	-0.33%



Calculations using DEC LPCB data and EPA Transformed LPCB data

Species	LPCB - Basis	Location	Annual % decline	Period of observation
Ictalurids	DEC	Griffen Island	4.46%	95-03
Ictalurids	EPA	Griffen Island	1.72%	95-03
Ictalurids	DEC	Coveville	5.96%	95-03
Ictalurids	EPA	Coveville	3.73%	95-03
Black Bass	DEC	Griffen Island	5.57%	95-03
Black Bass	EPA	Griffen Island	2.84%	95-03
Black Bass	DEC	Coveville	6.33%	95-03
Black Bass	EPA	Coveville	3.68%	95-03
Perch	DEC	Griffen Island	5.74%	97-03
Perch	EPA	Griffen Island	2.27%	97-03
Perch	DEC	Coveville	1.24%	97-03
Perch	EPA	Coveville	-2.37%	97-03
PKSD	DEC	Griffen Island	-3.86%	95-03
PKSD	EPA	Griffen Island	-9.44%	95-03
PKSD	DEC	Coveville	6.63%	95-03
PKSD	EPA	Coveville	4.63%	95-03
		Mean	2.45%	

Calculations using DEC LPCB data

Species	LPCB - Basis	Location	Annual % decline	Period of observation
Ictalurids	DEC	Griffen Island	4.46%	95-03
Ictalurids	DEC	Coveville	5.96%	95-03
Black Bass	DEC	Griffen Island	5.57%	95-03
Black Bass	DEC	Coveville	6.33%	95-03
Perch	DEC	Griffen Island	5.74%	97-03
Perch	DEC	Coveville	1.24%	97-03
PKSD	DEC	Griffen Island	-3.86%	95-03
PKSD	DEC	Coveville	6.63%	95-03
		Mean	4.01%	

Calculations using EPA Transformed LPCB data

Species	LPCB - Basis	Location	Annual % decline	Period of observation
Ictalurids	EPA	Griffen Island	1.72%	95-03
Ictalurids	EPA	Coveville	3.73%	95-03
Black Bass	EPA	Griffen Island	2.84%	95-03
Black Bass	EPA	Coveville	3.68%	95-03
Perch	EPA	Griffen Island	2.27%	97-03
Perch	EPA	Coveville	-2.37%	97-03
PKSD	EPA	Griffen Island	-9.44%	95-03
PKSD	EPA	Coveville	4.63%	95-03
		Mean	0.88%	

Calculations using simple linear regressions of DEC LPCB data and EPA Transformed LPCB data

Species	LPCB - Basis	Location	Annual % decline	Period of observation
Ictalurids	DEC	Griffen Island	1.98%	95-03
Ictalurids	EPA	Griffen Island	5.50%	95-03
Ictalurids	DEC	Coveville	2.00%	95-03
Ictalurids	EPA	Coveville	-1.95%	95-03
Black Bass	DEC	Griffen Island	4.07%	95-03
Black Bass	EPA	Griffen Island	-0.68%	95-03
Black Bass	DEC	Coveville	4.40%	95-03
Black Bass	EPA	Coveville	-0.33%	95-03
Perch	DEC	Griffen Island	5.66%	97-03
Perch	EPA	Griffen Island	1.65%	97-03
Perch	DEC	Coveville	0.74%	97-03
Perch	EPA	Coveville	-4.15%	97-03
PKSD	DEC	Griffen Island	5.40%	95-03
PKSD	EPA	Griffen Island	1.95%	95-03
PKSD	DEC	Coveville	8.40%	95-03
PKSD	EPA	Coveville	5.80%	95-03
		Mean	2.53%	

Calculations using simple linear regressions of DEC LPCB data

Species	LPCB - Basis	Location	Annual % decline	Period of observation
Ictalurids	DEC	Griffen Island	1.98%	95-03
Ictalurids	DEC	Coveville	2.00%	95-03
Black Bass	DEC	Griffen Island	4.07%	95-03
Black Bass	DEC	Coveville	4.40%	95-03
Perch	DEC	Griffen Island	5.66%	97-03
Perch	DEC	Coveville	0.74%	97-03
PKSD	DEC	Griffen Island	5.40%	95-03
PKSD	DEC	Coveville	8.40%	95-03
		Mean	4.08%	

Calculations using simple linear regressions of EPA Transformed LPCB data

Species	LPCB - Basis	Location	Annual % decline	Period of observation
Ictalurids	EPA	Griffen Island	5.50%	95-03
Ictalurids	EPA	Coveville	-1.95%	95-03
Black Bass	EPA	Griffen Island	-0.68%	95-03
Black Bass	EPA	Coveville	-0.33%	95-03
Perch	EPA	Griffen Island	1.65%	97-03
Perch	EPA	Coveville	-4.15%	97-03
PKSD	EPA	Griffen Island	1.95%	95-03
PKSD	EPA	Coveville	5.80%	95-03
		Mean	0.97%	

OFFICE OF THE COMMISSIONER

New York State Department of Environmental Conservation
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June 7, 2017

Ms. Catherine McCabe
Acting Regional Administrator, Region 2
United States Environmental Protection Agency
290 Broadway
New York, NY 10007-1866

Dear Administrator McCabe,

I write to convey New York State's immediate concerns with the US Environmental Protection Agency's (USEPA) recently released Five Year Review report on the Hudson River PCBs site, and more specifically, to formally request an extension of the public comment period to 90 days.

The New York State Department of Environmental Conservation (DEC) is in the process of carefully reviewing the nearly one thousand pages of documents associated with the draft Five Year Review report and will provide a detailed response once our review is complete. However, a thirty day comment period is insufficient to provide for a careful analysis of the information and respectfully urge for this comment period to be extended.

DEC steadfastly maintains a significant amount of PCB contamination remains in the Hudson River and rejects USEPA's conclusion that the remedy is protective of public health and the environment. To be clear, DEC has concluded that USEPA-led cleanup of the Hudson River is incomplete and the findings of the USEPA's draft Five Year Review Report are unacceptable and not based on science.

Current data continues to show that significant PCB contamination is still present in the River posing a threat to human health and the environment. Based on this information, the EPA should amend its conclusion in this five year review and find that the current remedy is not protective of public health and the environment. Furthermore, in light of USEPA's refusal to work with DEC to conduct additional sampling of the River to adequately determine the effectiveness of the cleanup, New York State is moving forward on its own with a sampling program this summer.

We appreciate your consideration of this request for an extension of the comment period, and will be providing additional detailed comments outlining our concerns on the five year report in the near future.

Sincerely,

Basil Seggos
Commissioner

c: Administrator Scott Pruitt

FW: Correspondence from Commissioner Basil Seggos Re: Hudson River Five Year Review

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Wed 9/6/2017 9:50 AM

To: 'epahrfo@outlook.com' <epahrfo@outlook.com>;

1 attachments (3 MB)

Aug30_2018.PruittLtr..pdf;

From: Farrar, Kevin (DEC) [mailto:kevin.farrar@dec.ny.gov]
Sent: Wednesday, August 30, 2017 3:03 PM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>
Subject: FW: Correspondence from Commissioner Basil Seggos Re: Hudson River Five Year Review

Hello, Gary;

See attached FYI.

I listened to your voice mail but was unable to leave you a message (full mailbox). However, in response to your questions, sometimes folks speak beyond their brief.

See you tomorrow,
Kevin

OFFICE OF THE COMMISSIONER

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August 30, 2017

Mr. Scott Pruitt
Administrator
Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Dear Administrator Pruitt:

I am sending this letter to provide the United States Environmental Protection Agency (EPA) with comments of the New York State Department of Environmental Conservation (DEC) to EPA's "Proposed Second Five Year Review Report for Hudson River PCBs Superfund Site," dated May 31, 2017.

EPA states in the proposed report that the remedy will not achieve its ultimate objective for the foreseeable future, and will only become protective "at some point" in time more than 55 years from now. This is unacceptable – a remedy that will take generations to safeguard public health and the environment is clearly not protective. It is also not what the people of the State of New York were promised when EPA announced its remedial decision for the Hudson River in 2002. At that time, EPA predicted that the dredging remedy would result in rapid reductions in PCB levels in fish so that fish consumption restrictions could be relaxed in five to ten years, as opposed to many decades as is now predicted.

Moreover, despite DEC calling for EPA to conduct additional sampling, EPA has disregarded the need for more data to determine the effectiveness of the remedy. EPA appears desperate to come to a conclusion which simply is not supported by the current conditions of the Hudson River. It is obvious that the remedy is not protective of public health and the environment.

As described in the enclosed technical commentary, and as stated in the DEC report provided during EPA's five-year review process with my December 20, 2016 letter, DEC disagrees with EPA's proposed protectiveness determination for this site. The most important criterion for evaluating protectiveness is the degree of human health and ecological risk posed by the site. EPA is fully aware that the current human health and ecological risks in both the Upper Hudson River, where the remedial work was done between Fort Edward and Troy, and the Lower Hudson River, south of the Federal Dam at Troy, are well in excess of EPA's acceptable risk range. Given the current and anticipated conditions for this site, along with EPA's own guidance on protectiveness determinations, the only reasonable conclusion that can be reached is that the remedy is "Not Protective."

EPA should follow the process laid out in the 2002 Record of Decision (ROD) for this site. The remedy selected by EPA in the ROD called for targeted environmental dredging, followed by "Monitored Natural Recovery." While EPA recognized that some PCBs would be left behind in the river, EPA erroneously estimated that a sufficient amount of contaminated sediment would be removed to allow for gradual natural processes, such as the influx of cleaner sediments into the system, to reach the remedial targets identified in the ROD for rapid reductions in human health and environmental risk.

However, because greater levels of PCBs were found in the river both during project design, and again during project implementation, significantly more PCBs were left behind than was intended when EPA selected remedy. The additional sediment sampling that DEC has nearly completed (after EPA and General Electric (GE) refused to take action) will quantify how much contamination was left behind. EPA has never considered adjusting the remedial work to take the increases in known PCB mass into account, and has not provided any satisfactory scientific rationale for dismissing such consideration. As a result, it is a near certainty that the targeted reductions in fish PCB concentrations required by the ROD will not be met throughout the Upper Hudson River in the near term. Rather, as described in EPA's previous five-year review report in 2012,[1] there will likely be delays in recovery as a result of more PCBs being left behind than anticipated.

EPA should perform the data gathering and analyses necessary to confirm the assumption being made by EPA that the amount of remedial work done to date will be sufficient to reach the remedial targets set in the ROD, the first of which is to be met in 2020. As described in the enclosed detailed comments on the proposed report, there is no valid reason for EPA to modify or abandon the targets for reductions of PCB levels in fish from the ROD. EPA's unwillingness to fulfill its commitments to New Yorkers is unacceptable.

Furthermore, if the targets are not to be met, EPA must direct that sufficient additional remedial work be done. To date, EPA's persistent refusal to collect and analyze a full array of data has run counter to EPA's original commitment to clean up the site. In order to perform the necessary evaluations, EPA should ensure the collection of sufficient water, sediment, and fish data to fully assess whether the remedy will meet the targets in the ROD, starting with the initial target of 0.4 ppm PCBs in fish by 2020. As indicated above, DEC raised the need for EPA to conduct additional sediment sampling in November of 2016. EPA formally rejected that request in December of 2016. DEC then took the necessary steps to begin taking its own sediment samples over the summer. Similarly, if EPA refuses to conduct additional fish sampling, DEC will do so.

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[1] First Five Year Report for the Hudson River PCBs Superfund Site, June 1, 2012 available at https://www3.epa.gov/hudson/plans.html

As you know, the targets in the ROD for rapid reductions in human health and ecological risk were the primary bases upon which EPA justified the dredging remedy. These same targets were the primary bases for the State to concur that the remedy would be protective of public health and the environment. EPA rejected (as the State rejected) remedial alternatives which would have resulted in delays in recovery of 10 or more years, as EPA recognized at the time of the ROD that the controls on risk, such as fish consumption advisories, provide insufficient protection to human health in the long term, and provide no protection to ecological resources. These principles are as true today as they were at the time of remedy selection. EPA should not rely on only partial controls on risk as justification for not performing any further necessary remedial work on this site.

I remain very concerned that EPA has abandoned its responsibilities under CERCLA to protect public health and the environment by failing to perform a complete Remedial Investigation for the portion of the site south of the Troy Dam. The Lower Hudson River is contaminated with PCBs from the Upper Hudson River throughout the entire Hudson River estuary south to New York Harbor. Human health and ecological risks associated with Lower Hudson River fish consumption are outside of EPA's acceptable risk range.

EPA has acknowledged in its May release of the proposed five-year review report that the remedial work conducted in the Upper Hudson River to date will not result in any significant reductions in public health and environmental risks. There is no longer any reason to delay the Lower Hudson River investigation and EPA should immediately ensure that it is undertaken.

I understand that EPA currently plans to end the public comment period on September 1. As noted above, DEC disagrees with EPA's current recommendation and finds that sufficient data exists to determine that the remedy is not protective. In addition, DEC believes that the data from the sampling we are currently conducting will further support this conclusion and will provide guidance on how to meet the goals of EPA's approved remedy. We will provide the results of this initiative this fall.

Enclosed to this letter is a set of general comments, and a set of more detailed technical comments on the proposed report and appendices. Please place this letter and attachments, my December 20, 2016 letter and attachments and any additional technical comments provided by staff, in the administrative record for this site. I look forward to receiving EPA's response to DEC's comments.

Sincerely,

Basil Seggos
Commissioner

Enclosure

NYSDEC General Comments on EPA's Proposed Hudson River Five Year Review Report

General Comment 1:

The Protectiveness Determination should be "Not Protective" for the river bottom remedy in the Hudson River between Hudson Falls and Troy (Operable Unit 2).

EPA's Five-Year Review guidance sets forth three critical questions that must be addressed for EPA to make a "Protectiveness" determination: (A) Is the remedy functioning as intended by the decision documents; (B) Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid; and (C) Has any other information come to light that could call into question the protectiveness of the remedy?

The DEC document provided to EPA on December 20, 2016 combined with these comments shows that, after answering the three critical questions above, the only appropriate protectiveness determination for this Five Year Review is "Not Protective" for the river bottom remedy being implemented by EPA for the Hudson River PCBs site. The current level of human health and ecological risk throughout the entire site is in excess of EPA's acceptable risk range, including in the Upper Hudson, which is the fundamental metric that justifies this finding.

DEC provided a detailed rationale in our December 2016 document and it is not necessary to repeat it here. Data which has become available since December 2016 (the small sediment data set gathered by GE at EPA's direction in late 2016) and the 2016 fish PCB data, do not indicate that the current conditions in the Hudson River are protective. Rather, these data – particularly the fish data, which demonstrates fish PCB concentrations which give rise to human health and ecological risks above EPA's acceptable risk range - support DEC's primary contention that the current state of the Hudson River remedy for the contaminated sediments of the Upper Hudson is "Not Protective".

It is also important to point out that, given the current PCB concentrations in sport fish in the Upper Hudson, it is extremely unlikely that the fish PCB concentrations in the Upper Hudson will achieve the ROD targets for fish PCB recovery in the Upper Hudson, the first of which was to be met within five years after dredging was completed (2020). The most recent data (from 2016) indicate that the reach and species weighted average is 1.25 parts per million PCB. With the dredging completed in 2015, and the targeted concentration being 0.4 parts per million five years after dredging is completed, it will take fifteen years at the anticipated natural recovery rate of 8% per year to reach the first target. It would take natural recovery rates of over 20% to reach the first target in the five year time frame specified in the ROD, which is unrealistic and highly unlikely.

NYSDEC General Comments on EPA's Proposed Hudson River Five Year Review Report

General Comment 2:

EPA must follow its own guidance for issuing five year reviews and making protectiveness determinations. EPA's own guidance prevents EPA from issuing a "not yet protective but will be protective" determination after a remedy has been constructed.

The EPA Guidance on Five Year Reviews and Protectiveness Determinations (See Comprehensive Guidance on Five Year Reviews (EPA July 2001) and Clarifying the Use of the Protectiveness Determination (EPA September 2012)) describe in detail how EPA should conduct Five Year Reviews for all NPL sites across the nation. Nowhere in this guidance is it contemplated that a site which has a constructed remedy could receive a protectiveness determination of "not yet protective but will be protective". EPA is violating its own guidance by making up a new category of protectiveness which has never been employed or contemplated in the sixteen years that the Agency has had guidance on Five Year Reviews.

As noted above, EPA must also answer the question if any other information has come to light which would question the effectiveness of the remedy. EPA appears to be ignoring or downplaying all of the information which has become available after remedy selection that calls into question it's modelling and predictions, contrary to the Five Year Review guidance.

General Comment 3:

EPA appears to be abandoning the ROD targeted reductions in fish PCB concentrations that formed the basis for justifying the dredging remedy, and in doing so is arbitrarily ignoring critical questions A & B in its own Five Year Review guidance.

In the proposed five year review report, EPA is now stating that the remedy will not be protective until the ultimate remedial goal of 0.05 parts per million PCB in fish is reached. DEC urges EPA to enforce the selected remedy in the ROD and take the actions necessary to ensure that the remedy achieves the targeted rapid reductions in fish PCB levels, and thus human health and environmental risk, identified in the ROD. The ROD identified that the remedy would achieve the first target (0.4 parts per million or ppm PCB in average fish concentrations) within five years after dredging, and 0.2 ppm in sixteen years. This was the primary basis upon which EPA justified the dredging remedy, and the primary basis for the State's concurrence with the remedy.

EPA also identified these targets as representing, in EPA's view, points where there would be opportunity for the fish consumption advisories to be modified. However, EPA now appears to be pointing only to the ultimate remedial goal of 0.05 parts per million in fish, and no longer appears to be prepared to manage the remedy to achieve the ROD targets.

These ROD targets, representing rapid significant reductions in fish PCB concentrations and thus human health and environmental risk, were the primary basis used by EPA to justify the dredging remedy and the primary basis upon which the State concurred that the remedy would be protective of human health and the environment. EPA has provided no valid justification in its proposed report for abandoning these targets, other than being content to have a "wait and see" approach, while exposing the people and environment of New York State to unacceptable risks for many decades.

EPA should manage the remedy for this site so that the remedial targets identified in the ROD are achieved, and not focus solely on the ultimate remedial goal, which would only be achieved several generations into the future regardless of whether the remedial work was done or not.

General Comment 4:

EPA needs to follow the ROD and adaptively manage this remedial action, now in the "Monitored Natural Recovery" phase with dredging completed in 2015 and habitat reconstruction (planting) completed in 2016.

EPA should recognize that the remedy in the ROD represented EPA's best estimate as to how much remedial work would be necessary to meet the targets and goals set in the ROD. This estimate, based upon the tools available at the time, is what led EPA to determine the extent of remediation necessary for the rapid reductions in fish PCB concentrations, and thus human health and environmental risk, identified in the ROD.

At the present time, with the data from the project design and construction being available over the fifteen years since the ROD was issued, EPA now needs to update the site conceptual model, and gather the data necessary to determine if the amount of remedial work identified in the ROD will achieve the targeted reductions in human health and environmental risk.

DEC asserts that it is likely that further remedial work would be necessary to achieve the targeted reductions in fish PCB concentrations. More PCB was left behind than anticipated, and the most recent fish PCB concentrations indicate that it will likely take unrealistically high natural recovery rates to reach the targeted fish PCB concentrations, the first of which is to be reached five years after dredging.

In order for the remedy to be protective, the remedy should be managed to meet the targets set in the ROD. If the targets are not going to be met, then EPA should adjust the amount of remedial work done, not abandon the targets.

NYSDEC General Comments on EPA's Proposed Hudson River Five Year Review Report

General Comment 5:

EPA must update the agency's understanding of how the PCBs remaining in Hudson River sediments impact the water column and fish in the river.

There have been several important findings since the ROD was issued as it pertains the understanding of the distribution of PCB in Hudson River sediments, and how they impact water column and fish. EPA needs to update this understanding (called the "conceptual site model") to take these findings into account.

First, during the Sediment Sampling and Analysis Program (SSAP), the major data gathering program during project design (done mostly in 2002-2005), much more PCB mass in the river was found than previously thought, and much of this additional mass was closer to the surface.

Second, during implementation of the dredging program, it was again found that in certain areas of the river where woody debris had accumulated, there was significantly more PCB at depth than initially found in the SSAP, due to the debris preventing adequate sampling depth.

Third, the impact of the dredging work on the fish clearly shows that the increase in water column PCB concentration did not have a commensurate impact on the fish in the Hudson River. Typically, only those fish in the immediate vicinity of the dredging work, or immediately downstream, showed a significant reaction to the dredging. This indicates to DEC that the local sediments are much more important in controlling fish PCB concentrations than impacts from upstream sources, which in the Hudson River primarily means upstream sediments. This is most important for the Lower Hudson River, where the fish showed little to no response to the dredging work upstream, and it can no longer be expected that the remedial program in the Upper Hudson will result in significant improvement in fish PCB concentrations south of Albany.

EPA is again using overly optimistic model projections anticipating rates of natural recovery which are likely higher than what is happening in the river.

DEC raised this issue in a July 31, 2000 letter to EPA Region 2 during remedy selection, stating that this overly optimistic view of natural recovery rates (due to underrepresentation of the relative impacts of the sediments on fish) likely understated the benefits of active remediation. EPA understood that there was uncertainty in the modeling effort and, with that understanding, still set the targets in the ROD for protectiveness. EPA is again using the same model, which likely again underestimates the impacts of the local sediments on the fish, and is again likely underestimating the impact of the remaining contaminated sediments on the fish.

If EPA continues to use modeling for this site, as the ROD directs, then it will be necessary for EPA to restructure and recalibrate the model to reflect an updated conceptual site model which properly takes into account what has been learned since the ROD was issued. EPA has never given DEC or the public a valid scientific reason for not updating its modeling and flawed predictions.

General Comment 6:

EPA needs to develop and implement a monitoring plan which is designed to quantify the performance of the remedy at the temporal and spatial scale necessary to understand the remedy performance in a time frame commensurate with the time to reach the targeted rapid reductions in fish PCB concentrations.

DEC believes that the data gathering should be sufficient to understand, with the appropriate degree of statistical certainty, if the remedy is meeting the anticipated recovery rates in the time to reach the first target in year 2020.

To ensure statistical certainty, and to avoid missing differences in remedy performance in one area of the river as compared to another, the data gathering must be done on a spatial scale commensurate with the exposure driving the fish PCB concentrations. As the fish generally do not move between pools (the reaches of river separated by locks and dams), the fish will be impacted primarily by the sediments in the pool where they live. Sediments in Schuylerville will not drive PCB concentrations in fish at Waterford, some twenty-five miles downstream, and sediments in Waterford certainly will not drive PCB concentrations in Schuylerville. EPA's current approach would average between large reaches of river, restricting any ability to discern the actual performance of the remedy at the scale where the exposure occurs.

DEC has identified the data gathering which is necessary to understand the performance of the remedy, and urges EPA to follow the recommendations provided by DEC over a year ago.

General Comment 7:

Moving forward, DEC urges EPA to recognize that there is much more work to be accomplished to address the human health and ecological risk posed by the disposal of PCBs in the Hudson River. EPA should do the work necessary to ensure that the remedy in the Upper Hudson is protective, and to implement a full investigation and remedial program in the Lower Hudson south of Troy.

EPA should acknowledge that the remedy is currently not protective of human health or the environment, as the current risks are beyond EPA's acceptable range. EPA should collect the monitoring data necessary to quantitatively evaluate remedy performance as compared to the ROD targets, and to gather the data necessary to determine how to

modify the remedial work should the data indicate that the remedy will not or is not meeting the targets. In short, should the remedy not meet the targets, EPA should modify the remedy, not change the targets to make it appear to be protective.

EPA also should immediately exercise the authority to implement a comprehensive Remedial Investigation of the Lower Hudson River. The entire estuarine portion of the river south of the Troy Dam is contaminated with PCBs from the Upper Hudson, and EPA no longer expects the remedial program in the Upper Hudson to have much impact on the Lower Hudson River, particularly in the area south of Albany. In the meantime, EPA has no plans to move forward with an investigation of the distribution and impacts of the PCB contamination already present in the Lower Hudson transported from the Upper Hudson.

There is no reason to wait – this portion of the river is already part of the "National Priorities List" site. EPA already has the authority to issue an order to GE to implement a Remedial Investigation and Feasibility Study to determine the nature and extent of PCBs throughout the Lower Hudson, and to evaluate remedial actions needed to abate the human health and ecological risks that EPA currently recognizes as above the acceptable risk range.

Detailed Technical Comments - Attached

DEC has performed a detailed review of the document text and has evaluated the scientific information and assessments presented in the appendices. Attached to this letter is a list of detailed comments on the report. Please provide DEC with a written response to the issues raised in this letter and the attached comments, as well as to the written comments first provided in December 2016 and to the statements read at the public meeting and provided to EPA. DEC is, as always, prepared to meet with EPA to help advance the remedial program for the Hudson River and work toward our common goal of abating the human health and ecological risk caused by the disposal of PCB in the Hudson River.

NYSDEC's Detailed Comments – Five Year Review Report Text and Appendices

#	Report citation	Issue	Quote from document	Comment / Discussion	References
1	Executive Summary, page 1	Errata	"The purpose of this second five-year review (FYR) is to determine whether the remedial actions at the Hudson River PCBs Superfund Site (Site) are protective of public health and the environment and functioning as designed."	This is actually the third five year review for this site overall; the third for OU1, and second for OU2. The initial review of the remedy selected in the 1984 ROD was started in 1989 and culminated in the 2002 ROD for OU2.	
2	Executive Summary, page 2	Models	"Although these recent data present some encouraging results, further monitoring will be required to verify remedy effectiveness, but the analyses presented in this report demonstrate that the models used to support decision making were well-designed, remedial action objectives (RAOs) were appropriately developed, and remedy implementation is proceeding as planned."	USEPA states here that: (1) the models used in remedy selection were "well designed", (2) that the RAOs were "appropriately developed", and (3) remedy implementation is "proceeding as planned". This contradicts later statements that the model projections for reaching the interim targets can not be relied upon, and that the interim targets identified as RAOs should no longer apply.	
3	Executive Summary, Page 4	Reductions in Surface Sediment and Fish PCBs	"Available surface sediment data in conjunction with fish and water column concentrations indicate that surface sediment PCB concentrations are decreasing with time. The reduction in surface sediment concentration associated with dredging alone by river section was 87%, 36%, and 5% in River Sections 1, 2, and 3, respectively. Although the reduction associated with dredging in River Section 2 (RS2) was less than expected and may cause a lag in recovery, the overall surface sediment reduction is within ROD expectations."	EPA and DEC agree that there should be a close relationship between surface sediment PCB concentrations and fish PCB concentrations, particularly at a local level. In order to achieve the desired reductions in fish PCB concentrations, a commensurate reduction in sediment PCB concentrations must be achieved. The RAO interim targets for reach and species averaged fish PCB concentrations are to reach 0.4 ppm total PCB five years after dredging is completed. However, EPA's report states that only at 22% reduction in surface sediment PCB concentrations on a River Section length weighted average. As a result, it is unlikely that natural processes will be able to result in sufficient improvement to allow for surface sediments, and thus fish, to reach the ROD targets for reductions in PCB concentrations over time. The 2016 river section and species weighted average fish PCB concentration is, according to EPA, 1.25 ppm; it would take 25% reductions annually to reach 0.4 ppm by 2020, five years after dredging. At 8% per year (the model projected post dredging recovery rate), at the current fish PCB levels it would take 15 years to reach 0.4 ppm, and 23 years to reach 0.2 ppm. EPA would have had to achieve reductions due to dredging down to 0.6 ppm for an 8% improvement rate per year to reach 0.4 ppm five years after dredging. This equates to a reduction from 2.15 ppm (the 2004-	River Section 1 – 6 miles / 87% reduction River Section 2 – 6 miles / 36% reduction River Section 3 – 28 miles / 5% reduction [(6x0.87)+(6x0.36)+(28x0.05)]/40= 21.95% River Section Length Weighted Average reduction in surface sediment PCB concentrations

				2008 BMP mean) to 0.6 ppm, or a 72% reduction. It is not realistic to anticipate a 72% reduction in fish PCB concentrations with only at 22% reduction in surface sediment PCB concentrations.	
4	Executive Summary, Page 4	PCB mass reduction	Total PCB and Tri+ PCB mass removed were greater than planned, due to underestimates of the depth of contamination during the original remedial design. PCB mass in non-dredged areas is also greater than estimated in the 2002 ROD, although to a lesser extent than within the dredged areas. As calculated by EPA, the volume of sediment, mass of total PCBs, and mass of Tri+ PCBs removed during both Phases 1 and 2 were approximately 2,642,000 cubic yards of sediment, 155,800 kg of TPCBs, and 48,600 kg of Tri+ PCBs, respectively.	EPA here focuses on the amount of PCB removed; conditions in the river after dredging are not controlled by what was removed, but rather by what was left behind. EPA should not focus on the comparison of what was removed as compared to what was anticipated to be removed, as it is not relevant to the evaluation of whether or not the remedy is protective. Protectiveness is determined by evaluating the current site risks, and comparing them to the acceptable risk range. The current site risks are well above the acceptable risk range, and as a result the remedy is currently not protective. The amount of PCB left behind is much greater than anticipated, resulting (as EPA stated in 2012) a delay in reaching the remedial action objectives.	
5	Executive Summary, Page 4	Habitat reconstruction	Habitat replacement and reconstruction was conducted as anticipated. OM&M of restored habitat will continue until project objectives are met.	As discussed between DEC and EPA over the past several years, DEC believes that EPA has not required GE to perform sufficient habitat reconstruction to allow for the work to reach the habitat reconstruction goals. While not specifically relevant to the protectiveness determination, DEC will provide to EPA specific areas where further habitat reconstruction work is necessary to meet the habitat reconstruction goals.	
6	Executive Summary, Page 5	Model Forecast accuracy	Monitored natural attenuation is occurring and rates of decline are generally in agreement with the modeling done for the ROD:	EPA is overstating the actual observed rates of natural recovery which were ongoing at the time of remedy selection and design. "General agreement" is not a quantitative comparison; a more detailed quantitative analysis, taking into account the uncertainty associated with using different data sets over time, needs to be performed.	
7	Executive Summary, Page 5	Model Forecast accuracy - water	For the pre-dredging MNA period (1995-2008), water column Tri+ PCB concentrations declined at rates ranging from approximately 5 to 13 percent per year at the four Upper Hudson	Unfortunately, there are changes in stations, sampling methodology, and changes in flow regimes which complicate this analysis. EPA should properly account for these sources of	

			monitoring stations, and HUDTOX model simulations for this period were generally faithful to both seasonal and long-term trends.	variability and not rely upon the water data to support excessive rates of recovery in this document.	
8	Executive Summary, Page 5	Model Forecast accuracy - fish	Fish tissue concentrations declined during the pre-dredging MNA period (1995-2008). Rates of decline in the Upper Hudson for wet weight and lipid-normalized fish tissue PCB concentrations were approximately 12 to 20 percent per year and approximately 8 percent per year, respectively, consistent with rates estimated from the FISHRAND model output. Lower rates of decline were observed at locations farther downstream in the Lower Hudson River.	EPA should not be using the wet weight PCB data from the time period after 2005, as GE has admitted that their lab did not follow the acceptable and approved sample preparation protocol, introducing a significant negative bias and high variability to the wet weight PCB fish data. EPA knows this and should not have used this data in their understanding of site conditions. The lipid based PCB concentrations, while biased low for these years, is not biased to the degree as being unusable; however, data users should understand that the later BMP, and subsequent RAMP fish data (until 2015) are biased low. As a result, the estimates of natural recovery are biased high (overstating the rate of recovery) since the earlier data are without this bias, and the later data are biased low.	
9	Executive Summary, Page 5	Model Forecast accuracy - sediment	Available surface sediment data in conjunction with fish and water column concentrations indicate that surface sediment PCB concentrations are decreasing with time. Although the exact rate of decline is difficult to determine, as there is no single consistent sediment data set, the results using the available data indicate a decay rate similar to that predicted at the time of the ROD.	DEC has requested that EPA gather the sediment data necessary to quantify the change in surface sediment PCB concentrations over time at a scale (pool by pool) and in a time frame (commensurate with the remedial targets in the ROD) needed to evaluate remedy performance. EPA has thus far refused to do so, and as a result DEC has begun gathering the needed sediment data starting in Summer 2017.	
10	Executive Summary, Page 6	Monitoring recovery in fish PCB concentrations	2016 fish data suggest that fish have begun to recover from dredging impacts and are generally declining. It is important to recognize that up to 8 or more years of fish tissue data may be necessary to draw statistically based conclusions about trends, with a high degree of confidence, depending on the actual rate of decline that is experienced (it is anticipated that it will require approximately 8 years for fish tissue to decline to 50% of its current PCB concentration based on	It is not necessary to wait eight years to have the data necessary to determine if the fish PCB concentrations are reducing at a rate sufficient to achieve the remedial targets set in the ROD. All EPA needs to do is to perform the statistical power analysis, determine the number of fish samples to collect given the sample variance, and collect/analyze the appropriate number of fish. EPA has thus far refused to do so. Also, it is important that EPA points out here that the agency at the present time expects that it will take eight years for fish PCB concentrations to decline by half; with fish PCB concentrations (in reach and species weighted average fish) currently more than three times	

			an 8% decrease in lipid-normalized fish tissue concentration per year).	the ROD target of 0.4 ppm PCB, EPA could conclude today that the remedial target will likely not be met.	
11	Executive Summary, Page 6	Impact of remedy on the Lower Hudson	The rate of decline of fish tissue PCB concentrations generally decreases with distance downstream. As a result, there is a decrease in the correlation between fish PCB concentrations in the Upper Hudson River and Lower Hudson River with distance downstream. This indicates that PCB sources in the Upper Hudson River have less of an impact on Lower Hudson River fish than on fish in the Upper Hudson.	DEC agrees that the remedy in the upper Hudson is not likely to have a significant impact on fish in the lower Hudson. EPA needs to clarify this statement, however, to point out that the GE sources in the upper Hudson are the primary source of PCBs in the Lower Hudson, and that presently GEs PCBs currently still coming out of the upper Hudson are much less of a source to lower river fish that GE's PCBs already in the Lower Hudson as a result of past discharges. EPA should not state that PCB sources other than GEs discharges in the upper Hudson are controlling lower Hudson fish PCB concentrations unless the agency has data to support such a conclusion.	
12	Executive Summary, Page 6	Impact of construction schedule on recovery rates	Overall, the project has been implemented as anticipated in the ROD. Dredging activities did include several operational differences from assumptions in the ROD with potential impacts on recovery rates in fish. Some of these differences included a delayed start to dredging, significantly increased mass removal, the use of a single processing facility, and dredging in multiple river sections simultaneously.	DEC agrees that there may be a delay in the start of recovery based upon construction schedule. However, this delay would only be one to two years at worst, in the landlocked reach in River Section 2 between the Fort Miller Dam and the Thompson Island Dam. There should be little or no delay in the Thompson Island Pool, as there was little remedial work in this River Section over the last two years of the remedy. Similarly, there should be no delay in River Section 3, as these reaches of river were dredged in a sequence nearly identical to that planned. ALSO, there should be no impact whatsoever on the post dredging recovery rates caused by the schedule of the work; in all cases, the post dredging recovery rates are and were assumed to be driven by the post dredging natural recovery processes which are not impacted by construction schedule. It is also important to point out that EPA, in the ROD, already built in a two year delay; while the model predicted reaching 0.4 in three years, the ROD says "within five".	Also - in RS1, the water quality data during the last few years of dredging showed little or no impact from dredging operations; the data were often non-detect for PCBs. The dredging work in the last year of the project were well downstream of the fish sampling locations (just above the TI Dam), or of short duration (two weeks) at the very northern end of the pool.
13	Executive Summary, Page 7	Health Risk Assessments	For OU2 (in-river sediments), the risks that were calculated for the ROD were re-assessed using current exposure assumptions, toxicity values, and standards to determine if the conclusions of the risk assessment or the protectiveness of the remedy has changed. Although there have been	EPA here makes one of the State's main points; the human health risk associated with this site is still well above the acceptable risk range, the conclusions reached in both the upper Hudson and lower Hudson human Health Risk Assessments done during the Reassessment RI/FS.	

			some updates to the exposure assumptions used in the human health risk, the updates do not change the conclusions of the risk assessment. Toxicity values for human health were taken from the Integrated Risk Information System for both cancer and non-cancer health effects, consistent with EPA guidance. EPA determined that the human health RAOs developed in the 2002 ROD are still valid and appropriate for the Site.		
14	Executive Summary, Page 7	Ecological Risk Assessment	For ecological risk, there were some changes to exposure parameters (some increasing and some decreasing) and toxicity values (i.e., the Lowest Observed Adverse Effect Level (LOAEL) and No Observed Adverse Effect Level (NOAEL)). Overall, use of these updated values would result in calculated risk ranges that are narrower than presented in the ROD, with a slight reduction in the upper bounds of the risk-based concentration ranges for PCBs in fish consumed by river otter and mink. This refinement results in risk-based ranges that reduce uncertainty and focus the range of PCBs in fish expected to be protective of the ecological exposure pathway. The lower bounds of the updated ranges are not lower than the lower bounds for both ranges identified in the ROD, and the refinements of toxicity values and exposure parameters do not affect the protectiveness determination of the selected remedy.	As with the human health risk assessments, EPA here makes one of the State's main points; the ecological risk associated with this site is still well above the acceptable risk range, the conclusions reached in the Ecological Risk Assessments done during the Reassessment RI/FS.	
15	Executive Summary, Page 8	Protectiveness Statement for OU2	OU2: Based on data collected and reviewed to date, EPA expects that the remedy at OU2 will be protective of human health and the environment upon completion. Remedial activities completed to date have substantially reduced PCB source materials in the Upper	The State disagrees with the protectiveness determination. The remedy is not protective if it will be several decades until PCB concentrations in fish will no longer require that the State recommend that human consumption of fish be limited, and until significant ecological risk has abated. The State also believes that EPA should recognize and articulate in the Five	

			Hudson River. As expected in the Record of Decision, average PCB concentrations in fish in the Upper Hudson are declining but have not yet reached protective levels. Therefore, as of the date of this five-year review, EPA recognizes the remedy at OU2 to be not yet protective of human health and the environment. Because the remedy includes not only the dredging component but also the subsequent period of monitored natural attenuation, EPA will not consider the OU2 remedy to be complete until the natural attenuation component also has been completed. Based on all the available data to date, EPA expects that continued natural attenuation following the completion of dredging will achieve the long-term remediation goal for the protection of human health with regard to fish consumption (0.05 mg/kg PCBs in species-weighted fish fillet). As EPA indicated in the Record of Decision, EPA believes it likely that improvement will occur gradually over several decades at least. In the interim, the State of New York has in place fishing restrictions and advisories against consumption of fish to control human exposure pathways that could result in unacceptable risks. EPA acknowledged in the 2002 ROD that the consumption advisories are not fully effective in that they rely on voluntary compliance in order to prevent or limit fish consumption. EPA will continue to work with New York State to ensure the ongoing maximum effectiveness of the advisories.	Year Review Report that there are currently, and will be for decades into the future, uncontrolled human health and ecological risk. EPA should also recognize and articulate in the Five Year Review Report that the fishing restrictions and consumption advisories are only partly effective in limiting human fish consumption, and are of no effect to address ecological risk.	
16	History of Contamination,	Reference to PCB mass discharges	From approximately 1947 to 1977, GE discharged an estimated 1.3 million pounds of PCBs into the Hudson River from its capacitor	Although this estimate of mass discharged to the river by GE from the capacitor plants in Hudson Falls and Fort Edward has been repeated many times, by many parties (including DEC) in	

	section 1.1.5, page 12		manufacturing plants at Hudson Falls and Fort Edward.	the past, DEC has concluded recently that there is no basis in the record for this estimate. DEC now believes that it is inaccurate and inappropriate to continue to cite this estimate. The actual mass discharged to the river is unknown, and may be much more than 1.3 million pounds (650 tons).	
17	Five Year Review Summary Form, page 14	Construction completion status	The form here states "No" in response to "Has the site achieved construction completion?"	Dredging was completed in late 2015. Habitat reconstruction as per the scope of work was reportedly completed in 2016. Facility decommissioning work was completed in late 2016. Construction is complete.	
18	Five Year Review Summary Form, page 14	Review Number	The form here states "2" in response to "Review Number".	This is actually the third five year review for this site overall; the third for OU1, and second for OU2. The initial review of the remedy selected in the 1984 ROD was started in 1989 and culminated in the 2002 ROD for OU2.	
19	Remedial Action Objectives (RAOs), pages 17-18	Identification of remedial action objectives	"Reduce the cancer risks and non-cancer health hazards for people eating fish from the Hudson River by reducing the concentration of PCBs in fish. The risk-based preliminary remediation goal (PRG) for the protection of human health is 0.05 mg/kg PCBs in fish fillet based on non-cancer hazard indices for the RME adult fish consumption rate of one half-pound meal per week (this level is protective of cancer risks as well). Other target concentrations are 0.2 mg/kg PCBs in fish fillet, which is protective at a fish consumption rate of one half-pound meal per month and 0.4 mg/kg PCBs in fish fillet, which is protective of the CT or average angler, who consumes one half-pound meal every two months. Attaining such levels might facilitate the relaxation of the fish consumption advisories and fishing restrictions (e.g., the "eat none" advisory for the Upper Hudson could be relaxed as conditions improve)." (AND) "In the	This is the portion of the ROD text where EPA specifically identifies the target concentrations in the ROD as remedial action objectives. In the ROD, EPA also states that "*The time to reach target PCB concentrations in fish was a primary factor in comparing remedial alternatives. As more fully described in Section 11.1 - Overall Protection of Human Health and the Environment, the time to reach target levels (e.g., 0.2 and 0.4 mg/kg) favors the active remediation alternatives.*" (ROD, pages 66-67)	See also ROD pages 71-72, Section 11.1; EPA relies upon the time to reach the 0.4 and 0.2 targets to differentiate between the alternatives and justify the selected remedy.

			ROD, EPA adopted the preliminary remediation goals identified above as the remediation goals for the Site."		
20	Institutional Controls, page 22	Effectiveness of Controls	"It is noted that the fish advisories rely on voluntary compliance and therefore are not completely effective in preventing fish consumption."	This understanding is a primary basis for the need, identified in the ROD, for rapid reductions in human health risk in the years immediately following remediation.	See also ROD page 104: "*Institutional controls do not protect ecological receptors, and human health risk reduction relies on knowledge of and voluntary compliance with the consumption advisories and fishing restrictions. Consequently, the active remedial alternatives are substantially more protective of people who do not follow the fish consumption advisories, because of the residual risk in consuming fish and the shorter time required to reach fish PCB target levels under those alternatives.*"
21	Operation and Maintenance, page 22	Scope of OMM	"EPA is currently considering whether any modifications are necessary to the OM&M programs identified in the Phase 2 OM&M Scope, which is an attachment to the consent decree under which GE is implementing the OU2 remedy."	DEC has already provided to EPA, by emails on February 10 and May 18, 2016, and by letter on March 10, specific recommendations on the needed scope of monitoring to evaluate remedy performance for this site. DEC also provided specific thoughts on the scope of sediment sampling by letter on November 14, 2016, when chose to approve a limited sampling effort for sediments in the upper Hudson.	
22	Operation and Maintenance, page 22	Scope of OMM	"The work plan for sediment sampling under OM&M was completed October 2016 in part to get the sediment samples collected as soon as possible post-dredging since it takes long periods of time (5 years) between sample events to properly measure changes in concentration."	EPA is incorrect in stating that long periods of time are necessary to properly measure changes in concentrations. EPA can make the appropriate evaluations to measure changes in concentration by using standard power analyses to determine the numbers of samples needed for the desired statistical power. In other words, EPA need only collect more samples to decrease the time needed to understand the changes in PCB concentrations over time.	

23	Technical Assessment, Section 5.1, pages 30-31	Analytical Bias in Fish Data due to Failure to Follow Proper Sample Preparation Protocols	"However, from 2007 to 2013 the GE fillet samples were processed while excluding the ribs of the fillet (*i.e.,* "rib-out" fillets), which is not consistent with New York State protocols. For this period, time trend analyses of PCB levels in fish fillets on a wet weight basis do not include these data, although the data are displayed in the various graphs of the report. The "rib-out" issue does not apply to whole body trend analysis (typically performed on fish collected in the fall) and does not affect lipid-normalized fillet trend analyses."	The failure of GE's contractor to follow the proper sample preparation protocols does impact the lipid normalized trend analyses. There is a downward bias in the lipid normalized PCB data on the order of 15%. EPA has determined that this is not significant; however, analyses of the data from the period 2007 to 2013 should include the understanding that the LPCB data from the GE lab is biased low.	
24	Technical Assessment, Section 5.1, page 31	Interpretation of Fish PCB data	"Dredging was completed in 2015 and, thus, the most recent data available (collected in 2016) reflect conditions less than a year after completion of dredging and that were still influenced by dredging-related impacts."	The 2016 spring sport fish in the upper Hudson (black bass, bullhead, perch) should be assessed as being impacted by the dredging work which ended in 2015, as the trend in fish PCB data indicates that the spring fish represent the previous years' conditions. The fall 2016 forage fish, however, should indicate the first year of post dredging conditions, as they went through an entire growth season in 2016 without dredging impacts.	
25	Technical Assessment, Section 5.1, page 31	Rationale for EPA's abandoning the targeted fish PCB concentrations identified in the ROD.	"Further monitoring will be required to verify remedy effectiveness, but the analyses presented in this report demonstrate that the models used to support decision making were well-designed, RAOs were appropriately developed, and remedy implementation is proceeding as planned. The project is currently transitioning from remedial action to the OM&M phase."	If the models used to support decision making were well-designed, the RAOs appropriately developed, and remedy implementation proceeded as planned, then why is EPA no longer seeking to reach the 0.4 ppm and 0.2 ppm fish PCB targets in the ROD?	
26	Technical Assessment, Section 5.1, page 31	PCB remaining in un-dredged areas in the Upper Hudson	"It is recognized that PCB mass in non-dredged areas is also greater than originally estimated, although not to the same extent as within the dredge areas."	EPA has not yet made a quantitative assessment of the PCB mass remaining in non-dredged areas as compared to previous estimates. This assessment is important in understanding long term performance of the remedy.	

27	Technical Assessment, Section 5.1, page 31	Reduction in surface sediment PCB concentrations	"The overall reduction in surface sediment Tri+ PCB concentrations in the three river sections as a result of dredging was 87%, 36%, and 5% in River Sections 1, 2, and 3, respectively. Although the reduction associated with dredging in River Section 2 was less than expected and may cause a lag in recovery, the overall surface sediment reduction in PCB levels is within ROD expectations."	According to EPA's previous five year review report (Appendix A, Table 1 - the EPA prediction from the model, used for the ROD), the reduction anticipated in the ROD was 79% for River Section 1, 64% for River Section 2, and 4.4% for River Section 3. Clearly, the reductions in River Section 2 were not within expectations. This Appendix also states that a delay of ten years in fish PCB recovery should be expected in River Section 2 as a result of this increase in remaining PCBs over what was anticipated.	
28	Technical Assessment, Section 5.1, page 32	Habitat reconstruction	Habitat reconstruction and replacement was conducted as anticipated to mitigate impacts from the dredging operations. OM&M of reconstructed habitats will continue until project metrics are met.	There are significant problems with the habitat reconstruction effort. DEC has provided to EPA, on multiple occasions, detailed comments on the need for further habitat reconstruction work to facilitate recovery of impacted habitats.	
29	Technical Assessment, Section 5.1, page 33	Sediment Recovery Rates	EPA has estimated an annual natural recovery rate of approximately 5 percent for surface sediment	If EPA believes that the sediment recovery rate is 5% on an annual basis, then the agency should also conclude that the fish recovery rate will also be ~ 5%.	
30	Technical Assessment, Section 5.1, page 33	Fish Recovery Rates	2016 fish data suggest that fish have begun to recover from dredging impacts and are generally back to pre-dredging levels. The average PCB concentration in Upper Hudson River fish at the time of the 2002 ROD was approximately 3 mg/kg (species-weighted, wet weight); prior to the start of dredging in 2009 the species-weighted, wet weight average was 1.4 mg/kg; in 2016 the average was 1.3 mg/kg.	The river section and species weighted average fish PCB concentration is here stated to be 1.3 ppm (mg/kg). At 5% annual recover rates (in keeping with the sediment recovery rate above) it will be 24 years until the 0.4 ppm target is reached, and 38 years until the 0.2 ppm target is reached. These were stated in the ROD to be reached in 5 and 16 years after dredging.	
31	Technical Assessment, Section 5.1, page 33	Fish Recovery Rates	It is recognized that up to 8 or more years of fish tissue data may be necessary to draw statistically valid conclusions about trends.	EPA can draw statistically valid conclusions about trends simply by gathering more samples per year.	

32	Technical Assessment, Section 5.1, page 33	Purpose of OMM program	Monitoring of water, fish, and sediment will continue under the OM&M program to confirm that natural attenuation continues to occur and the remedy is functioning as intended.	If the purpose of the OMM monitoring of water, sediment and fish is to confirm that natural attenuation continues to occur, and that the remedy is functioning as intended, then the sampling program must be designed to answer those questions. The remedy was intended to meet 0.4 ppm in reach and species weighted fish within five years; the water, sediment and fish monitoring must therefore be designed to answer the question "Will the reach and species weighted average fish PCB concentrations reach 0.4 ppm within five years after dredging?" The environmental medium in which the attenuation is to occur naturally is surface sediments; therefore the monitoring program must be designed to answer the question "Is the attenuation of PCB concentrations in surface sediments occurring at the rates necessary for the fish to reach 0.4 ppm within five years after dredging?"	
33	Technical Assessment, Section 5.1, page 33	Lower River	Limited data collection from the lower river indicates that recovery rates are slower than in the Upper Hudson River and may not be strongly associated with PCB loading from the Upper Hudson River.	EPA here admits that conditions in the Hudson River are such that a full investigation is needed in the lower River to understand how GE's PCBs already in the sediments of the lower Hudson are controlling water column and fish PCB concentrations, and to determine what remedial actions may be necessary to address the human health and ecological risks in the lower Hudson posed by the sediment PCBs.	
34	Technical Assessment, Section 5.1, page 33-34	Impact of Schedule on Remedy	Overall, the project has been implemented as anticipated in the ROD. The project implementation did include several operational differences from assumptions in the ROD with potential impacts on recovery rates in fish. Some of these differences included a delayed start to dredging, significantly increased mass removal, the use of a single processing facility, and dredging in multiple river sections simultaneously.	EPA, in Appendix 8, describes how there may be an up to two-year impact on recovery rates associated with construction sequencing and the apparent lack of consideration of construction impacts in the model predictions. However, (as EPA now states) EPA anticipated reaching the 0.4 ppm targeted fish PCB concentration two years after dredging, and the ROD text said up to five years to reach the 0.4 ppm target, there is no need to adjust expectations based upon this issue...EPA already took in into account at the time of the ROD, in making the time to target five years instead of two.	

35	Project Operated and Functioned as Designed, Section 5.1.1.2, p. 36	Purpose of OMM program	The OM&M sediment sampling program, specifically designed to monitor long-term changes in sediment PCB concentrations, will produce the most comprehensive sediment dataset to evaluate PCB concentration trends in Upper Hudson River sediments. As there are no RAOs or remediation goals specifically linked to sediment PCB concentrations, the OM&M sampling is intended to create a diagnostic dataset to better understand recovery from dredging-induced disturbances in the Upper Hudson River, but not as a direct means to determine whether (nor where) further remediation of the Upper Hudson River may be warranted.	EPA here misstates the purpose of gathering the sediment data during OMM. According to the OMM Scope document, the objectives are (see section2.3.1) (1) Determine post-remediation PCB levels in sediments in non-dredge areas of the Upper Hudson River; (2) Provide data on Select Areas that exceeded the MPA removal criteria that were not targeted for removal because they were buried by cleaner sediments to assess whether the deposits have experienced erosion; (3) Determine sediment recovery rates in non-dredge areas of the Upper Hudson River; and (4) Examine the changes to surface PCB concentrations in backfill areas. EPA needs to design and implement a sediment sampling program to be used in OMM which meet the overall goal for the OMM program which is to "provide data on PCB levels over time to assess whether the Remedial Action Objectives (RAOs) and Remediation Goals (RGs) set forth in the ROD are being achieved." EPA must focus monitoring effort toward assessing progress toward reaching the near term objectives, and not just on long term changes.	
36	Project Operated and Functioned as Designed, Section 5.1.1.2, p. 37	Basis for Five Year Review analysis of remedy function	For this five-year review, the following criteria represent the primary metrics for evaluation of remedy function: (1) Baseline trends and construction impacts (Water column PCB concentrations prior to and during Phase 1 and Phase 2 dredging (refer to Section 5.1.1.3.3) and fish tissue PCB concentrations prior to and during Phase 1 and Phase 2 dredging (refer to Section 5.1.1.3.4); (2) Sediment and PCB mass removal via Phase 1 and Phase 2 dredging (refer to Section 5.1.1.3.2); (3) Pre-dredging MNA period trends (refer to Section 5.1.1.3.5) and (4) Capping Effectiveness (refer to Section 5.1.1.4).	This passage summarizes the primary problem with the rationale used by EPA to support a protective determination other than the appropriate "not protective". Here EPA states that the evaluation of how the dredging remedy is performing on conditions before the remedy was implemented, and during implementation. The conditions before and during dredging did not, do not, and will not control the rates of decline in fish, water and sediment PCB concentrations. EPA should not use these data from before and during dredging to quantify the rates of improvement after dredging due to natural recovery.	

37	Evaluation of PCB Mass Removal, Section 5.1.1.2.1, p. 39-41	Detailed Evaluation of PCB Mass Removed	(This entire section focuses on the percentage of PCB mass removed within the upper Hudson River)	EPA misses the point. The ability of natural recovery to achieve the recovery in surface sediment PCB concentrations needed to meet EPA's remedial goals is driven by how much PCB was left behind, not on how much was removed.	
38	Habitat Reconstruction, Section 5.1.1.2.3, p. 43-44	Loss of Habitat due to Remedy	(This entire section focuses on the reconstruction of habitat in the project area)	DEC believes that substantial unnecessary habitat loss occurred during the remedial work due to the failure of EPA to follow applicable State guidance and law.	
39	PCB Levels in Fish, Sediment and Water are Declining, Section 5.1.1.3, p. 44	Post Dredging Recovery	The length of time needed to achieve remedial goals and remedial action objectives was an important factor considered by EPA in the 2002 ROD.	EPA should continue to manage this site as though the time to reach remedial goals continues to be important. However, EPA has chosen to ignore the interim targets and instead focus on the long term goal, which would be achieved in about the same time whether or not the dredging occurred. EPA needs to manage this site to meet the ROD interim targets upon which the remedial decision were based - achieving 0.4 ppm in reach and species averaged fish five years after dredging, and achieving 0.2 ppm 16 years after dredging.	
40	PCB Levels in Fish, Sediment and Water are Declining, Section 5.1.1.3, p. 44	Post Dredging Recovery	The HUDTOX model computed an effective rate of decay in sediment concentrations of approximately 8 percent per year for the calibration period. Consistent with the close relationships among sediment, water, and fish tissue PCB concentrations, FISHRAND generated rates of decline of PCBs in fish tissue similar to rates observed in HUDTOX over the 1977-1998 time period, as discussed in Appendix 3. Following dredging, the models predicted continued declines in tissue concentrations, although the upstream project boundary PCB load ultimately results in asymptotic non-zero PCB concentrations in fish (see, e.g., 2002 ROD, p. 54).	EPA here states a fundamental assumption made in selecting remedy - the removal of the sediment targeted in the ROD would result in a post dredging recovery rate of 8% per year, which, because sediment and fish PCB concentration are closely related, would result in post dredging recovery rates of 8% in both media. If this is not being achieved, then EPA should adjust the amount of sediment removed.	

41	PCB Levels in Fish, Sediment and Water are Declining, Section 5.1.1.3, p. 44-45	Post Dredging Recovery	In addition, EPA considered a target concentration of 0.2 ppm PCBs (wet weight) in fillet based on one half-pound meal per month, and a target concentration of 0.4 ppm based on the average (central tendency) consumption rate of one half-pound meal every 2 months. The target concentrations (which can be considered interim milestones) correspond to points at which the fish consumption advisories could be relaxed from the current "eat none" recommendation in the Upper Hudson River to allow a limited number of fish meals (*i.e.,* ranging from 6 to 12) per year, as recovery of the river progresses to the point where unlimited consumption is safe. It should be noted that the fish consumption advisories are under the control of NYSDOH.	EPA here confirms the importance of the 0.4/0.2 ppm targeted (reach and species average) fish PCB concentrations, noting the importance of reaching PCB levels which correspond, in EPA's view, to levels representing reduced human health risk allowing consumption.	
42	PCB Levels in Fish, Sediment and Water are Declining, Section 5.1.1.3, p. 45	Post Dredging Recovery	Modeling presented as species-weighted averages in Table 11-2 of the ROD showed that neither MNA nor the selected remedy would achieve the human health remediation goal of 0.05 ppm PCBs for RS1, RS2, or for the Upper Hudson River as a whole, within the modeling time frame (to 2067) unless the upstream source was virtually eliminated, but would be achieved within 40 years in RS3 (RM168-154).	Here EPA confirms the critical importance of the interim targets, which provided the basis for selecting the dredging remedy. There is no difference between any of the alternatives in reaching the long term target of 0.05 ppm total PCB; the only difference was the time to reach the interim targets (the dredging remedy provided significant rapid risk reductions compared to not dredging).	
43	PCB Levels in Fish, Sediment and Water are Declining, Section 5.1.1.3, p. 45	Post Dredging Recovery	The model results averaged over three species in the entire Upper Hudson River, as presented in Table 11-2 of the ROD, project that a target level of 0.4 mg/kg wet weight could be achieved several years after completing dredging and after 15 years for the 0.2 mg/kg wet weight target level.	The ROD text actually says five and sixteen years, respectively, to reach the 0.4 and 0.2 ppm reach and species weighted average concentration.	
44	PCB Levels in Fish, Sediment and Water	Post Dredging Recovery	The median largemouth bass concentration of PCBs is close to the 0.4 mg/kg target level, and	Here EPA is "comparing apples and oranges." The median is different than the average (also known as mean). Given the	

	are Declining, Section 5.1.1.3, p. 45		the yellow perch median is below this target level. Similarly, Figures A3-3 and A3-4 show that in RS2 (RM184) and RS3 (RM154-168), largemouth bass median tissue concentrations are close to 0.4 mg/kg and median yellow perch levels have achieved the 0.4 mg/kg target concentration.	distribution of the fish PCB data, the median is less than the mean. EPA in discussing the interim targets which were AVERAGES, should not be presenting data in terms of MEDIANS, which are not the same thing. It is also important to point out that the targeted concentrations in the ROD were species weighted as well as river section length weighted, meaning that the comparisons to individual species at individual locations are not particularly meaningful when comparing to the metric EPA chose in the ROD. DEC does agree, however, that comparisons at specific locations are very important in understanding remedy performance over time, and encourages EPA to gather fish, sediment, and water data on a pool by pool basis rather than river section basis.	
45	PCB Levels in Fish, Sediment and Water are Declining, Section 5.1.1.3, p. 46	Post Dredging Recovery	As also discussed earlier, actual dredging activities deviated from the upstream-to-downstream pattern of dredging anticipated at the time of the ROD. For example, dredging occurred in RS1, the most upstream river section, during the final year of the remedy. As a result of this and other operational modifications (described in Appendix 8), specific predictions of dredging-related impacts to water column, sediment, and fish tissue concentrations as presented in the ROD differed in some respects from what was observed. Appendix 8 also discusses short term impacts to fish tissue concentrations as a result of these modifications. As expected, these impacts were spatially and temporally transient.	The work in River Section 1 in the last year of dredging, with the exception of a small area for a few weeks near Rogers Island, was all at the extreme south end of the pool, well downstream of the five fish sampling locations in this pool. This work would not have had a significant effect on the fish PCB concentrations gathered in River Section 1 in either the spring or the fall of 2015 or 2016.	
46	PCB Levels in Fish, Sediment and Water are Declining, Section 5.1.1.3, p. 46	Post Dredging Recovery	Less than one year of post-dredging data is available, and additional years of monitoring data are required for a robust statistical evaluation of post-dredging MNA trends. This five-year review assesses the current status of the river	This statement contradicts much of the report, which relies on the use of pre-dredging data to support the effectiveness of the modeling effort to support the use of the model to predict success of the remedy. A true assessment of the post remedy fish data suggests that it is very unlikely that the ROD targeted	

			using the most current post-dredging data for sediment, water column and fish tissue PCB concentrations, and provides preliminary indications of system response to implementation of the remedy.	rapid reductions in fish PCB concentrations, which formed the primary basis for selecting and implementing the remedy, will not be achieved in the time frames identified in the ROD.	
47	Page 61	Consumption Advisories	"…commercial fisherman…"	Replace with "…fisherman's associations…"	
48	Page 62	Consumption Advisories	"According to NYSDOH, since 2011 certain communities in the Lower Hudson Region (south of Bear Mountain Bridge) may ***have been*** less aware…"	Replace with "According to NYSDOH, previous consumption surveys indicate that certain communities in the Lower Hudson Region (south of Bear Mountain Bridge) may be less aware…"	
49	Page 62	Consumption Advisories	"Therefore, according to NYSDOH, in 2011 NYSDOH ***began focusing more of*** its outreach efforts on the Lower Hudson River Region and since 2012, to more recently observed demographic groups."	Replace with "Therefore, according to NYSDOH, in 2011 NYSDOH continued its outreach efforts in the Lower Hudson River Region and since 2012, to more recently observed demographic groups."	
A4-1	Appendix 4 - General Comment	Surface Sediment Concentrations	(General Comment)	It is important to point out that in Appendix 4, EPA is making a fundamental error - assuming that all of the changes in sediment PCB concentrations are the result of natural recovery. Since 1977, there have been a number of significant events which impact sediment PCB concentrations which are not the result of natural processes. Assuming that changes in sediment PCB concentrations are the result of natural processes (only once in Appendix 4 is source control even mentioned as a potential factor in changes of sediment PCB concentrations) fundamentally confounds the use of Appendix 4 as a source of understanding the rates of natural recovery. For example, looking at rates of recovery between 1977 and 1991, without taking in to account the impact of the cessation of PCB discharges to the Hudson in 1977, remedial measures at the remnant sites, and initial abatement measures at the GE plant sites, significantly overestimates the rates of improvement due to "natural recovery" during this time period. Similarly, the reduction in PCB sources to the river due to numerous remedial	

				measures taken at the GE plant sites over many years starting in the late 1980s at Fort Edward in the early 1990s at Hudson Falls, throughout the period leading up to the start of dredging in 2009 similarly confounds the use of the sediment data to understand rates of "natural recovery". Without taking the impact of source control into account, all of EPA's estimates of rates of natural recovery represent overestimations and upper bounds; recovery rates could be no higher, but the recovery due to natural processes are very likely much less, as the impacts of source control likely dominated changes to the system during the periods in question.	
A4-2	Appendix 4, page 1-1	Sediment - Fish relationship	The reduction in fish tissue PCB concentrations that will be achieved by the overall reduction in the PCB mass that may become bioavailable is closely related to the surface sediment PCB concentration throughout the Upper Hudson. In the selected remedy, reduction of PCBs in surface sediment is achieved through two important processes: 1) sediment removal by dredging and backfilling, and 2) monitored natural attenuation (MNA). Both processes are required to achieve the goals of the ROD. In general, fish body burdens are expected to track with the changes in surface sediment PCB concentrations (*i.e.*, if residues decrease in the surface sediment, then they should also decrease in the overlying water column, and with reductions in sediment and water, the residues in fish should decline as well). Bioaccumulation relationships are site-specific, and in any given setting, if a 10- fold reduction in fish body burden is targeted, then, at a minimum, a 10-fold reduction must be achieved in the media to which fish are exposed (sediments and overlying water).	There is no reason to believe that the sediment - water - fish relationship is different from one reach of the upper Hudson to another. EPA can not have it both ways - either the local surface sediments drive fish PCB concentration, or they do not. EPA has no basis to suggest that the sediments in River Sections 1 and 2 control fish PCB concentrations, while the water column controls fish PCB concentrations in River Section 3. The fish and water PCB data gathered during implementation of the remedy indicate that there could be large increases in water PCB concentrations without corresponding increases in fish PCB concentrations, indicating that the surface sediment PCB concentrations are much more significant contributors to fish PCB than water column PCBs at this site. Local sediments drive local fish. In order to achieve reductions in fish PCB concentrations, reduction in local surface sediment PCB concentrations are likely necessary.	

			This may be achieved directly by reducing contaminant concentrations in sediments composing the feeding/home range of the fish, or as in River Section (RS) 3, indirectly by reducing water column concentrations impacting prey downstream of sediment remediation areas.		
A4-3	Appendix 4, page 2-1	Usability of past sediment sampling programs for temporal analysis	Sediment data are inherently spatially limited, and are typically obtained from samples collected using a coring device or a grab sampler. In trying to characterize large areas of the river bottom, care must be taken to obtain spatially representative samples. Because of the highly variable nature of PCB sediment concentrations, even over short distances (less than 2 meters), a statistically appropriate number of samples and an appropriate sample design are needed to accurately measure the mean concentration in a given area. Thus, any program to monitor temporal changes in surface sediments must be designed accordingly and, in addition, multiple sample rounds need to be collected over time in a consistent way. None of the sediment sampling programs conducted to date was designed specifically with this objective (*i.e.,* to represent changes in sediment PCB concentrations over time), with the exception of the 2016 data collection. As a result, conclusions about concentration trends should be drawn cautiously and their limitations clearly discussed.	DEC agrees; none of the past sediment sampling programs were designed and implemented in a manner which allows for meaningful quantitative temporal analysis of trends in surface sediment PCB concentrations. EPA had an opportunity to implement such a program, designed to achieve the needed data quality objectives - to determine if the post dredging improvement in surface sediment concentrations due to natural recovery is occurring at the rate necessary to achieve the ROD objectives. This is why DEC undertook a sediment sampling program in 2017 to answer this essential question. EPA's sediment sampling program, due to insufficient number of samples, will not answer this question until several years after the first ROD targets are to be reached.	
A4-4	Appendix 4, page 2-1	Usability of past sediment sampling programs for temporal analysis	(See above)	It is also important to point out that EPA here clearly describes the limitations of the analyses presented in Appendix 4. DEC views these analyses as informative, but not quantitative with the degree of certainty needed to evaluate the rate of recovery prior to dredging. It is also an error to try to anticipate or	

				estimate the rate of post-remedial recovery in surface sediment concentrations based upon the rate of improvement before the remedy, as there has been fundamental changes in the system due to source control before dredging and sediment removal/backfilling as part of the dredging.	
A4-5	Appendix 4, page 2-8	2016 Sediment sampling	GE's 2016 surface sediment sampling program was designed under EPA direction as part of the OM&M sediment monitoring program to assess long-term recovery following the completion of the dredging remediation via the collection and analysis of surface sediment samples from both non-dredged and dredged areas in the Upper Hudson River. The 2016 sampling event establishes the initial year of the required sampling design in non-dredged areas. The required sampling of the dredged areas will occur in 2017. Determination of the required number of samples and their locations was based on EPA's sampling design analysis.	EPA should reveal here the fundamental basis for the sample design analysis - understanding the rate of change over ten years, on a river section by river section average basis, which is not sufficient to understand the performance of the remedy in a time frame commensurate with the remedial targets.	
A4-6	Appendix 4, page 2-8	2016 Sediment sampling	The OM&M surface sediment sampling design5 is a probability-based program developed around the objective of supporting rigorous, unbiased estimates of overall post-dredging average PCB concentrations, and associated uncertainty bounds, in RS1, RS2, and RS3. The data collection will be used to quantify changes in overall average surface sediment concentrations over time by river section and to support investigation of relationships among fish, water and sediment during the post-remedial monitoring period.	Unfortunately, EPA's sediment sampling design will confound the ability to use the sediment data to understand the sediment-fish relationship, as EPA will be averaging the sediment PCB concentration between pools in River Section 2 (two pools, six miles) and particularly in River Section 3 (five pools, over 28 miles). Fish in Schuylerville are not controlled by sediments in Waterford. The averaging of sediment between pools, and fish between pools, will dilute out any actual relationships. Fish in one pool are not driven by sediments in another pool. As discussed above, local surface sediments drive local fish PCBs in this system. As a result, the data necessary for the understanding of the fish-sediment relationships needs to be gathered on a pool by pool basis.	

A4-7	Appendix 4, page 3-7 to 3-8	Usability of past sediment sampling programs for temporal analysis	Ultimately, the pairing of sediment surveys to determine the rate of decay in Tri+ PCB concentrations in surface sediments is challenged by the lack of comparability among the data sets. Each survey has unique features that make direct comparison difficult and yield inconsistent rates of change. The 1991 and 1998 surveys utilized composite samples which mask the spatial heterogeneity that is more clearly defined in the dense sampling grid used during the collection of the 2002-2005 discrete samples. In particular, analysis based on sediment compositing is challenged by the difficulties of achieving true homogeneity among discrete portions when concentrations can vary by orders of magnitude, and sediment textures can vary significantly in the proportion of coarse *vs.* fine particles. The use of the available sediment survey data as an independent basis to determine the rate of decay of Tri+ PCB concentrations in surface sediments in the Upper Hudson is highly uncertain.	DEC agrees; none of the past sediment sampling programs were designed and implemented in a manner which allows for meaningful quantitative temporal analysis of trends in surface sediment PCB concentrations. EPA should not rely on such analyses, and instead rely upon the statistically representative and robust data gathering program designed and implemented by the State to understand the relationship between fish and sediment PCB concentrations on a pool by pool basis, with a monitoring program designed to achieve the data quality objectives in a time frame commensurate with the remedial targets in the ROD, ie. five years.	
A13-1	Appendix 13, page 1-3	Editorial	commercial fishermen	fishermen's associations and recreational anglers	
A13-2	1-4 paragraph 2	Editorial	Despite ongoing outreach efforts, as of 2011 communities in the Lower Hudson River region	Despite ongoing outreach efforts, previous consumption surveys indicate that communities in the Lower Hudson River region	
A13-3	1-4 Paragraph 2	Editorial	Therefore, in 2011 NYSDOH began focusing more of its outreach efforts on the Lower Hudson River region.	Therefore in 2011 NYSDOH noted the continued need for outreach efforts in the Lower Hudson River region.	
A13-4	1-5 paragraph below bullets	Editorial	understand the demographics of the project study area fish consumption	understand the project study area fish consumption	
A13-5	1-8 first paragraph	Editorial	feedback from focus groups that reviewed existing signs suggested	Feedback from community partners that reviewed existing outreach materials suggested	
A13-6	1-8 second paragraph	Editorial	Chinese America Planning Council	Chinese-American Planning Council	

NYSDEC's Detailed Comments – Five Year Review Report Text and Appendices

August 30, 2017

FW: Hudson River Five Year Review

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Wed 9/6/2017 9:57 AM

To: 'epahrfo@outlook.com' <epahrfo@outlook.com>;

2 attachments (3 MB)

2017 09 01 OAG to EPA re HR 5 YR.pdf; 2016 09 16 OAG to EPA re HR 5 YR w Attach.pdf;

From: Maureen F. Leary [mailto:Maureen.Leary@ag.ny.gov]
Sent: Friday, September 01, 2017 3:58 PM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>; Fischer, Douglas <Fischer.Douglas@epa.gov>; Kautsky, Peter (ENRD) <Peter.Kautsky@usdoj.gov>; Brian Donohue <brian.donohue@usdoj.gov>
Cc: Guglielmi, Andrew O (DEC) (andrew.guglielmi@dec.ny.gov) <andrew.guglielmi@dec.ny.gov>
Subject: Hudson River Five Year Review

Please see attached letters regarding EPA's Second Five Year Review. Thank you.

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DIVISION OF SOCIAL JUSTICE
ENVIRONMENTAL PROTECTION BUREAU

September 1, 2017

Scott Pruitt, Administrator
United States Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Walter Mugdan, Director
Emergency and Remedial Response Division
United States Environmental Protection Agency, Region 2
290 Broadway
New York, New York 10007-1866

Re: *Hudson River Superfund Site: EPA's Five Year Review and Certificate of Completion of Remedial Action*

Dear Administrator Pruitt and Mr. Mugdan:

Please accept this letter on behalf of the New York Attorney General s Office regarding EPA s May 31, 2017 Five Year Review for the Hudson River Superfund Site. Rather than repeat the content of our previous communications to you with respect to the sufficiency and completeness of the remedial action, we enclose our September 16, 2017 letter. We also reiterate the August 30, 2017 comment letter of Basil Seggos, Commissioner of the New York State Department of Environmental Conservation.

Thank you for your consideration of the foregoing. We look forward to your response and to our continued discussions regarding the Hudson River.

Very truly yours,

Maureen F. Leary
James C. Woods
Brittany Haner
Assistant Attorneys General
John D. Davis
Environmental Scientist

Attachment

cc: Honorable Basil Seggos
Mathy Stanislaus
Brian Donohue (by email)
Peter Kautsky (by email)
Douglas Fisher (by email)
Gary Klawinski (by email)

STATE OF NEW YORK
OFFICE OF THE ATTORNEY GENERAL

ERIC T. SCHNEIDERMAN
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DIVISION OF SOCIAL JUSTICE
ENVIRONMENTAL PROTECTION BUREAU

September 16, 2016

By Electronic Mail

Judith Enck, Regional Administrator
United States Environmental Protection Agency
290 Broadway
New York, New York 10007-1866

Walter Mugdan, Director
Emergency and Remedial Response Division
United States Environmental Protection Agency
290 Broadway
New York, New York 10007-1866

Re: *Hudson River Superfund Site: EPA's Five Year Review and Certificate of Completion of Remedial Action*

Dear Administrator Enck and Mr. Mugdan:

Please accept this letter on behalf of the New York Attorney General's Office as a part of our ongoing dialogue with EPA regarding the Hudson River Superfund Site. We believe that additional steps are necessary to assure that the remedial action objectives set forth in EPA's 2002 Record of Decision ("ROD") are timely met and that the remedy is fully protective of human health and the environment. Completion of those steps is necessary before EPA issues a certificate of completion of the remedial action to GE pursuant to 42 U.S.C. § 9622(f)(3) and the November 2006 Consent Decree between EPA and GE.

EPA's issuance of the certificate of completion must comply with the statutory requirements of CERCLA Section 122(f), 42 U.S.C. § 9622(f), insofar as it will give rise to a covenant not to sue and a release from liability for GE. EPA should not predicate the certificate of completion solely upon completion of the technical engineering performance tasks undertaken pursuant to the Consent Decree. Rather, it should be issued only upon completion of the remedial action in accordance with CERCLA and only upon EPA's finding after a comprehensive review that the remedy is protective of human health and the environment, as contemplated by the ROD. The covenant that arises upon issuance of the certificate of completion is in direct conflict with CERCLA's intent absent compliance with the remedial action objectives in the ROD and completion of the remedy contemplated, and absent a fully

supported finding that the remedy is protective of human health and the environment. 42 U.S.C. § 9622(f).

Accordingly, we request that EPA, at a minimum, ensure that the remedial action objectives of the ROD and the requirements of CERCLA are met by taking the following actions:

(1) Defer issuance of a certificate of completion of the remedial action until EPA finds that the remedy is completed and is fully protective of human health and the environment in compliance with 42 U.S.C. § 9622(f)(3), (5);

(2) Determine with reasonable certainty how long it will take for the PCB concentrations in fish to drop to the levels necessary to achieve those objectives and to enable lifting human consumption health advisories for PCBs in all contaminated River reaches for all affected species;

(3) Undertake a comprehensive fish consumption survey along all contaminated reaches of the River, from Hudson Falls to the Battery, to quantify present and future human exposure to PCBs from the consumption of contaminated fish; and

(4) Define in writing the scope and objectives of the Five Year Review and the participation and respective roles of the review team members, including non-EPA members.

These issues are discussed in detail below.

(1) Defer Issuance of a Certificate of Completion of the Remedial Action until EPA Finds that the Remedy is Protective of Human Health and the Environment in Compliance with 42 U.S.C. § 9622(f)

We understand that EPA is considering issuing to GE a certificate of completion of the remedial action, notwithstanding evidence establishing the ineffectiveness of the remedy in achieving the ROD's remedial action objectives, and despite EPA's on-going Five Year Review to determine the effectiveness of the remedial action. It is inconsistent with the requirements of CERCLA Section 122(f)(3) to issue the certificate of completion without finding that the ROD's remedial action objectives have been achieved and that the remedial work necessary to achieve those objectives is complete. 42 U.S.C. § 9622(f)(3). That provision prohibits a covenant not to sue from taking effect unless EPA "certifies that the remedial action has been completed in accordance with the requirements of this chapter at the facility that is the subject of such covenant." 42 U.S.C. § 9622(f)(3). The question presented here is whether EPA can certify that the Hudson River remedial action has been completed in accordance with the requirements of CERCLA.

EPA's issuance of a certificate of completion is contrary to CERCLA's statutory scheme in the absence of a finding that the remedial action objectives of the ROD have been achieved and the remedy is protective of human health and the environment. For the Hudson River, the preeminent remedial action objective in the ROD is the reduction of the concentration of PCBs in fish within specific time-frames. *See* ROD, p. 73. It is now clear that the ROD's express remedial action objective to reach 0.4 mg/kg of PCBs in fish by 2016 has not been achieved. Based on the most recent data available, the 2014 PCB fish concentration is 2.71 mg/kg, which is more than 600% greater than the remedial objective of 0.4 mg/kg for 2016. No reasonable observer expects the upcoming 2015 or 2016 data to demonstrate that the 0.4 mg/kg concentration has now been achieved. The ROD's objective of achieving more dramatic reductions (0.05 mg/kg) later also is questionable, as EPA recognized in its 2012 Five-Year Review. *See* "First Five Year Review Report for Hudson River PCBs Superfund Site," p. 34 (2012).

In addition to the ROD's remedial action objective for PCB fish concentrations not being met, EPA has failed to provide a revised projected time-frame by which they will be met. EPA has not publicly amended the ROD or explained the significant difference between its objectives and the current status of PCB concentrations in fish. Because EPA cannot conclude that the ROD's remedial action objectives have been met, it cannot conclude that the remedial action has been completed in accordance with CERCLA's requirements. 42 U.S.C. § 122(f)(3). Consequently, a certification of completion and the associated covenant not to sue would be improper.[1]

Furthermore, a certificate of completion is premature before EPA completes its Five Year Review, which is presently underway. If EPA finds that the remedy is not protective (or if it improperly defers a protectiveness finding because of the absence of data showing declining PCBs in fish),[2] EPA may have limited recourse against GE once the certificate is issued. Indeed, EPA's finding that the remedial action has not met the ROD's standard of "protective" would be

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[1] Unlike numerous other consent decrees for Superfund sites, the 2006 Hudson River Consent Decree does not specify that certification of the remedial action shall be issued in compliance with CERCLA Section 122(f), 42 U.S.C. § 9622(f)(3). *See, e.g., U.S. v. Atlantic Richfield Co.*, CV-83-317-HLN-SHE (D. Montana, 2008), Consent Decree, Clark Fork River Operable Unit (p. 16) ("'Certification of Completion of the Remedial Action' shall mean EPA's certification, in consultation with the State, pursuant to Section 122(f)(3) of CERCLA, 42 U.S.C. § 9622(f)(3), that the Remedial Action . . . have been completed . . . in accordance with the requirements of CERCLA, the NCP, and the ROD . . . including certification that Performance Standards have been attained."); *U.S. v. NCR Corp., et. al.*, CV-10-C-910 (E.D.Wisc. 2010), Notice of Lodging of Consent Decree (p. 14) (". . . these covenants shall take effect upon certification of completion of the remedial action by EPA pursuant to 42 U.S.C. § 9622(f)(3)"). The 2006 Hudson River Consent Decree, however, must be read as incorporating that statutory requirement.

[2] EPA's guidance indicates that deferral of a protectiveness finding in the Hudson River's Five Year Review is not appropriate because exposure pathways are well-known, no new exposure pathways have been identified, no new contaminants have been identified, and an ecological risk assessment has been done. *See* OSWER Memo 9200.2-111: "Clarifying the Use of Protectiveness Determinations for CERCLA Five-Year Reviews," p. 4 (Sept. 13, 2012).

well-supported because there continues to be human exposure to PCBs from fish consumption and the migration of PCBs down-River.

The Five Year Review is intended to assure the protectiveness of the remedy in situations where contamination remains. 42 U.S.C. § 9621(c). The purpose of the Review is directly related to determining whether a responsible party may be granted a covenant not to sue. A remedy that is not protective of human health and the environment may not be deemed by EPA as complete, and granting a covenant not to sue in those circumstances is not appropriate under the statute.

Moreover, GE has not met a fundamental requirement for a covenant not to sue under CERCLA Section 122(f). As required by CERCLA Section 122(f)(5), 42 U.S.C. § 9622(f)(5), GE has not completed all outstanding obligations under the 2006 Consent Decree including, but not limited to, work related to (1) successfully restoring all River habitat damaged during implementation of remedial work, and (2) decommissioning and decontaminating the contaminated sediment processing facility.

A certification of completion of the remedial action should not issue to GE absent EPA's finding that the ROD's remedial action objectives have been met, that the remedy is *protective,* and that GE is in compliance with, and has *completed,* all obligations under the Consent Decree within the meaning of CERCLA Section 122(f), 42 U.S.C. § 9622(f).

(2) EPA Should Determine with Reasonable Certainty the Time-Frame Necessary to Achieve the Remedial Action Objectives for the Reduction of PCB Concentrations in Fish

As discussed above, it is now clear that the remedy has not met the remedial action objective of reducing PCB concentrations in fish to 0.4 mg/kg by 2016, and may not reach the ROD's more dramatic reductions to 0.05 mg/kg. Accordingly, EPA must determine with reasonable certainty the time-frame by which there will be a reduction of PCB concentrations in fish so that fish consumption advisories for PCBs may be lifted in all contaminated River reaches of the Hudson River for all species.

New York's concurrence in the ROD was premised upon timely achieving the stated remedial action objectives for reducing PCB concentrations in fish. The State has been prejudiced not only by the failure to achieve those objectives timely, but by the current lack of certainty regarding when they will be achieved so that Hudson River fish can be safely consumed. We request that as a part of the Five Year Review process, EPA clearly define the time-frame for achieving the remedial action objectives set forth in the ROD.

In evaluating that time-frame, EPA must take into account the change in fish tissue sampling that occurred during GE's implementation of the baseline and remedial fish monitoring. The consequence of the fish data being collected in a manner that was inconsistent with what New York believed was required is important to answering the question of the time-frame for achieving the remedial objectives. This issue requires credible review by EPA.

(3) EPA's Determination of the Remedy's Protectiveness Must Be Supported By a Comprehensive Fish Consumption Survey to Quantify Current and Potential Future Human Exposure

The short- and long-term effectiveness of fish consumption advisories as an institutional control of human exposure to PCBs in edible fish is questionable. Despite the New York Department of Health's ("NYSDOH's") annual issuance of the advisories, the public is still consuming fish from the Hudson River, a circumstance of which EPA is aware. Human consumption and exposure need to be quantified and evaluated for all contaminated River reaches in order to determine whether the advisories are sufficiently protective over both the short- and long-term. In addition, the localized effects of human exposure in certain more contaminated areas of the River also should be evaluated as part of EPA's Five Year Review and protectiveness determination.

In 1996, the NYSDOH conducted a survey and found that the public was still consuming Hudson River fish in significant amounts despite extensive public outreach and widespread knowledge of the levels of PCB contamination in fish (Survey Report attached). The public's level of fish consumption and exposure that is documented in NYSDOH's Survey Report may be greater today in light of the public's understandable - but incorrect - perception that the River has been cleaned up and that the fish may safely be eaten.

An updated survey of fish consumption along all contaminated reaches of the River from Hudson Falls to the Battery should be undertaken to accurately assess current and future human exposure and the efficacy of fish consumption advisories as short- and long-term institutional controls to protect human health.

(4) The Scope and Objectives of the Five-Year Review

Presently, EPA is undertaking a second Five Year Review for the Hudson River remedy in which it is required to make a finding that human health and the environment are being protected. CERCLA § 121(c) provides:

> If the President selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site, the President shall review such remedial action no less often than each 5 years after the initiation of such remedial action *to assure that human health and the environment are being protected by the remedial action* being implemented.

42 U.S.C. 9621(c) (emphasis added). Thus, fundamental to the Five Year Review is EPA's required finding that the remedy is protective.

Under applicable guidance, a protectiveness determination requires EPA to find that the remedy is functioning as intended by the ROD; that the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives used at the time the remedy was selected are still valid; and that no information has come to light that could call the protectiveness of the remedy

into question. *See* EPA's "Comprehensive Five Year Review Guidance," p. 4-1 (June 2001). Given this criteria, EPA cannot make those findings now because of the flaws in the models on which the ROD was based, and because of the lack of data quantifying current human exposure.

We understand that EPA expects to complete its Five Year Review by April 2017. However, the contemplated schedule does not provide sufficient time for EPA to make the necessary finding that human health and the environment are protected, particularly in the absence of (1) fish data showing current, post-dredging PCBs levels in fish, and (2) results of a fish consumption survey quantifying current human exposure. EPA should issue the Five Year Review only if it can be well-supported. EPA's Guidance indicates that the Five Year Review team be a multi-disciplinary team with relevant technical expertise to properly review the protectiveness of the remedy. *Id.* at p. 3-1, 3-2. Rather than issue a determination that lacks sufficient data and information, EPA should expedite the generation of necessary information, such as post-dredging fish and sediment data, should immediately initiate a fish consumption survey, and should involve independent experts in evaluating the flawed models.

EPA's Five Year Review process would benefit from greater formality, such as a written scope of work identifying objectives, the participants in the process and their areas of expertise, the tasks to be undertaken, areas of responsibility, a timetable for completing the tasks, and criteria for transparency in the process. We suggest that EPA's scope of work be issued for public comment so that interested parties understand how the process will proceed.

Thank you for your consideration of the foregoing. We look forward to your response and to continuing our discussions regarding the Hudson River.

Very truly yours,

Maureen F. Leary

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New York State Department of Health
Center for Environmental Health

HEALTH CONSULTATION:

1996 SURVEY OF HUDSON RIVER ANGLERS

HUDSON FALLS TO TAPPAN ZEE BRIDGE AT TARRYTOWN, NEW YORK

FINAL REPORT
February 10, 2000

CERCLIS No. NYD980763841

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Prepared under a Cooperative Agreement with

U.S. Department of Health & Human Services
Public Health Service
Agency for Toxic Substances and Disease Registry

Summary

The Hudson River PCB (polychlorinated biphenyl) site is a National Priority List (NPL) site including about 200 miles of the Hudson River between Hudson Falls (Washington County) and the Battery in New York City. PCB contamination of fish and the potential for PCBs to cause health effects led the New York State Department of Health (NYS DOH) to issue health advisories encouraging people to limit or avoid eating fish from portions of the river and New York City Harbor. The goals of this study were to measure the awareness and understanding of the fishing public (anglers) of these health advisories and to evaluate changes in this awareness since the 1991-92 survey of anglers conducted on the river by the Hudson River Sloop Clearwater organization. The results of this assessment will be used to inform decisions concerning further education and outreach activities on the Hudson.

This survey included 172 miles of the Hudson River from Hudson Falls to the Tappan Zee Bridge at Tarrytown, but did not include the New York City area, which was surveyed in 1991-92, because of the greater complexity of interviewing anglers there. The study area was divided into three areas that correspond to the different health advisories and fishing regulations. In the Upper Hudson or Area 1 (Hudson Falls to the Federal Dam at Troy), anglers must have a fishing license and fish must be returned to the river (i.e. only catch-and-release fishing is permitted). In Area 2 (the Federal Dam at Troy to Catskill), no fishing license is required, but anglers are advised to eat no fish they catch (except American shad) from this part of the river. In Area 3 (Catskill to the Tappan Zee Bridge), anglers do not need a fishing license and only infrequent eating of fish from this part of the river is advised.

In 1996, 294 anglers fishing in the study area were interviewed about their fishing habits and awareness of health advisories on eating fish from the river. This group was more than the 166 anglers who were interviewed in the same part of the river using the same questionnaire in 1991-92. Because the two studies were very similar in design and represented a similarly broad cross-section of shoreline anglers, the changes over time in angler awareness of the advisories and the factors which influence this awareness are believed to be accurately represented. These surveys do not represent the behavior of boat anglers who may represent about half of all anglers in this part of the Hudson River. The demographic characteristics of all Hudson River anglers may also not be accurately represented by these two samples of primarily shoreline anglers.

As in 1991-92, when Hudson River anglers were also interviewed, about half the anglers knew of the state health advisories. Between the two surveys, awareness of the advisories had increased for anglers fishing in the Upper Hudson River and decreased for those fishing between Catskill and the Tappan Zee Bridge. Most anglers who knew about the advisories had learned of them through publication in the fishing regulations guide provided when they purchased a fishing license. Although a license is not required to fish in the Hudson River, three-quarters of anglers fishing between Troy and Catskill and one-third of anglers fishing between Catskill and the Tappan Zee Bridge had a license. Many anglers learned of advisories through media coverage, word-of-mouth and signs posted in the Upper Hudson.

In both surveys, more than 90% of anglers surveyed said they were fishing primarily for recreation or other similar reasons, and only 6-7% of anglers said they were fishing primarily for food. However, in both surveys, between Catskill and the Tappan Zee Bridge 13-15% of anglers said their primary reason for fishing was food and, in 1996 almost half of anglers fishing in this area said food was one of their reasons for fishing. Upstream of Catskill (in Areas 1 and 2), no one included food as a reason for fishing in 1996 and only 6-7% of 1991-92 anglers said that food was a reason for fishing. In both surveys, half of anglers reported catching fish. In 1996, a

third of all anglers had kept at least some fish that they caught, but this information was not available for the 1991-92 survey. Some anglers (18%) fishing in the Upper River(Area 1) had fish when interviewed, and a few (11%) of the anglers interviewed in Area 1 had kept more than one fish, suggesting that they may eat the fish even though they did not say so when directly asked. In both surveys, about a third of anglers said that they ate fish from the Hudson and they shared their catch with again as many individuals. Most of the individuals with whom fish were shared were women and children who NYS DOH advises to eat no fish from the Hudson.

In both surveys, the most important species (by number) caught by anglers were white perch and blue crab. Striped bass, white catfish and American eel were also important. Blueback herring were important in the 1991-92 survey, but not in 1996. Blueback herring are caught primarily in April and May, before interviewing began in 1996. In 1996 the species and size of fish kept by anglers was recorded. The most important species kept by anglers (by weight and in order) were white perch, white catfish, striped bass and carp. Largemouth and smallmouth bass, bluefish and American eel were also important. These eight species accounted for 83% (by weight) of the fish kept. The weights of blue crab could not be estimated, but crabs were the second most numerous species reported caught by anglers. Eighty three percent of all the fish kept by anglers were from the Hudson River between Catskill and the Tappan Zee Bridge where women and children are advised to eat no fish and others are advised to eat no more than a meal per month of the species that are being kept.

The species of fish kept by anglers are among the species with the highest PCB levels. Using PCB data collected from 1992 to 1996 in Area 3, PCB levels in all the species exceeded the 2 milligrams per kilogram (parts per million - ppm) action level established by the US Food and Drug Administration for fish in interstate commerce. Some anglers and others who eat Hudson River fish are being exposed to PCB levels that are a health concern and are at risk of adverse health effects. Based on ATSDR's present public health category classification (Appendix F), the Hudson River PCB site is a public health hazard. This classification is chosen based on information in the March 31, 1994 Site Review and Update for the Hudson River PCBs site and on information in this health consultation that shows that anglers and others who eat fish from the Hudson River are being exposed to levels of PCBs that are a health concern.

Until PCB levels in fish from the river decline, health advisories should continue to be issued and efforts to inform the public about these advisories should be expanded. In 1999, additional community health education efforts were undertaken to inform those who still fish in the Hudson River of the health risk posed by eating contaminated fish. The following public health actions will be implemented:

1. NYS DOH will continue to evaluate new data regarding contaminants in fish and issue appropriate health advisories as needed.
2. NYS DOH will continue to work with NYS DEC and others to distribute updated versions of the NYS DOH health advisories to anglers who fish in the Hudson River, New York Harbor and other affected marine waters.
3. NYS DOH and NYS DEC will work with local communities, state and federal agencies, non-government organizations and anglers to implement effective ways to inform anglers and others who eat Hudson River fish about the health advisories and ways to reduce their health risks from eating contaminated fish.

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Background and Statement of Issue

Introduction

The Hudson River PCB (polychlorinated biphenyl) site is a National Priority List (NPL) site that includes about 200 miles of the Hudson River between Hudson Falls (Washington County) and the Battery in New York City. PCBs in the site have contaminated sediments, water and fish. Because the PCB levels in fish exceed the US Food and Drug Administration action level for PCBs and because eating these fish is a health concern, the New York State Department of Health (NYS DOH) has issued health advisories. These advisories caution people to limit or avoid eating fish from the site and to limit eating striped bass, bluefish and eels from marine waters at the mouth of the Hudson River.

For almost thirty years, the General Electric Company (GE) discharged PCBs into the Hudson River from two capacitor manufacturing facilities at Hudson Falls and Fort Edward, New York (Sofaer, 1976). These discharges probably began as early as 1947 when the Fort Edward facility began operation and were substantially ended in 1977 when GE stopped using PCBs (Horn *et al.*, 1979). However, more recent information suggests that PCBs are seeping into the river from the Hudson Falls plant site (O'Brien and Gere, 1994 and 1998).

In late 1975, the New York State Department of Environmental Conservation (NYS DEC) discovered elevated PCB levels in fish from the Hudson River (Spagnoli and Skinner, 1977). In February 1976, the NYS DEC issued regulations prohibiting all fishing in the Upper Hudson River (from Hudson Falls to the Federal dam at Troy) and prohibiting commercial harvest of most fish from Troy to New York City. The public was advised to eat no fish from the Upper Hudson and to restrict consumption of fish from the rest of the river. These advisories were subsequently modified on several occasions as new data suggested that additional advisories were needed or that existing advisories could be relaxed. In 1985, the advisories were extended to striped bass caught in marine waters and commercial harvest of striped bass in marine waters was prohibited. Appendix D provides a more detailed chronology of events related to PCB contamination of the Hudson River.

For several years, the U.S. Environmental Protection Agency has been reassessing its 1984 interim decision to take no remedial action for the PCB-contaminated sediments in the Upper Hudson River (Area 1). The ecological and human health risk assessments are scheduled to be completed by August 1999. A proposed remedial action plan is currently scheduled for the end of 2000 and a Record of Decision is planned for June 2001. The most important issue is whether, in the foreseeable future, PCB levels in fish will diminish to a point that health advisories are no longer needed without additional remedial action. The known sources of PCBs to the river at the two General Electric facilities have been substantially reduced, but continued low-level releases from the facility sites and/or releases from heavily contaminated river sediments, primarily upstream of the Thompson Island Dam, may be large enough to contaminate fish for many years or could become a significant source of fish contamination in the future.

In 1989, the Agency for Toxic Substances and Disease Registry (ATSDR) completed a health assessment for the site (ATSDR, 1989). This assessment recognized that eating PCB-contaminated fish is the primary exposure pathway of concern to human health. In 1994, the NYS DOH completed a site review and update committing, among other things, to 1) continue community health education; 2) review and revise the consumption advisories; and 3) work with NYS DEC to distribute updated versions of the health advisories to anglers who fish in the Hudson River, New York City harbor and marine waters (NYS DOH, 1994). In 1996, NYS

DOH issued a Public Health Action Plan Update which reiterated these commitments and noted that new brochures were being distributed to Hudson River anglers, particularly targeted at minority and low-income groups who are less aware of the advisories (NYS DOH, 1996). In addition, the update noted that "NYS DOH [was] investigating Hudson River angler's exposure to PCBs from [eating] fish and assessing angler awareness of the advisory."

For many years, NYS DEC and NYS DOH have publicized the health advisories through annual press releases and publication in NYS DEC's fishing regulations guide given to each licensed angler. In recent years, about one million anglers purchase a fishing license each year. Since 1984, NYS DOH has published an annually revised brochure containing the statewide health advisories and additional background information. In recent years, about 20,000 copies have been distributed each year. NYS DOH staff have met with many groups to explain the advisories and county health department staff have also provided information to individuals and groups. From 1994 to 1997, NYS DEC and NYS DOH increased efforts to inform anglers in the Hudson River valley about the health advisories. The NYS DEC effort was focused on minority anglers, most of whom were unlicensed and fishing in the river downstream of the Troy dam, who appeared to be less aware of the advisory. NYS DOH distributed booklets and brochures to local health units, state parks, bait and tackle shops and a number of other groups that might provide information to unlicensed anglers.

In August 1995, fishing regulations were changed to permit catch-and-release fishing in the Hudson River between Hudson Falls and the Troy dam, a portion of the river where fishing had been prohibited since February 1976. With the 1995 change in regulations, a special brochure was prepared and distributed and signs were posted at fishing access points throughout this portion of the river informing anglers of the new regulations and PCB contamination of the fish.

Community Health Concerns

Many individuals and community groups have expressed concern that people are being exposed to unsafe levels of PCBs by eating fish from the Hudson River because many people are not aware of the advisories and others do not follow the advice. In 1991 and 1992, the Hudson River Sloop Clearwater (a not-for-profit environmental education organization in Poughkeepsie, NY) used volunteer staff to interview anglers who were fishing on the Hudson River between Hudson Falls and Staten Island about their fishing habits and awareness of health advisories. The survey found that many Hudson River anglers were not aware of the consumption advisories and others who were aware did not heed the advice (Barclay, 1993). The report highlighted health concerns for people who were eating fish from the river, particularly women of childbearing age and children under the age of 15 who appear to be at particular risk, for non-whites and for low-income people. The author concluded that the prohibition of fishing in the Upper Hudson River and the health advisories were "having only limited success in preventing unsafe levels of exposure to PCBs through consumption of Hudson River fish." The report included thirteen recommendations for improving angler awareness of, and adherence to, the health advisories, including both educational and research efforts.

In New York City, anecdotal reports have expressed concern that minorities and non-English-speaking immigrants are eating contaminated fish from the Hudson River and other waters around New York City. Some reports suggest that subsistence fishing is a concern. Others have suggested that signs and other educational materials need to be available in many languages, e.g. Spanish, Russian, Polish, Hmong, Chinese.

During the public review of this report (March 19, 1999 to May 1, 1999), several concerns were voiced in two written comments, one from an angler and the other from a public agency. Responses to these comments can be found in Appendix E.

Study Objectives

This report describes the results of a resurvey of Hudson River anglers conducted in the summer and fall of 1996. Specifically, the objectives of this study were to:

- measure awareness of the health advisories among Hudson River anglers,
- measure angler understanding of the health advisories,
- measure whether the advisories influenced fishing behavior or whether anglers eat fish,
- assess what characteristics of anglers might contribute to lack of awareness or understanding of or compliance with the advisories, and
- assess whether awareness, understanding or compliance had changed among Hudson River anglers between 1991-92 and 1996.

Site Description and Study Area

Excluding the Niagara and St. Lawrence Rivers on New York's northern border, the Hudson River is New York's largest river, with a watershed of 13,390 square miles. The Hudson River PCB site is the National Priority List (NPL) site which includes 192 miles of the Hudson River between Hudson Falls (Washington County) and the southern tip of Manhattan (Battery Park) in New York City. This survey included 172 miles of the Hudson River from Hudson Falls to the Tappan Zee Bridge (Figure 1).

For this survey, the study area was divided into areas that correspond to the different health advisories and fishing regulations:

- Area 1 - Hudson Falls to Federal Dam at Troy
- Area 2 - Federal Dam at Troy to Catskill
- Area 3 - Catskill to Tappan Zee Bridge

The advisories for Area 3 extend into the New York City Harbor waters, but because of logistical problems, the survey did not include anglers in the New York City area.

In Areas 1 and 3, the advisory and fishing regulations were different at the time of the two surveys (Table 1). In Area 1, fishing was prohibited in 1991-92, but catch-and-release fishing with a fishing license was permitted in 1996. In Area 3, the advisory recommended that no fish except American shad be eaten in 1991-92. By 1996, anglers in Area 3 were advised to restrict eating many fish to no more than one meal per month, but the advisories for women of childbearing age and children remained to eat no fish or crabs (Table 1).

Physically, the river between Hudson Falls and Troy (Upper Hudson River) is quite different from the estuarine portion of the river downstream of Troy. In the Upper Hudson River (Area 1), eight dams make the river navigable to barges and other large boats. These dams create pools which are good habitat for a variety of warmwater fish species. The dams have also slowed the downstream movement of PCB-contaminated sediments. Fish in the pool behind the first dam downstream of Hudson Falls (Thompson Island Dam) are the most heavily

Figure 1. Map of study area.

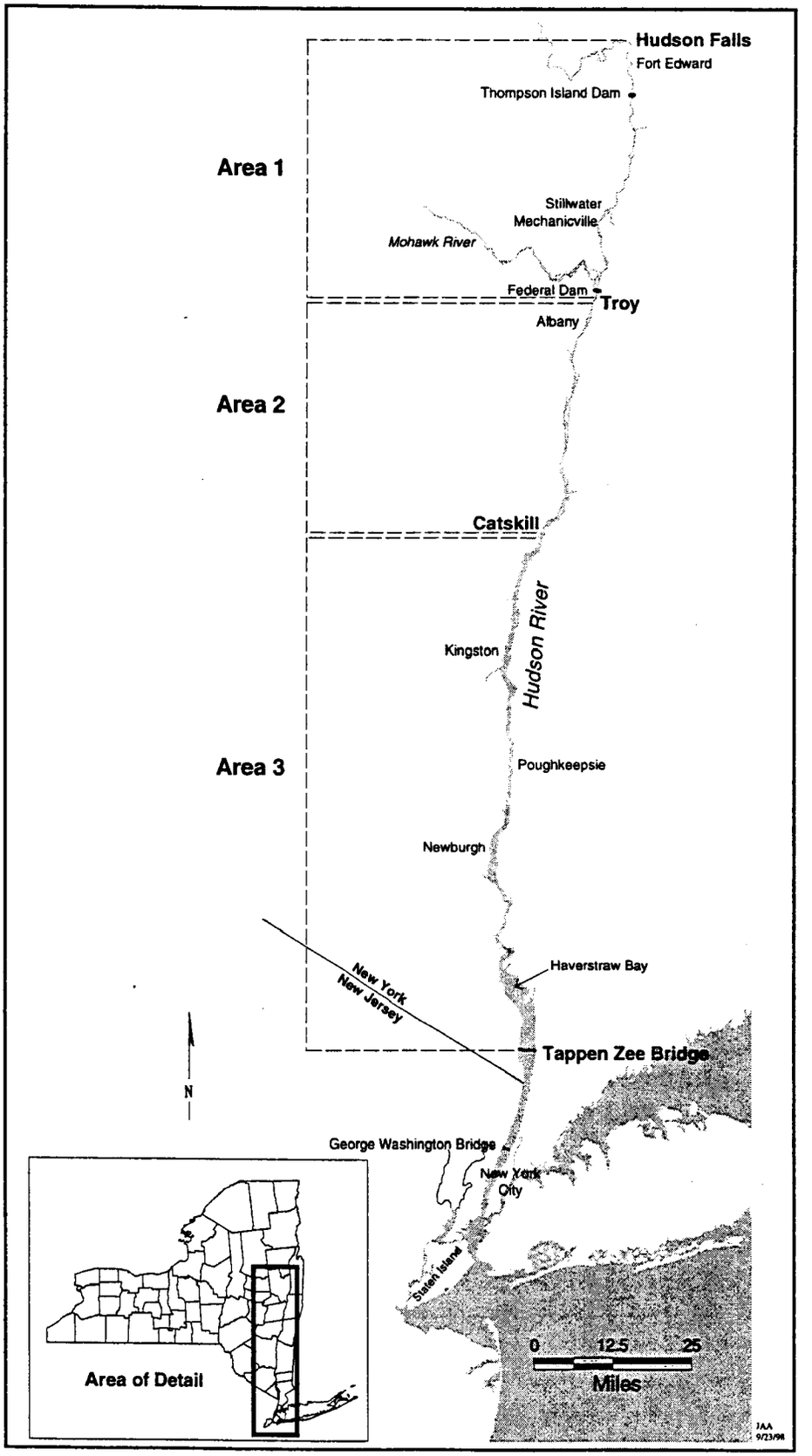

Table 1. Fishing regulations and health advisories for the Hudson River in 1991-92 and 1996.

Area of River	1991-92	1996
Area 1 Hudson Falls to Troy	Fishing prohibited. Eat NONE of any species.	Fishing permitted with license[1], possession of fish prohibited. Eat NONE of any species.
Area 2 Troy to Catskill	Fishing permitted, no license[1]. Eat NONE of any species except American shad.	Fishing permitted, no license[1]. Eat NONE of any species, except American shad.
Area 3 Catskill to Tappan Zee	Fishing permitted, no license[1]. Eat NONE of any species except American shad.	Fishing permitted, no license[1]. Infrequent eating advised[2].

[1] North (upstream) of Troy, a state fishing license is required to fish. No license is required to fish in the tidal portion of the Hudson (south or downstream of Troy).

[2] Women of childbearing age, infants and children under the age of 15 are advised to EAT NONE of any species. Other anglers are advised to eat NO MORE THAN SIX PER WEEK for blue crabs and to EAT NONE of the blue crab hepatopancreas (mustard, tomalley or liver); to eat no more than ONE MEAL PER MONTH for American eel, Atlantic needlefish, bluefish, carp, goldfish, largemouth and smallmouth bass, rainbow smelt, striped bass, walleye, white catfish and white perch; For other species, anglers are advised to eat no more than ONE MEAL PER WEEK.

contaminated, and species which anglers catch include largemouth and smallmouth bass, carp, brown and yellow bullhead, yellow perch, white sucker and several sunfish, e.g., pumpkinseed, bluegill, red-breast sunfish, rock bass (see Appendix A for scientific and common names of fish reported in these surveys to have been caught by anglers). Throughout the Upper Hudson River, fish communities are similar to one another, although American eels, white perch, blueback herring and alewife are found in the river near Troy but are not a significant component of the fish communities further up-river. Immediately upstream of the Federal dam at Troy, the Mohawk River joins the Hudson, increasing water flows by 85%.

Downstream of the Federal dam at Troy, the Hudson is an estuary subject to daily tidal cycles. Upstream of Poughkeepsie, the river is always fresh water and downstream of the George Washington Bridge at New York City the river is essentially marine throughout the year. In the estuary (Areas 2 and 3), the fisheries include a number of species not found to any significant extent in the Upper Hudson River, e.g. striped bass, American shad, white catfish, and blue crab. In the Haverstraw Bay/Tappan Zee region of the river, Atlantic tomcod and bluefish are also caught.

Methods

Survey methods

The 1996 survey used the same questionnaire and interviewing technique (with a few minor modifications) as was used in a 1991-92 survey of Hudson River anglers conducted by the Hudson River Sloop Clearwater organization (Barclay, 1993). Trained interviewers visited known fishing access sites along the Hudson River and asked anglers a series of questions (see Appendix B for a copy of the 1996 survey instrument). In 1996, the questionnaire was the same,

except that a question in the 1991-92 survey about how fish were prepared for eating was deleted and a question was added at the end of the interview that asked whether the angler had a fishing license. In the 1996 survey, interviewers identified, counted, and measured the total length of each fish being kept by anglers. In the 1991-92 survey, the species and number of fish caught were reported, but not their sizes or whether they were kept.

In 1996, an interviewer was assigned to each of the three study areas. During the course of the survey two different individuals conducted interviews in Areas 1 and 2. The interviews were conducted from early June until the end of October. Efforts were made to interview anglers on weekdays as well as weekends and at various times of the day. In 1991-92, 14 interviewers were employed to question anglers, and efforts were made to interview anglers at different times of the week and day.

The 1996 survey did not extend into the New York City metropolitan area as the 1991-92 survey did, because logistical concerns (e.g. finding qualified, multilingual interviewers; supervising their work; ensuring interviewer safety) could not be satisfactorily addressed when the survey was being planned. In the 1991-92 survey, the interviewers visited 20 different sites, but six of the sites were south of the Tappan Zee Bridge, so only 14 sites in Areas 1-3 were visited. During the course of the survey in 1996, interviewers visited the same 14 sites and an additional 18 sites. Very few anglers refused to participate, although some anglers did not respond to all questions. A few anglers did not speak English, but in almost all cases others who were fishing at the same location were able to translate for the interviewer. The actual number of these non-responders was not recorded, but they were few in number.

Data Management and Statistical Analysis

Responses from both surveys were coded and double entered into dBase IV databases. Inconsistencies were corrected and the data then transferred to SAS. Initial data analysis identified a few coding errors, and these were corrected before analysis began. Careful examination of outliers during subsequent data analysis also uncovered a few additional coding errors. The initial analyses described the cohort demographics with calculated distributions of subjects by age, gender, race/ethnicity, income, household number, and possession of a fishing license. Next, specific hypotheses were tested using summary statistics of univariate and bivariate analyses. All the tests were performed using SAS programs (SAS Institute, Cary, North Carolina). To further examine the relationship between respondents' demographic characteristics and their knowledge of health risks and water pollution, a stepwise logistic regression model was used with a 95% confidence level ($p<0.05$) for including independent variables.

Results and Discussion

In both surveys, several factors may have influenced whether the results represent anglers on the river. Ideally, anglers would have been surveyed randomly. However, the interviewing effort could not be truly randomized for date, day-of-week or time-of-day as the interviewers were part-time employees and data which would permit designing a rigorous random sampling plan were not available. Additionally, in 1996, the surveys did not get underway until June, and therefore, anglers who were fishing for American shad or striped bass in April and May, when these fish are most available in the river, were not surveyed. In both surveys, only a few boat anglers were interviewed. In May through August 1990, Green and Jackson (1991) surveyed 678 anglers fishing in the Hudson River between Stuyvesant and Kingston. They found that about half of the

angler fishing effort is from boaters. Boat anglers were found to target largemouth and smallmouth bass to a greater extent than shore anglers. In April through June 1997, Peterson (1998) assessed the striped bass fishery by aerial survey and interviews of more than 2,700 anglers. He found that 71% of angling effort and 84% of the striped bass catch was from boat anglers. Because neither the 1991-92 nor the 1996 survey interviewed very many boat anglers, the results do not adequately represent the behavior of this group.

In 1996, fewer interviews were conducted in June than in other months and most (52%) were conducted in August and September. During the 1991-92 survey, about half of interviews occurred in June and July and about 18% were conducted in May. About half of the interviews occurred on weekends in both studies. In 1996, most (51%) were conducted in the afternoon or evening (33%) and fewer (16%) were conducted in the morning. In the 1991-92 survey, the interviews were evenly distributed among morning, afternoon and evening. As noted above, during both surveys, the distribution of interviews by date, day and time may have been influenced by the interviewers availability rather than when anglers were actually fishing. Table C-1 summarizes these data (Appendix C).

In 1996, 38 different fishing locations were visited, 8 locations in Area 1, 9 locations in Area 2 and 21 locations in Area 3. A total of 294 questionnaires were completed, with almost half (48%) of the interviews being conducted in Area 3 (Table C-2). The other interviews were evenly divided among Areas 1 and 2. In 1991-92, 323 questionnaires were completed, but at the 14 sites in Areas 1, 2, and 3 (1 in Area 1, 3 in Area 2 and 10 in Area 3), 166 questionnaires were completed. The interviews were more evenly distributed among the areas than in the 1996 survey (each of the three areas had between 30% and 36% of completed questionnaires).

Within each of the three areas, the 1996 survey locations were more broadly distributed than in the 1991-92 survey (Table C-2). For example, in the 1991-92 survey, almost all (82%) of the interviews in Area 1 were from the Mechanicville to Stillwater portion. No interviews were conducted near Catskill (Area 2) or in Haverstraw Bay (Area 3). In 1996, 92 interviews (31% of all interviews) were conducted in portions of the river where no anglers were interviewed in 1991-92.

As discussed earlier, few boat anglers were interviewed and other studies indicate that they may constitute half or more of the fishing effort along the river. So, these surveys do not represent the behavior of boat anglers. These surveys did interview shoreline anglers over a broad range of fishing access sites in each of the three study Areas. The interviews were reasonably evenly distributed over the months surveyed, weekdays and weekends, and time-of-day. These data may not accurately represent the behavior of all shoreline anglers, but because the studies were very similar in design and represented similarly broad cross-sections of the angling public, the changes over time in angler awareness of the advisories and the factors which influence angler awareness are believed to be accurately represented.

Demographics

Respondents to this survey and to the 1991-92 survey had a similar distribution of age; but the distribution of genders, race and income were significantly different between the two surveys ($p<0.05$ by χ^2, see Table C-3). The majority of anglers were male in both surveys (87-93%), but women were more common in the 1996 survey (13%) than in the 1991-92 survey (8%). This difference was not statistically significant ($p>0.05$ by χ^2). In both surveys, most anglers were Caucasian (69-80%). African-Americans were represented equally in the two surveys (12-15%), but in this survey a greater proportion of the anglers were Hispanic (13% versus 4%). Asian,

Amerindian and East Indian anglers represented only 2-5% of the anglers in each survey. A large proportion (13-26%) of anglers did not provide their income bracket, but of those who did respond, almost half had annual incomes less than $30,000 and about 10% reported incomes of $50,000 or more. In 1996, about 41% of respondents from Area 3 declined to provide income information, considerably more than in the other areas (5% and 20%) and than in 1991-92 (10-16% for each of the three areas). In both surveys, about half of the anglers reported living in households of 2 or 3 people. Household sizes ranged from 1 to 12.

Awareness and understanding of the advisories

Several questions on these surveys (Questions 20, 28, 29, 31a and 31b) were designed to show how knowledgeable anglers were about fish contamination and water pollution prior to asking whether the angler were aware of "official health warnings" (Question 32). If respondents were aware of the advisories, they were asked how they learned about the advisories (Question 34) and whether and how they had changed their fishing or eating habits in response to learning of the advisories (Questions 37 and 38).

Responding to Question 32, about half of anglers said they knew of health warnings (51% in 1991-92 and 49% in 1996, see Table C-4). In 1991-92, awareness of the health advisories did not differ among the Areas, with 55% of respondents in Area 1 and 3 reporting that they knew of the health warnings and only 42% of anglers in Area 2 saying so. In 1996, the differences among the Areas were more dramatic, ranging from 75% of anglers in Area 1 aware of the health warnings to only 31% of anglers in Area 3. About 58% of anglers in Area 2 were aware of the health warnings. Other interesting patterns include:

1. License-holders were much better informed than unlicensed anglers. About 73% of anglers with a fishing license were aware of the health advisories and only 18% of unlicensed anglers knew about them.
2. In 1996, ethnic minorities were less informed than whites (13-22% compared to 63%). In the earlier survey, awareness of the advisories was more similar between minorities and whites (43-67% for minorities and 50% for whites). However, the observations for 1991-92 are based on only 34 minority responses compared to 90 minority responses in 1996, and therefore this difference may just be the result of the small sample size in 1991-92.
3. In both surveys, men were more aware that the health advisories exist than women (53-54% for men versus 18-27% for women).
4. In general, low-income respondents (less than $10,000 annual income) were less aware of the health advisories than the others (21-34% compared to 49-75%).
5. Age did not appear to dramatically influence awareness. However, in both surveys the 35-44 year-old respondents were somewhat more likely to be aware of the advisories (56% compared to 40-51% for other ages in 1996 and 72% compared to 17-52% for other ages in 1991-92). Also, in the 1991-92 survey, only 17% of respondents less than 24 years old were aware of the health advisories; but in 1996, 41% of this group were aware of the health advisories. For the other age groups, 40-72% of respondents were aware of the health advisories in both surveys.

The principal mechanism for informing New York anglers about health advisories for sportfish has been through the regulations guide provided when each licensed angler in the state purchases their license. The 1991-92 survey did not ask whether the angler had a fishing license. In 1996, somewhat more than half (58%) of the anglers said that they had a license (Table C-3). However, this percentage varied considerably by area of the Hudson River with 86% of anglers

in region 1 (where a license is required for anglers 16 years and older) saying they had a license. In Area 3, only 32% of anglers had licenses. Some people interviewed in Area 3 did not understand the concept of a fishing license.

In both surveys, the greatest proportion of respondents (37% in 1991-92 and 44% in 1996) became aware of the health advisories by reading them in the regulations guide distributed with fishing licenses (Table C-5). In 1991-92, media (35%) and word-of-mouth (24%) were the only other important sources of information. Media was the primary source of information for the majority of respondents (51%) in Area 3, and the regulations guide was more important in Areas 1 and 2. In 1996, media, posters (signs) and word-of-mouth were the source of awareness for 14-18% of respondents, but the regulations guide was the most important. In Area 1, signs were the second most important source of awareness of health advisories. Even in Area 2, where no signs have been posted, 16% of anglers reported that they became aware of health advisories from postings. The boundary between Areas 1 and 2 is an urban area and some anglers who fish in Area 2 may also fish in Area 1.

Three slightly different questions asked about potential health risks from eating fish. Question 20 asked if any fish in the immediate area of where the person was fishing were "not safe to eat". Question 28 asked if eating fish "poses a serious risk", "a slight risk" or "no risk"; and Question 31b asked "do you believe that eating fish caught at this site would pose a risk to your health?" Two other related questions asked anglers whether they thought the water was polluted (Question 29) or the fish contaminated (Question 31a) where they were fishing. If one looks at the responses of individuals who were consistent in their response to all five of these questions (i.e. answered "yes" to Questions 20, 31a and 31b and thought eating fish posed at least a slight risk and the water was at least slightly polluted), almost half (42%) of respondents responded affirmatively and only 4% consistently said that there was no pollution, no fish contamination and no health risks (Table C-6). In 1991-92, the responses were quite similar with 40% responding affirmatively and only 2% consistently denying any problem or concern. However, in 1996, the responses differed considerably among the areas. About 76% of respondents in Area 1 were consistent in affirming these concerns, while many fewer in Area 2 (46%) and Area 3 (22%) thought so. No one in Areas 1 or 2 consistently denied any problem or concern, but in Area 3 almost 9% of the respondents were consistently unconcerned. In the 1991-92 survey, the responses did not differ much by area, ranging from 33% to 52% for positive responses in each area and 2 to 4% for negative responses in each area.

In both surveys, about half of the individuals who said they were aware of the health advisories also consistently responded in the affirmative on these five questions and no one aware of the health advisories consistently denied any problem or concern. Broadly speaking, in both surveys, few black and Hispanic respondents (10-28%) consistently responded that there was a concern and 10% or fewer consistently denied any problem or concern. Income and age did not appear to influence perceptions of health risk or river pollution in any consistent way.

Multivariate analyses by means of logistic regression were performed to evaluate whether these and other demographic factors were significantly associated with responses to these five questions. The preliminary assessment suggests that, at the 95% confidence level,

- anglers who were older than 45 years had less knowledge of pollution, fish contamination and health risk issues than those who were younger,
- respondents in Area 2 and 3 had less knowledge of these concerns than those in Area 1 and
- anglers who were aware of the advisories were more likely to consistently respond affirmatively to the five questions.

This analysis suggests that gender, race, income and possession of a fishing license were not statistically significant factors in an anglers response to the five questions about pollution and health risks. Differences between surveys are probably the result of differences in demographic characteristics between the two surveys.

Although the percentages of responses were not exactly the same for the individual questions, the patterns described above were generally the same for responses to each of the five questions separately as for the combined questions.

What are Hudson River anglers catching?

Anglers were asked to identify what type of fish they were trying to catch (Question 3) and what fish they had caught (Question 5). Many anglers did not know the identity of the fish that they were catching and in some cases identifying the species referred to was difficult. For example, in some instances, names could not be made more specific than "catfish", which can mean white catfish or brown or yellow bullhead. Others like "bass" could refer to largemouth or smallmouth bass or striped bass. In the 1996 survey, when anglers had kept fish, the interviewers recorded the species, number and length of each fish.

Slightly more than half of all anglers surveyed (58% in 1991-92 and 53% in 1996) reported catching fish (Table C-7). In 1991-92, respondents in Area 1 were somewhat more successful than those from Area 2 & 3 (66% compared to 52% and 57%). But in 1996, Area 3 anglers were the most successful (70% compared to 32% of Area 1 anglers and 42% of Area 2 anglers). In the 1991-92 survey, the fish kept by anglers were not reported separately from those said to be caught. In 1996, only 30% of anglers had fish when interviewed. More than three-quarters (76%) of these anglers were fishing in Area 3 where almost half (47%) of anglers there had kept fish. In Area 1, where NYS DEC regulations prohibit keeping fish, 14 anglers (18% of those interviewed in this area) had fish when they were interviewed.

In the two surveys, 25 species of fish were reported to be caught in the three areas combined (Table C-8). About half of the total number of fish caught were white perch, blue crab or striped bass, and only 10 species account for almost 90% of the numbers caught. In 1996, anglers had kept 17 different species (Table C-9). The greatest number of anglers (45% of anglers who kept fish) had kept white perch. Striped bass, American eel, white catfish, bluefish and smallmouth bass were kept by between 10 and 17 anglers each (12-20% of anglers who kept fish). The total weights of each fish were estimated from published regressions based on length (Table C-10). The weights of all the fish kept by anglers were summed for each species and area (Table C-9). Overall, white perch was the most important species (comprising 22% of the catch by weight), followed closely by white catfish (16%) and striped bass (14%). Carp (12%), largemouth and smallmouth bass (7% each), bluefish (7%) and American eel (6%) were also somewhat important contributors to the overall catch which was kept by anglers. These eight species account for 91% of the catch by weight. Striped bass are probably more important than represented by the 1996 survey, because interviews were not conducted in April and May when the striped bass fishery is very active. In addition, blueback herring and shad are caught primarily in April and May, and their importance was therefore under-represented in the 1996 survey.

What are Hudson River anglers doing with the fish they catch?

Anglers identified up to three reasons why they were fishing. In 1996, most anglers (91%) said their primary reason for fishing was some form of "recreation", and only a small proportion

(6%) of anglers listed fishing for food as the primary reason that they were fishing (Table C-11). All of the anglers who said that they were fishing for food were actually fishing in Area 3, where they comprised 12% of anglers. Less than one quarter (23%) of all anglers included food among the reasons for fishing, but again, all of these anglers were in Area 3. Thus, almost half (47%) of anglers in Area 3 said that food was one of the reasons that they were fishing. No one in Areas 1 and 2 included food as a reason for fishing. In 1991-92, the overall responses were similar; 7% of anglers listed fishing for food as the primary reason that they were fishing. Some of these anglers were from Areas 1 and 2 (1% of all respondents). Only 16% included food among the reasons for fishing. Again, a greater proportion of anglers in Area 3 included fishing for food as a reason for fishing.

In 1996, most anglers (93%) said that they sometimes or often returned fish to the river (Table C-12). About one-third of anglers (36%) reported sometimes or often eating fish they caught from the Hudson. Some of these anglers were from Area 2 (7 anglers, 9% of respondents in Area 2), but everyone in Area 1 responded that they never ate fish they caught from the river. In Area 3, 70% of anglers reported sometimes or often eating fish from the Hudson. As noted earlier, fourteen anglers in Area 1 (18% of those interviewed in Area 1) had fish in their possession when they were interviewed. Eight of the anglers had only one fish and two anglers had three fish. Each angler said he was going to return the fish to the river, but the interviewer did not check to see that the fish were returned. Most of the 24 fish that were kept were largemouth or smallmouth bass or bluegill. Two of the bluegills were only 4 inches long and another bluegill, the rock bass and the striped bass were only 6 inches long. The other fish were all 8 inches or larger and one largemouth bass was 16 inches long. It seems likely that at least some of the fish caught in Area 1 were kept for eating. The six anglers who had more than one fish were 11% of the anglers interviewed in Area 1.

Almost one in four anglers (23%) sometimes or often gave away the fish they caught, and about 35% of anglers at least rarely gave away fish. Some of these anglers were fishing in Area 2, but everyone in Area 1 said they never gave fish away. In Area 3, almost half (46%) of anglers said they often or sometimes gave fish away, and about 65% said they gave fish away at least rarely.

Overall, only a very few individuals (two, less than 1%) reported sometimes or often selling fish, and four others (1%) said they sold fish rarely. A few individuals (4 individuals or 1%) in Area 3 did not respond when asked if they sold fish.

About 30% of anglers reported using the fish they caught for bait at least rarely. All of these anglers were in Areas 2 and 3. Very few anglers said they used the fish they caught for fertilizer (5 individuals or 2%) or threw them in the trash (9 individuals or 3%) sometimes or rarely.

In 1991-92, the pattern of responses was similar, but a greater proportion of anglers said they ate, gave away or sold the fish they caught (Table C-12). The difference in proportion of anglers who said that they ate their catch was not statistically significant, but the greater proportion who gave away or sold their fish in 1991-92 was statistically significant ($p<0.05$ by χ^2). A greater proportion of these individuals were fishing in Areas 1 and 2. All but one individual (99%) said they often or sometimes returned fish to the river. About 30% of anglers said they often or sometimes ate the fish they caught, and several were fishing in Area 1. About 40% of anglers responded that they sometimes or often gave fish away, and a few (7 individuals or 5%) said they sold fish at least rarely. Some of the anglers who said they gave fish away were fishing in Area 1. Another 12 individuals (7%) provided no response when asked if they sold fish and half of these individuals were fishing in Area 1.

In both surveys, women where less apt to eat fish from the Hudson than all anglers. In 1996,

10 of the 37 women surveyed (27%) said they ate fish from the river at least rarely. In 1991-92, two of the eleven women surveyed (18%) ate fish. In 1996, 45% of all anglers surveyed in 1996 and 40% of all anglers surveyed in 1991-92 reported eating fish from the river.

How often are Hudson River anglers eating the fish they catch?

The 133 anglers (45% of those surveyed) who said they ate fish at least rarely were asked how frequently they had eaten fish or crabs from the Hudson River during the last week (i.e. in the last 7 days) and last month (i.e. in the last 30 days). More than half (57%) had not eaten Hudson River fish or crabs in the last week and about a quarter (26%) had not eaten fish in the last month (Table C-13). About half of anglers (i.e. the median angler) who ate fish from the Hudson ate 2 meals or less in the previous month. Five percent of anglers (95th percentile consumers) reported eating 3 meals or more in the past week and 12 meals or more in the past month. Four individuals (3% of anglers who reported eating fish) said they had eaten 20 meals in the previous month.

In 1991-92, 66 anglers (40% of those surveyed) said they ate fish or crabs from the Hudson River at least rarely. More than half (65%) had not eaten fish from the Hudson in the previous week and about half (52%) had not eaten fish or crabs in the last month. Thus, the median angler ate less than one meal in the past month from the river. Five percent of anglers reported eating 3 meals or more in the past week and 10 meals or more in the past month. One individual said that he had eaten 30 meals of fish or crabs from the river in the previous month.

In 1991-92, anglers were advised to eat no fish from the study areas, so the 40% of anglers who said they ate fish were not complying with the advisories. In 1996, all anglers were advised to eat no fish caught from the river upstream of Catskill. For many species caught from the river downstream of Catskill (Area 3), men are advised to eat no more than one meal per month and women and children are advised to eat no fish. Compliance with these advisories is more complicated to calculate. The 15 anglers who said they ate fish from Area 2 and the 76 anglers who said they ate more than a meal per month of fish from Area 3 clearly ate more than is advised (Table C-13). Of the 42 individuals who said they ate one meal or less per month, five were women or younger than 15. Thus, in 1996, 96 anglers (33% of all respondents) were not following the advice provided in NYS DOH advisories. Most of these anglers (81 of the 96) were fishing in Area 3, so more than half (57%) of Area 3 anglers were eating more than is advised.

Are others eating Hudson River fish?

As noted above, about half of all anglers (45% in 1996 and 49% in 1991-92) said they gave fish away to others at least rarely (Table C-12). Most of the anglers who said they gave fish away (90% in 1996 and 85% in 1991-92) thought that the fish were eaten (Table C-14). Very few anglers acknowledged selling fish (2% in 1996 and 5% in 1991-92) or might have sold fish, assuming those who did not respond probably sold fish at least rarely (another 1% in 1996 and 7% in 1991-92).

Compared to 1991-92, many fewer anglers in 1996 said they gave fish away but did not eat the fish themselves (Table C-15). In 1996, about 8% of anglers who said they gave fish away said they did not eat the fish they caught. In 1991-92, 39% of anglers who gave fish away did not eat the fish themselves.

Anglers who responded that they ate fish at least rarely were also asked if they shared fish

that they caught with others. Some anglers were expected to share fish with family members or others that would not have been considered "given away". In both surveys, about two thirds of anglers who ate their fish (68% in 1996 and 64% in 1991-92) said that they did share fish (Table C-16). In 1996, 90 anglers shared fish with 108 other individuals, 70 of whom (65%) were women of child-bearing age and children under the age of 15. In 1991-92, 42 anglers shared fish with 96 other individuals, 42 of whom (44%) were women and children. Women of child-bearing age and children under the age of 15 are advised to eat no fish from these parts of the Hudson River (Table 1).

Environmental Contamination and Adult and Children's Health Concerns

Since 1976, NYS DEC has monitored PCB levels in fish from the Hudson River and marine waters, including the New York City Harbor (Skinner *et al.* 1996, Sloan *et al.* 1984, Sloan and Horn 1986, Sloan and Armstrong 1988, Sloan, Stang and O'Connell 1988, Sloan *et al.* 1988, Sloan and Hattala 1991, Sloan 1994, Sloan *et al.* 1995). For most of this time, some species have been collected annually from about ten different locations throughout the study area (Hudson Falls to the Tappan Zee Bridge). Generally, a standard filet is removed from each fish and each filet analyzed separately for PCBs.

In general, PCB levels in fish were quite elevated when they were first measured. With control of active discharges in the late 1970's, fish PCB levels declined precipitously for several years and continued a very slow decline until they increased dramatically in response to apparently fresh or increased discharges of PCBs near the General Electric facility at Hudson Falls which were discovered in late 1991. Since 1993, fish PCB levels have again generally begun to diminish, but they remain quite elevated (Table C-17). From 1992 to 1996 in Area 1, average PCB levels have ranged from about 6 to 61 milligrams per kilogram wet-weight or parts per million (ppm), depending on species. Largemouth and smallmouth bass comprised 58% by weight of the fish that were kept and averaged 15 and 8.0 ppm, respectively. In Areas 2 and 3, average PCB levels in fish range from less than 1 ppm to about 9 ppm. In Area 2, largemouth and smallmouth bass comprised 81% by weight of the fish that were kept and averaged 4.9 and 7.6 ppm, respectively. In Area 3, the catch was more varied. The most important six species represent 77% by weight of the fish that were kept: white perch (3.9 ppm), white catfish (8.0 ppm), striped bass (2.2 ppm), carp (no PCB data), largemouth and smallmouth bass (no PCB data). Fish that exceed the US Food and Drug Administration tolerance of 2 ppm cannot be sold in the marketplace.

PCBs cause cancer in laboratory animals exposed to high levels over their lifetimes (ATSDR, 1997). Chemicals that cause cancer in laboratory animals may also increase the risk of cancer in humans who are exposed to lower levels over long periods of time. Whether PCBs cause cancer in humans is not known.

PCBs also produce a variety of noncarcinogenic effects, primarily to the skin, liver, and to the nervous, immune and reproductive systems. Some PCBs cause birth defects in offspring born to animals exposed to high levels during pregnancy. Some studies of pregnant women suggest a link between a mother's increased exposure to PCBs from eating contaminated fish or from other environmental sources and slight effects on her child's birth weight, short-term memory, and learning. A recent study suggested that women who eat contaminated fish have slightly shorter menstrual cycles. In all these epidemiological studies, the women were also exposed to other chemicals and the effects of these chemicals on them and their children are not understood. Overall, the data from animal and human studies suggest that the fetus and newborns may be

more sensitive to PCBs than adults.

As noted above (p. 1), a previous health assessment noted that the primary exposure pathway of concern to human health was eating fish. The risks of health effects from eating fish depend primarily on contaminant concentration in the fish, how often an angler eats fish and for how many years. The available data clearly indicate that some anglers and others who eat fish from the Hudson River are being exposed to levels of PCBs that are a health concern and are at risk of adverse health effects.

Conclusions

Results from this survey and the previous one done in 1991-92 by the Hudson River Sloop Clearwater organization (Barclay, 1993) may not accurately represent all Hudson River anglers. Very few boat anglers were surveyed and information was not available to develop a random sample of the shoreline anglers. Nonetheless, a number of conclusions can be reached from the responses to these surveys:

1. Based on ATSDR's present public health category classification (Appendix F), the Hudson River PCB site is a public health hazard. This classification is chosen based on information in the March 31, 1994 Site Review and Update for the Hudson River PCBs site and on information in this health consultation. The health consultation provides data showing that anglers and others who eat fish from the Hudson River are being exposed to levels of PCBs that are a health concern.
2. Numerous anglers remained unaware of the NYS DOH health advisories for the Hudson River, particularly those who were fishing downstream of Catskill.
3. In 1996, a larger proportion of anglers in the Upper Hudson River were aware of the advisories and appear to be complying with the advice than in 1991-92. Compared to 1991-92, a greater proportion of 1996 anglers in the river between Troy and Catskill said they never ate fish they caught in the Hudson.
4. In 1996, no respondents in the Upper Hudson River said that they were eating, giving away or selling the fish they caught. However, about 18% had fish in their possession when interviewed and 11% had more than one fish. Most of the fish were largemouth and smallmouth bass or bluegill, species that are often eaten. In 1991-92, about 10% of anglers had said that they ate the fish they caught at least sometimes and almost 20% said that they gave fish away sometimes or frequently.
5. In 1996, 10% or fewer of anglers fishing between Troy and Catskill said that they ate, gave away or sold the fish they caught at least sometimes, and 8% actually had fish when interviewed.
6. In 1996, two-thirds of anglers fishing between Catskill and the Tappan Zee Bridge continued to report eating their fish at least sometimes and almost half (46%) of anglers gave fish away sometimes or frequently. More than half (57%) of anglers in this Area ate more fish than is advised by the NYS DOH advisories.
7. Overall, the difference in proportion of anglers who said they ate their catch was not significantly different, but significantly fewer anglers reported giving fish away in 1996 compared to 1991-92.
8. In 1996, half of the anglers who said they ate fish from the Hudson River reported eating two meals or less in the previous month and 5% of these anglers said they ate 12 meals or more in the previous month.

9. In 1996, 8% of anglers who did not eat the fish they caught gave them to others compared to 39% of these anglers in 1991-92.
10. Anglers who eat fish also share the fish they catch with others even though they may not say they gave the fish away. In 1996, anglers said they shared their catch with again as many other people, mostly family members. Most of these people were in the groups advised to eat no fish from the Hudson.
11. In both surveys, the fish that anglers kept were among the most contaminated species in each part of the river.
12. Both surveys suggest that most anglers became aware of the health advisories through the fishing regulations guide provided when they purchased a fishing license or through media coverage. Word-of-mouth was also important, and in 1996, signs placed along the Upper Hudson River, where most anglers had a license, appear to have contributed to improved awareness of the advisories.

Recommendations

Until the PCB levels in fish from the river decline, health advisories should continue to be issued and efforts to inform the public about the advisories should be continued. NYS DOH should continue to review data on levels of fish contamination and public health risks and revise the health advisories accordingly. The federal Agency for Toxic Substances and Disease Registry (ATSDR) and NYS DOH should continue to review all data to determine the need for additional actions at the site.

The findings of this study reinforce the recommendation in the NYS DOH 1994 Site Review and Update that "[a]dditional community health education efforts may be needed to inform those who still fish in the Hudson River of the health risk posed by PCB exposure to contaminated fish." Particular needs identified by this study and others are:

1. Additional educational efforts should be focused on the Lower Hudson River and contiguous waters with the same advisories (e.g., Harlem and East River, New York Harbor).
2. New techniques should be explored for finding those people who are eating Hudson River fish. Individuals who do not speak or read English may need to receive particular attention, especially in the New York City area.
3. Additional assessments are needed to more accurately estimate what Hudson River and New York Harbor anglers are eating, giving away or selling and to better understand how to convince anglers to follow the health advice.

Public Health Action Plan

The Public Health Action Plan (PHAP) for the Hudson River PCB site contains a description of the actions to be taken by the US EPA, ATSDR, and/or the New York State Department of Health (NYS DOH) at or near the site subsequent to the completion of this health consultation. The purpose of the PHAP is to ensure that this health consultation not only identifies public health hazards, but provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of the ATSDR/NYS DOH to follow-up on this plan to ensure that it is implemented. The following public health actions related to the health advisories for eating fish have been taken or will be implemented.

1999 Actions

1. NYS DOH evaluated 1997 data regarding PCBs and mercury in fish and modified the health advisories for some fish species between the Federal dam at Troy and Catskill.
2. NYS DOH provided the modified health advisories to NYS DEC for publication in the 1999-2000 fishing regulations guide.
3. Beginning October 1998, US EPA awarded the state a one-year grant to significantly increase education and outreach in the Hudson River estuary and New York Harbor area. NYS DOH and NYS DEC worked with local communities, state and federal agencies, non-government organizations and anglers to develop and implement effective ways to inform anglers and others who eat Hudson River fish about the health advisories and ways to reduce their health risks from eating contaminated fish. Six student intern rangers visited fishing access sites along the Hudson River from Fort Edward to New York City. They made almost 1150 site visits and spoke with almost 500 anglers, providing information about the advisories and learning about angler perceptions of them. Signs notifying anglers about the advisories were developed with assistance from three focus groups comprised of Hudson River anglers. The signs were distributed to owners or managers of marinas, parks, boat launching sites and other fishing access sites for posting along the river. Fish advisory brochures in English and Spanish were updated and distributed to bait and tackle shops, maritime museums and community groups. Radio public service announcements were prepared in Spanish and English and broadcast during prime time from mid-July through August 1999.

Actions Planned

1. NYS DOH will continue to evaluate new data regarding contaminants in fish and issue appropriate health advisories as needed.
2. NYS DOH will continue to work with NYS DEC to distribute updated versions of the NYS DOH health advisories to anglers who fish in the Hudson River, New York Harbor and other affected marine waters.
3. NYS DOH and NYS DEC will work with local communities, state and federal agencies, non-government organizations and anglers to implement effective ways to inform anglers and others who eat Hudson River fish about the health advisories and ways to reduce their health risks from eating contaminated fish.

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Preparers of Report and Acknowledgments

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Certification

The Health Consultation for the Exposure Investigation at the Hudson River PCB site was prepared by the New York State Department of Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the health consultation was initiated.

Technical Project Officer, SPS, SSAB, DHAC

The Division of Health Assessment and Consultation (DHAC), ATSDR, has reviewed this Health Consultation and concurs with its findings.

Acting Chief, SSAB, DHAC, ATSDR

Appendix A - List of Common and Scientific Names of Fish and Crabs Reported Being Caught

Common Name	Scientific Name	Family
Alewife	*Alosa pseudoharengus*	Clupeidae
American eel	*Anguilla rostrata*	Anguillidae
American shad	*Alosa sapidissima*	Clupeidae
Atlantic silverside	*Menidia menidia*	Atherinidae
Black crappie	*Pomoxis nigromaculatus*	Centrarchidae
Blueback herring	*Alosa aestivalis*	Clupeidae
Blue crab	*Callinectes sapidus*	Portunidae
Bluefish	*Pomatomus saltatrix*	Pomatomidae
Bluegill	*Lepomis macrochirus*	Centrarchidae
Brown bullhead	*Ameiurus nebulosus*	Ictaluridae
Brown trout	*Salmo trutta*	Salmonidae
Carp	*Cyprinus carpio*	Cyprinidae
Channel catfish	*Ictalurus punctatus*	Ictaluridae
Freshwater drum	*Aplodinotus grunniens*	Sciaenidae
Golden shiner	*Notemigonus crysoleucas*	Cyprinidae
Largemouth bass	*Micropterus salmoides*	Centrarchidae
Northern pike	*Esox lucius*	Esocidae)
Pumpkinseed	*Lepomis gibbosus*	Centrarchidae
Red hake	*Urophycis chuss*	Gadidae
Rock bass	*Ambloplites rupestris*	Centrarchidae
Smallmouth bass	*Micropterus dolomieu*	Centrarchidae
Striped bass	*Morone saxatilis*	Moronidae
White catfish	*Ameiurus catus*	Ictaluridae
White perch	*Morone americana*	Moronidae
White sucker	*Catostomus commersoni*	Catostomidae
Yellow perch	*Perca flavescens*	Percidae

More than 200 species of fish have been reported from the Hudson River. The species listed above were reported caught by anglers surveyed in 1996 and 1991-92.

Appendix B - Survey Instrument

QUESTIONNAIRE - HUDSON RIVER ANGLER SURVEY, 1996

Interviewer: _____

Date: _____/_____/_____ Day of Week: _____
month day year

Time Started: _____ Time Finished: _____

Site: _____

Gender of Person Being Interviewed: M F

1) I am taking a survey of fishing activity along the Hudson River. May I ask you some questions?

_____Yes
_____No - **(THANK PERSON AND TERMINATE INTERVIEW)**

2) Have you already been interviewed this year about recreational fishing?

_____Yes - **(END INTERVIEW)**
_____No

3) What types of fish are you trying to catch? **(LIST)**

4) What fishing or crabbing equipment are you using today?
_____ Hook and line
_____ Trap
_____ Net
_____ Other: _____

5) Have you caught anything here today, and if so, what?

Species Number caught Size **(MEASURE!)**

6) How many times have you fished or crabbed on the Hudson River in the last seven days (that is from _____ until today)?

7) How many times have you fished or crabbed on the Hudson River in the last month (that is from _____ until today)?

8) What is the main reason that you fish or crab?

9) For what other reasons do you fish or crab?
(LIST IN ORDER GIVEN)

10) We would like to know what you do with the fish or crabs that you catch. Do you do any of the following with your catch- often, sometimes, rarely, or never?
(READ FROM THE LIST BELOW, CHECK EACH APPLICABLE ANSWER)

	OFTEN	SOMETIMES	RARELY	NEVER
Eat:				
Toss back:				
Fertilizer:				
Bait:				
Throw in trash:				
Give away:				
Sell:				

If you give them away, what do the people you give them to do with them?

Eat: _____
Fertilizer: _____
Bait: _____
Other: _____ **(WHAT)**
Don't know: _____

If you sell them, what do the people you sell them to do with them?

Eat: _____
Fertilizer: _____
Bait: _____
Other: _____ **(WHAT)**
Don't know: _____

11) What do you think most people here do with their catch?
(RECORD IN ORDER GIVEN)

(IF RESPONDENT DOES NOT EAT CATCH, CONTINUE. IF THEY DO EAT CATCH, SKIP TO QUESTION 17)

12) Have you ever eaten fish or crabs from here in the past?

Yes _____ **(SKIP TO QUESTION 14)**
No _____

13) Why don't you eat your catch?

(SKIP TO QUESTION 20)

14) What kind of fish or crab did you eat?
(RECORD ALL ANSWERS GIVEN)

15) How often during the fishing season did you eat these fish or crabs? **(READ ALL CHOICES)**

4 or more times a week _____
2 or 3 times a week _____
once a week _____
2 to 3 times a month _____
once a month _____
less than once a month _____

16) Why did you stop eating these fish?

(SKIP TO QUESTION 20)

(RESUME QUESTIONS HERE IF RESPONDENT DOES EAT THEIR CATCH)

17) How many times in the last week (that is from _____ until today) did you eat fish or crabs from the Hudson River?

# of meals _____ **(EMPHASIZE NUMBER OF MEALS, NOT FISH)**

18) How many times in the last month (that is from _____ until today) did you eat fish or crabs from the Hudson River?

# of meals _____ **(EMPHASIZE NUMBER OF MEALS, NOT FISH)**

19) Who, besides yourself, eats the fish or crabs you catch from this area? **(FOR EACH PERSON LISTED, RECORD THE FOLLOWING)**

-Relation to respondent
-Age
-What kind of fish or crab they eat
-Whether they eat more, the same, or less than respondent

Relation	Age	Type of fish/crab	Amount (more, same, less)

(RESUME QUESTIONING HERE WITH ALL RESPONDENTS)

20) Are there any fish or crabs that people catch here, that are not safe to eat?

Yes _____
No _____ **(SKIP TO QUESTION 26)**
No opinion/Don't know _____ **(SKIP TO QUESTION 26)**

21) What fish or crabs that people catch here are not safe to eat?

22) Is it the whole fish or crab that is not safe to eat, or just parts of them?

23) Why are they not safe to eat?

24) What would happen if you ate them?

25) If you ate these fish or crabs and had no reaction within a day or two, would that mean the fish or crabs are safe to eat?

Yes _____
No _____
Don't know _____

26) How can you tell if the fish or crabs caught here, or their parts, are safe to eat?

27) Is there any way to make the fish or crabs that are caught here safer to eat after they have been caught?

No _____
If yes, what are they?

28) For the fish or crabs that you catch here, would you say that eating them: **(READ ALL CHOICES)**

Poses no risk at all _____
Poses a slight risk _____
Poses a serious risk _____

29) Would you say the water here is: **(READ ALL CHOICES)**

Not at all polluted _____
Slightly polluted _____
Quite polluted _____

30) **(IF RESPONDENT BELIEVES THAT THE WATER IS MORE POLLUTED THAN THE FISH- COMPARE ANSWERS TO 28 & 29):** If the water is slightly/quite polluted, why does eating the fish pose no risk/a slight risk?

31) Please answer yes, no, or don't know for each of the following questions:

	Yes	No	Don't Know
-Do you think the fish you catch here are contaminated?			
-Do you believe that eating fish caught at this site would pose a risk to your health?			
-Would you like more information about the potential risks from eating fish that are contaminated			
-Would you like more information about how you can control the risks from eating contaminated fish?			

32) Do you happen to know if there are any official health warnings about eating the fish that are caught here?

Yes _____
No _____ **(SKIP TO QUESTION 39)**
Don't know _____ **(SKIP TO QUESTION 39)**

33) What warnings are you aware of?

34) How did you originally learn about them?

35) Do you happen to know who makes these health advisories?
(READ ALL CHOICES)

Federal gov't _____
State gov't _____
County _____
Town _____
Other _____
Don't know _____

36) Do you agree, disagree, or have no opinion about the following statements?

	Agree	Disagree	No Opinion
The health advisories provide me with enough information to decide whether or not to eat certain fish.			
Many of the health advisories are not needed or are exaggerated.			

37) Since you learned about the health advisories, have you made any changes in either your fishing habits or in eating the fish you catch?

Yes _____
No _____ **(SKIP TO QUESTION 39)**

38) What changes have you made since you learned of the health advisories? Do you: **(READ, CHECK EACH THAT APPLIES)**

No longer eat the fish you catch	_____
Eat less of the fish you catch	_____
Eat more of the fish you catch	_____
Clean or cook the fish differently	_____
Fish in different locations	_____
Fish less often	_____
Fish more often	_____
Change the type of fish you try to catch	_____

Other:

39) What age group are you in? **(READ)**

under 10	_____	35-39	_____
10-14	_____	40-44	_____
15-19	_____	45-49	_____
20-24	_____	50-54	_____
25-29	_____	55-59	_____
30-34	_____	60+	_____

40) What is your race or ethnic background?

41) In what range is your yearly household income before taxes? **(READ CHOICES)**

<$10,000	_____
$10,000-$29,999	_____
$30,000-$49,999	_____
$50,000-$69,999	_____
$70,000-$89,999	_____
$90,000+	_____

42) How many people are there in your household?

43) Do you have a New York fishing license?

Yes	_____
No	_____

THANK YOU VERY MUCH FOR YOUR TIME!

Appendix C - Detailed Data Tables

Table C-1. Distribution of interviews by area and time. Number and percentage of interviews conducted in each area by month, day of week and time of day.

Interview Variables		1991-92 Survey				1996 Survey			
		Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
		n (%)	n (%)	n (%)	N (%)	n (%)	n (%)	n (%)	N (%)
Month	May	11 (19.6)	10 (20.0)	8 (13.3)	29 (17.5)	-	-	-	-
	June	7 (12.5)	12 (24.0)	24 (40.0)	43 (25.9)	5 (6.6)	14 (18.4)	17 (12.0)	36 (12.2)
	July	11 (19.6)	9 (18.0)	21 (35.0)	41 (24.7)	16 (21.1)	19 (25.0)	18 (12.7)	53 (18.0)
	August	5 (8.9)	6 (12.0)	4 (6.7)	15 (9.0)	25 (32.9)	5 (6.6)	43 (30.3)	73 (24.8)
	September	10 (17.9)	6 (12.0)	2 (3.3)	18 (10.8)	28 (36.8)	14 (18.4)	38 (26.8)	80 (27.2)
	October	8 (14.3)	7 (14.0)	1 (1.7)	16 (9.6)	2 (2.6)	24 (31.6)	26 (18.3)	52 (17.7)
	Nov.-Dec.	4 (7.1)	-	-	4 (2.4)	-	-	-	-
Day	Weekday	24 (42.9)	13 (26.0)	34 (56.7)	71 (42.8)	47 (61.8)	33 (43.4)	69 (48.6)	149 (50.7)
	Weekend	32 (57.1)	37 (74.0)	26 (43.3)	95 (57.2)	29 (38.2)	43 (56.6)	73 (51.4)	145 (49.3)
Hour	Morning	21 (37.5)	14 (28.0)	15 (25.0)	50 (30.1)	15 (19.7)	9 (11.8)	22 (15.5)	46 (15.6)
	Afternoon	17 (30.4)	15 (30.0)	22 (36.7)	54 (32.5)	36 (47.4)	23 (30.3)	90 (63.4)	149 (50.7)
	Evening	18 (32.1)	21 (42.0)	16 (26.7)	55 (33.1)	25 (32.9)	44 (57.9)	30 (21.1)	99 (33.7)
	Unknown	-	-	7 (11.7)	7 (4.2)	-	-	-	-
TOTAL		56 (33.7)	50 (30.1)	60 (36.1)	166 (100)	76 (25.9)	76 (25.9)	142 (48.3)	294 (100)

Table C-2. Distribution of interviews among the survey areas. Number and percentage of completed surveys in each area.

	1991-92 Survey N (%)	1996 Survey N (%)
Area 1	**56 (33.7)[1]**	**76 (25.9)**
Thompson Island Pool (RM 187-196)	7 (12.5)	6 (7.9)
Stillwater Pool (RM 167-182)	-	22 (28.9)
Mechanicville-Stillwater (RM 154-166)	46 (82.1)	48 (63.2)
Area 2	**50 (30.1)**	**76 (25.9)**
Albany-Troy (RM 140-153)	45 (90.0)	43 (56.6)
Stuyvesant-Coxsackie (RM 115-139)	5 (10.0)	21 (27.6)
Catskill (RM 110-114)	-	12 (15.8)
Area 3	**60 (36.1)**	**142 (48.3)**
Kingston-Esopus (RM 90-109)	15 (25.0)	6 (4.2)
Poughkeepsie (RM 70-89)	17 (28.3)	18 (12.7)
Hudson Highlands (RM 43-69)	26 (43.3)	54 (38.0)
Haverstraw Bay (RM 34-42)	-	58 (40.8)
Tappan Zee Bridge-Croton Pt (RM 24-33)	2 (3.3)	6 (4.2)
Total	**166[1] (100)**	**294 (100)**

RM is the river mile index. The value is the number of miles along the river centerline upstream from the Battery (southern tip of Manhattan).

[1] In the 1991-92 survey, the location of 3 responses could not be accurately determined within Area 1. Thus, the totals are larger than the sum of locations that include Area 1 in 1991-92.

Table C-3. Demographic characteristics of anglers responding to 1991-92 and 1996 surveys.

Demographic Variables		1991-92 Survey				1996 Survey			
		Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
		n (%)	n (%)	n (%)	N (%)	n (%)	n (%)	n (%)	N (%)
Age	<24 yrs	15 (26.8)	11 (22.0)	9 (15.0)	35 (21.1)	19 (25.0)	8 (10.5)	12 (8.5)	39 (13.3)
	25-34 yrs	14 (25.0)	11 (22.0)	19 (31.7)	44 (26.5)	19 (25.0)	26 (34.2)	31 (21.8)	76 (25.9)
	35-44 yrs	10 (17.9)	13 (26.0)	16 (26.7)	39 (23.5)	24 (31.6)	22 (28.9)	50 (35.2)	96 (32.7)
	45-59 yrs	13 (23.2)	9 (18.0)	10 (16.7)	32 (19.3)	5 (6.6)	13 (17.1)	29 (20.4)	47 (16.0)
	60 + yrs	4 (7.1)	6 (12.0)	6 (10.0)	16 (9.6)	9 (11.8)	7 (9.2)	19 (13.4)	35 (11.9)
	Refusal	-	-	-	-	-	-	1 (0.7)	1 (0.3)
Gender	Male	54 (96.4)	43 (86.0)	58 (96.7)	155 (93.4)	65 (85.5)	66 (86.8)	126 (88.7)	257 (87.4)
	Female	2 (3.6)	7 (14.0)	2 (3.3)	11 (6.6)	11 (14.5)	10 (13.2)	16 (11.3)	37 (12.6)
Race/Ethnicity	White	53 (94.6)	42 (84.0)	37 (61.7)	132 (79.5)	75 (98.7)	60 (78.9)	69 (48.6)	204 (69.4)
	Black	2 (3.6)	6 (12.0)	16 (26.7)	24 (14.5)	1 (1.3)	6 (7.9)	29 (20.4)	36 (12.2)
	Hispanic	-	1 (2.0)	6 (10.0)	7 (4.2)	-	3 (3.9)	36 (25.4)	39 (13.3)
	Others	1 (1.8)	1 (2.0)	1 (1.7)	3 (1.8)	-	7 (9.2)	8 (5.6)	15 (5.1)
Income	<10 K	9 (16.1)	8 (16.0)	11 (18.3)	28 (16.9)	11 (14.5)	13 (17.1)	20 (14.1)	44 (15.0)
	$10-29 K	18 (32.1)	19 (38.0)	21 (35.0)	58 (34.9)	33 (43.4)	20 (26.3)	28 (19.7)	81 (27.6)
	$30-49 K	15 (26.8)	12 (24.0)	15 (25.0)	42 (25.3)	15 (19.7)	27 (35.5)	22 (15.5)	64 (21.8)
	50+ K	5 (8.9)	5 (10.0)	7 (11.7)	17 (10.2)	2 (2.6)	12 (15.8)	14 (9.9)	28 (9.5)
	Refusal	9 (16.1)	6 (12.0)	6 (10.0)	21 (12.7)	15 (19.7)	4 (5.3)	58 (40.8)	77 (26.2)
Household	1 person	11 (19.6)	7 (14.0)	9 (15.0)	27 (16.3)	8 (10.5)	10 (13.2)	16 (11.3)	34 (11.6)
	2 people	17 (30.4)	11 (22.0)	13 (21.7)	41 (24.7)	21 (27.6)	27 (35.5)	48 (33.8)	96 (32.7)
	3 people	15 (26.8)	16 (32.0)	12 (20.0)	43 (25.9)	14 (18.4)	23 (30.3)	17 (12.0)	54 (18.4)
	4 people	9 (16.1)	5 (10.0)	11 (18.3)	25 (15.1)	20 (26.3)	10 (13.2)	27 (19.0)	57 (19.4)
	5 + people	4 (7.1)	11 (22.0)	15 (25.0)	30 (18.1)	13 (17.1)	6 (7.9)	34 (23.9)	53 (18.0)
License	Yes	-	-	-	-	65 (85.5)	58 (76.3)	46 (32.4)	169 (57.5)
	No	-	-	-	-	11 (14.5)	18 (23.7)	96 (67.6)	125 (42.5)
TOTAL		56 (33.7)	50 (30.1)	60 (36.1)	166 (100)	76 (25.9)	76 (25.9)	142 (48.3)	294 (100)

Table C-4. Angler awareness of health advisories. Responses to Question 32.

Demographic Variables		1991-92 Survey				1996 Survey			
		Yes	No	Don't know	Total	Yes	No	Don't know	Total
		n (%)	n (%)	n (%)	N (%)	n (%)	n (%)	n (%)	N (%)
Age	<24 yrs	6 (17.1)	14 (40.0)	15 (42.9)	35 (21.1)	16 (41.0)	9 (23.1)	14 (35.9)	39 (13.3)
	25-34 yrs	23 (52.3)	12 (27.3)	9 (20.5)	44 (26.5)	38 (50.0)	14 (18.4)	24 (31.6)	76 (25.9)
	35-44 yrs	28 (71.8)	5 (12.8)	6 (15.4)	39 (23.5)	54 (56.3)	11 (11.5)	31 (32.3)	96 (32.7)
	45-59 yrs	19 (59.4)	7 (21.9)	6 (18.8)	32 (19.3)	19 (40.4)	4 (8.5)	24 (51.1)	47 (16.0)
	60 + yrs	9 (56.3)	5 (31.3)	2 (12.5)	16 (9.6)	18 (51.4)	5 (14.3)	12 (34.3)	35 (11.9)
	Refusal	-	-	-	-	-	-	1 (100)	1 (0.3)
Gender	Male	83 (53.5)	36 (23.2)	36 (23.2)	155 (93.4)	135 (52.5)	31 (12.1)	91 (35.4)	257 (87.4)
	Female	2 (18.2)	7 (63.6)	2 (18.2)	11 (6.6)	10 (27.0)	12 (32.4)	15 (40.5)	37 (12.6)
Race/Ethnicity	White	66 (50.0)	37 (28.0)	29 (22.0)	132 (79.5)	129 (63.2)	31 (15.2)	44 (21.6)	204 (69.4)
	Black	14 (58.3)	3 (12.5)	7 (29.2)	24 (14.5)	8 (22.2)	6 (16.7)	22 (61.1)	36 (12.2)
	Hispanic	3 (42.9)	2 (28.6)	2 (28.6)	7 (4.2)	5 (12.8)	1 (2.6)	33 (84.6)	39 (13.3)
	Others	2 (66.7)	1 (33.3)	-	3 (1.8)	3 (20.0)	5 (33.3)	7 (46.7)	15 (5.1)
Income	<10 K	6 (21.4)	14 (50.0)	8 (28.6)	28 (16.9)	15 (34.1)	10 (22.7)	19 (43.2)	44 (15.0)
	$10-29 K	31 (53.4)	18 (31.0)	9 (15.5)	58 (34.9)	40 (49.4)	17 (21.0)	24 (29.6)	81 (27.6)
	$30-49 K	30 (71.4)	4 (9.5)	8 (19.0)	42 (25.3)	48 (75.0)	4 (6.3)	12 (18.8)	64 (21.8)
	50+ K	10 (58.8)	2 (11.8)	5 (29.4)	17 (10.2)	19 (67.9)	4 (14.3)	5 (17.9)	28 (9.5)
	Refusal	8 (38.1)	5 (23.8)	8 (38.1)	21 (12.7)	23 (29.9)	8 (10.4)	46 (59.7)	77 (26.2)
Area	1	31 (55.4)	13 (23.2)	12 (21.4)	56 (33.7)	57 (75.0)	15 (19.7)	4 (5.3)	76 (25.9)
	2	21 (42.0)	14 (28.0)	15 (30.0)	50 (30.1)	44 (57.9)	23 (30.3)	9 (11.8)	76 (25.9)
	3	33 (55.0)	16 (26.7)	11 (18.3)	60 (36.1)	44 (31.0)	5 (3.5)	93 (65.5)	142 (48.3)
License	Yes	-	-	-	-	123 (72.8)	24 (14.2)	22 (13.0)	169 (57.5)
	No	-	-	-	-	22 (17.6)	19 (15.2)	84 (67.2)	125 (42.5)
TOTAL		85 (51.2)	43 (25.9)	38 (22.9)	166 (100)	145 (49.3)	43 (14.6)	106 (36.1)	294 (100)

Table C-5. How anglers became aware of health advisories and their general opinion of them.

	1991-92 Survey				1996 Survey			
	Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
How did you learn about advisories?								
Pamphlet with fishing license	12 (38.7)	13 (61.9)	6 (18.2)	31 (36.5)	23 (40.4)	22 (50.0)	19 (43.2)	64 (44.1)
Media	8 (25.8)	5 (23.8)	17 (51.5)	30 (35.3)	6 (10.5)	9 (20.5)	11 (25.0)	26 (17.9)
Posters (signs)	2 (6.5)	-	-	2 (2.4)	13 (22.8)	7 (15.9)	-	20 (13.8)
Word-of-mouth	9 (29.0)	2 (9.5)	9 (27.3)	20 (23.5)	8 (14.0)	2 (4.5)	12 (27.3)	22 (15.2)
Handouts	-	-	-	-	-	2 (4.5)	-	2 (1.4)
Environmental organizations	-	-	-	-	1 (1.8)	-	-	1 (0.7)
NYS DEC	-	-	-	-	1 (1.8)	1 (2.3)	2 (4.5)	4 (2.8)
Don't know or no response	-	1 (4.8)	1 (3.0)	2 (2.4)	5 (8.8)	1 (2.3)	-	6 (4.1)
Do you agree that health advisories provide you with enough information?								
Yes	16 (51.6)	12 (57.1)	11 (33.3)	39 (45.9)	33 (57.9)	33 (75.0)	30 (68.2)	96 (66.2)
No	14 (45.2)	7 (33.3)	14 (42.4)	35 (41.2)	16 (28.1)	8 (18.2)	-	24 (16.6)
No opinion	1 (3.2)	2 (9.5)	8 (24.2)	11 (12.9)	8 (14.0)	3 (6.8)	14 (31.8)	25 (17.2)
Do you agree that health advisories are not needed or are exaggerated?								
Yes	6 (19.4)	5 (23.8)	14 (42.4)	25 (29.4)	2 (3.5)	8 (18.2)	1 (2.3)	11 (7.6)
No	24 (77.4)	13 (61.9)	14 (42.4)	51 (60.0)	40 (70.2)	30 (68.2)	12 (27.3)	82 (56.6)
No opinion	1 (3.2)	3 (14.3)	5 (15.2)	9 (10.6)	15 (26.3)	6 (13.6)	31 (70.5)	52 (35.9)
Have you made any changes since learning about the advisories?								
Yes	12 (38.7)	11 (52.4)	12 (36.4)	35 (41.2)	23 (40.4)	4 (9.1)	18 (40.9)	45 (31.0)
No	19 (61.3)	10 (47.6)	21 (63.6)	50 (58.8)	34 (59.6)	40 (90.9)	26 (59.1)	100 (69.0)
TOTAL	31 (36.5)	21 (24.7)	33 (38.8)	85 (100)	57 (39.3)	44 (30.3)	44 (30.3)	145 (100)

Table C-6. Angler understanding of the advisories. Combined responses to questions 20, 28, 29, 31a and 31b.

Demographic Variables		1991-92 Survey				1996 Survey			
		Yes	No	Don't know	Total	Yes	No	Don't know	Total
		n (%)	n (%)	n (%)	N (%)	n (%)	n (%)	n (%)	N (%)
Age	<24 yrs	12 (34.3)	-	23 (65.7)	35 (21.1)	24 (61.5)	-	15 (38.5)	39 (13.3)
	25-34 yrs	20 (45.5)	-	24 (54.5)	44 (26.5)	35 (46.1)	2 (2.6)	39 (51.3)	76 (25.9)
	35-44 yrs	20 (51.3)	2 (5.1)	17 (43.6)	39 (23.5)	42 (43.8)	2 (2.1)	52 (54.2)	96 (32.7)
	45-59 yrs	11 (34.4)	2 (6.3)	19 (59.4)	32 (19.3)	12 (25.5)	5 (10.6)	30 (63.8)	47 (16.0)
	60 + yrs	4 (25.0)	-	12 (75.0)	16 (9.6)	11 (31.4)	3 (8.6)	21 (60.0)	35 (11.9)
	Refusal	-	-	-	-	-	-	1 (100)	1 (0.3)
Gender	Male	61 (39.4)	2 (1.3)	92 (59.4)	155 (93.4)	112 (43.6)	7 (2.7)	138 (53.7)	257 (87.4)
	Female	6 (54.5)	2 (18.2)	3 (27.3)	11 (6.6)	12 (32.4)	5 (13.5)	20 (54.1)	37 (12.6)
Race/Ethnicity	White	59 (44.7)	3 (2.3)	70 (53.0)	132 (79.5)	104 (51.0)	4 (2.0)	96 (47.1)	204 (69.4)
	Black	6 (25.0)	1 (4.2)	17 (70.8)	24 (14.5)	10 (27.8)	3 (8.3)	23 (63.9)	36 (12.2)
	Hispanic	1 (14.3)	-	6 (85.7)	7 (4.2)	4 (10.3)	4 (10.3)	31 (79.5)	39 (13.3)
	Others	1 (33.3)	-	2 (66.7)	3 (1.8)	6 (40.0)	1 (6.7)	8 (53.3)	15 (5.1)
Income	<10 K	10 (35.7)	2 (7.1)	16 (57.1)	28 (16.9)	20 (45.5)	2 (4.5)	22 (50.0)	44 (15.0)
	$10-29 K	21 (36.2)	2 (3.4)	35 (60.3)	58 (34.9)	45 (55.6)	3 (3.7)	33 (40.7)	81 (27.6)
	$30-49 K	19 (45.2)	-	23 (54.8)	42 (25.3)	30 (46.9)	1 (1.6)	33 (51.6)	64 (21.8)
	50+ K	8 (47.1)	-	9 (52.9)	17 (10.2)	10 (35.7)	-	18 (64.3)	28 (9.5)
	Refusal	9 (42.9)	-	12 (57.1)	21 (12.7)	19 (24.7)	6 (7.8)	52 (67.5)	77 (26.2)
Area	1	29 (51.8)	1 (1.8)	26 (46.4)	56 (33.7)	58 (76.3)	-	18 (23.7)	76 (25.9)
	2	18 (36.0)	2 (4.0)	30 (60.0)	50 (30.1)	35 (46.1)	-	41 (53.9)	76 (25.9)
	3	20 (33.3)	1 (1.7)	39 (65.0)	60 (36.1)	31 (21.8)	12 (8.5)	99 (69.7)	142 (48.3)
Warning	Yes	42 (49.4)	-	43 (50.6)	85 (51.2)	95 (65.5)	-	50 (34.5)	145 (49.3)
	No/Don't know	25 (30.9)	4 (4.9)	52 (64.2)	81 (48.8)	29 (19.5)	12 (8.1)	108 (72.5)	149 (50.7)
License	Yes	-	-	-	-	91 (53.8)	2 (1.2)	76 (45.0)	169 (57.5)
	No	-	-	-	-	33 (26.4)	10 (8.0)	82 (65.6)	125 (42.5)
TOTAL		67 (40.4)	4 (2.4)	95 (57.2)	166 (100)	124 (42.2)	12 (4.1)	158 (53.7)	294 (100)

Yes is counted only if all 5 questions were answered "yes", and *no* is counted only if all 5 questions were answered "no".

Table C-7. Anglers who had fish when interviewed in 1996.

	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	N (%)
Caught fish in 1991-92				
Yes	37 (66.1)	26 (52.0)	34 (56.7)	97 (58.4)
No	19 (33.9)	24 (48.0)	26 (43.3)	69 (41.6)
TOTAL	56 (33.7)	50 (30.1)	60 (36.1)	166 (100)
Caught fish in 1996				
Yes	24 (31.6)	32 (42.1)	99 (69.7)	155 (52.7)
No	52 (68.4)	44 (57.9)	43 (30.3)	139 (47.3)
Kept fish in 1996				
Yes	14 (18.4)	6 (7.9)	67 (47.2)	87 (29.6)
No	62 (81.6)	70 (92.1)	75 (52.8)	207 (70.4)
TOTAL	76 (25.9)	76 (25.9)	142 (48.3)	294 (100)

Table C-8. Numbers of fish reported as caught by anglers.

	Area 1		Area 2		Area 3		Total	
	Ang (%)[1]	n (%)[2]	Ang (%)[1]	n (%)[2]	Ang (%)[1]	n (%)[2]	Ang (%)[1]	n (%)[2]
1991-92 Survey								
White perch	9 (24.3)	45 (19.7)	4 (15.4)	63 (30.9)	8 (23.5)	28 (12.2)	21 (21.6)	136 (20.5)
Blue crab	-	-	-	-	12 (35.3)	126 (54.8)	12 (12.4)	126 (19.0)
Blueback herring	2 (5.4)	43 (18.9)	4 (15.4)	37 (18.1)	-	-	6 (6.2)	80 (12.1)
Striped bass	4 (10.8)	15 (6.6)	7 (26.9)	26 (12.7)	10 (29.4)	27 (11.7)	21 (21.6)	68 (10.3)
White catfish	2 (5.4)	2 (0.9)	4 (15.4)	38 (18.6)	9 (26.5)	25 (10.9)	15 (15.5)	65 (9.8)
Black crappie	2 (5.4)	32 (14.0)	-	-	-	-	2 (2.1)	32 (4.8)
Brown bullhead	7 (18.9)	22 (9.6)	2 (7.7)	5 (2.5)	-	-	9 (9.3)	27 (4.1)
Smallmouth bass	10 (27.0)	16 (7.0)	3 (11.5)	4 (2.0)	-	-	13 (13.4)	20 (3.0)
American eel	2 (5.4)	3 (1.3)	3 (11.5)	5 (2.5)	6 (17.6)	10 (4.3)	11 (11.3)	18 (2.7)
Yellow perch	6 (16.2)	16 (7.0)	2 (7.7)	2 (1.0)	-	-	8 (8.2)	18 (2.7)
American shad	1 (2.7)	1 (0.4)	3 (11.5)	17 (8.3)	-	-	4 (4.1)	18 (2.7)
Carp	1 (2.7)	1 (0.4)	-	-	3 (8.8)	11 (4.8)	4 (4.1)	12 (1.8)
Rock bass	2 (5.4)	12 (5.3)	-	-	-	-	2 (2.1)	12 (1.8)
Largemouth bass	5 (13.5)	5 (2.2)	2 (7.7)	5 (2.5)	-	-	7 (7.2)	10 (1.5)
Bluegill	2 (5.4)	3 (1.3)	1 (3.8)	1 (0.5)	1 (2.9)	2 (0.9)	4 (4.1)	6 (0.9)
Pumpkinseed	4 (10.8)	6 (2.6)	-	-	-	-	4 (4.1)	6 (0.9)
Northern pike	4 (10.8)	6 (2.6)	-	-	-	-	4 (4.1)	6 (0.9)
Alewife	-	-	1 (3.8)	1 (0.5)	1 (2.9)	1 (0.4)	2 (2.1)	2 (0.3)
Total	37 (38.1)	228 (34.4)	26 (26.8)	204 (30.8)	34 (35.1)	230 (34.7)	97 (100)	662 (100)
1996 Survey								
White perch	-	-	9 (20.0)	27 (22.3)	4 (3.4)	257 (35.0)	13 (6.8)	284 (31.4)
Blue crab	-	-	-	-	22 (19.0)	146 (19.9)	22 (11.5)	146 (16.1)
Striped bass	1 (3.3)	1 (2.0)	3 (6.7)	3 (2.5)	20 (17.2)	105 (14.3)	24 (12.6)	109 (12.0)
Largemouth bass	11 (36.7)	26 (52.0)	4 (8.9)	38 (31.4)	6 (5.2)	10 (1.4)	21 (11.0)	74 (8.2)
Atlantic silverside	-	-	-	-	2 (1.7)	67 (9.1)	2 (1.0)	67 (7.4)
Bluefish	-	-	-	-	12 (10.3)	62 (8.4)	12 (6.3)	62 (6.9)
American eel	-	-	7 (15.6)	12 (9.9)	15 (12.9)	30 (4.1)	22 (11.5)	42 (4.6)
White catfish	-	-	6 (13.3)	14 (11.6)	12 (10.3)	21 (2.9)	18 (9.4)	35 (3.9)
Smallmouth bass	10 (33.3)	14 (28.0)	4 (8.9)	7 (5.8)	4 (3.4)	4 (0.5)	18 (9.4)	25 (2.8)
Bluegill	5 (16.7)	6 (12.0)	5 (11.1)	5 (4.1)	5 (4.3)	13 (1.8)	15 (7.9)	24 (2.7)
Brown bullhead	-	-	3 (6.7)	7 (5.8)	1 (0.9)	1 (0.1)	4 (2.1)	8 (0.9)
Carp	-	-		-	4 (3.4)	6 (0.8)	4 (2.1)	6 (0.7)
Golden shiner	1 (3.3)	1 (2.0)	1 (2.2)	2 (1.7)	1 (0.9)	2 (0.3)	3 (1.6)	5 (0.6)
White sucker	1 (3.3)	1 (2.0)	-	-	3 (2.6)	3 (0.4)	4 (2.1)	4 (0.4)
Red hake	-	-	-	-	1 (0.9)	2 (0.3)	1 (0.5)	2 (0.2)
Freshwater drum	-	-	-	-	1 (0.9)	2 (0.3)	1 (0.5)	2 (0.2)
Yellow perch	-	-	-	-	1 (0.9)	1 (0.1)	1 (0.5)	1 (0.1)
Pumpkinseed	-	-	-	-	1 (0.9)	1 (0.1)	1 (0.5)	1 (0.1)
Brown trout	-	-	-	-	1 (0.9)	1 (0.1)	1 (0.5)	1 (0.1)
Rock bass	1 (3.3)	1 (2.0)	1 (2.2)	1 (0.8)	-	-	2 (1.0)	2 (0.2)
Northern pike	-	-	2 (4.4)	5 (4.1)	-	-	2 (1.0)	5 (0.6)
Total	30 (15.7)	50 (5.5)	45 (23.6)	121 (13.4)	116 (60.7)	734 (81.1)	191 (100)	905 (100)

[1] *Ang* is the number of anglers who had at least one of the species listed. The total is the number of anglers who caught fish (from Table C-7).
[2] *n* is the number of fish reported caught for that species.

Table C-9. Number of anglers who kept fish and number and weight of fish kept by anglers in 1996 survey.

Fish Species	Area 1			Area 2			Area 3			Total		
	Ang[1] (%)	n[2] (%)	Weight[3] (%)	Ang[1] (%)	n[2] (%)	Weight[3] (%)	Ang[1] (%)	n[2] (%)	Weight[3] (%)	Ang[1] (%)	n[2] (%)	Weight[3] (%)
White perch				2 (33.3)	7 (33.3)	211 (3.8)	37 (55.2)	227 (44.2)	15498 (25.6)	39 (44.8)	234 (41.9)	15709 (22.0)
White catfish				2 (33.3)	8 (38.1)	786 (14.0)	11 (16.4)	20 (3.9)	10512 (17.3)	13 (14.9)	28 (5.0)	11298 (15.8)
Striped bass	1 (7.1)	1 (4.2)	37 (0.7)				16 (23.9)	84 (16.4)	10068 (16.6)	17 (19.5)	85 (15.2)	10105 (14.2)
Carp							4 (6.0)	6 (1.2)	8209 (13.5)	4 (4.6)	6 (1.1)	8209 (11.5)
Largemouth bass	3 (21.4)	3 (12.5)	1605 (31.2)	1 (16.7)	2 (9.5)	2332 (41.5)	1 (1.5)	1 (0.2)	1348 (2.2)	5 (5.7)	6 (1.1)	5285 (7.4)
Smallmouth bass	7 (50.0)	11 (45.8)	2211 (42.9)	2 (33.3)	3 (14.3)	2242 (39.9)	1 (1.5)	1 (0.2)	648 (1.1)	10 (11.5)	15 (2.7)	5101 (7.1)
Bluefish							11 (16.4)	60 (11.7)	4954 (8.2)	11 (12.6)	60 (10.8)	4954 (6.9)
American eel				1 (16.7)	1 (4.8)	51 (0.9)	13 (19.4)	27 (5.3)	4139 (6.8)	14 (16.1)	28 (5.0)	4190 (5.9)
White sucker	1 (7.1)	1 (4.2)	192 (3.7)				2 (3.0)	2 (0.4)	2256 (3.7)	3 (3.4)	3 (0.5)	2448 (3.4)
Bluegill	5 (35.7)	6 (25.0)	818 (15.9)				4 (6.0)	11 (2.1)	1162 (1.9)	9 (10.3)	17 (3.0)	1980 (2.8)
Rock bass	1 (7.1)	1 (4.2)	87 (1.7)							1 (1.1)	1 (0.2)	87 (0.1)
Freshwater drum							1 (1.5)	2 (0.4)	634 (1.0)	1 (1.1)	2 (0.4)	634 (0.9)
Brown bullhead							1 (1.5)	1 (0.2)	323 (0.5)	1 (1.1)	1 (0.2)	323 (0.5)
Pumpkinseed							1 (1.5)	1 (0.2)	268 (0.4)	1 (1.1)	1 (0.2)	268 (0.4)
Brown trout							1 (1.5)	1 (0.2)	244 (0.4)	1 (1.1)	1 (0.2)	244 (0.3)
Atlantic silverside							2 (3.0)	67 (13.1)	213 (0.4)	2 (2.3)	67 (12.0)	213 (0.3)
Golden shiner	1 (7.1)	1 (4.2)	200 (3.9)				1 (1.5)	2 (0.4)	140 (0.2)	2 (2.3)	3 (0.5)	340 (0.5)
TOTAL	14 (16.1)	24 (4.3)	5150 (7.2)	6 (6.9)	21 (3.8)	5622 (7.9)	67 (77.0)	513 (91.9)	60616 (84.9)	87 (100)	558 (100)	71388 (100)

[1] *Ang* is number of anglers who had at least one of the species listed. The total is the number of anglers who kept fish (from Table C-7).

[2] *n* is number of fish reported caught for that species.

[3] *Weight* is sum of weights (in grams wet-weight) estimated from regressions on length as noted in Table C-10.

N.B. In Area 2, one angler had kept 5 white catfish, but the lengths were not recorded and weights could not be estimated.

Table C-10. List of regression equations used for calculating weights of Hudson River fishes.

Species	Equation	Source
American eel	ln wt(g) = -14.631 + 3.253 ln TL(mm)	Helfman *et al.* 1984- Georgia estuary
Atlantic silverside	log wt(g) = -4.2538 + 2.5234 log TL(mm)	Wilk *et al.* 1978 - New York Bight
Bluefish	log wt(g) = -4.9533 + 3.0359 log TL(mm)	Wilk *et al.* 1978 - New York Bight
Bluegill	log wt(g) = -5.515 + 3.371 log TL(mm)	Carlander 1977 - Pennsylvania
Brown bullhead	log wt(g) = -5.061 + 3.065 log TL(mm)	Carlander 1969 - p. 535
Brown trout		Use table, p. 213 in Carlander 1969
Carp	log wt(kg) = 1.86 + 0.027 TL(cm)	Mongomery & Schmidt 1993 - Hudson River
Freshwater drum		Use table, p. 815 in Scott & Crossman 1972
Golden shiner		Use table, p 409 in Carlander 1969
Largemouth bass	log wt(g) = -5.11 + 3.117 log TL(mm)	DEC - Hudson River
Pumpkinseed	log wt(g) = -5.213 + 3.262 log TL(mm)	Carlander 1977 - Pennsylvania
Rock bass		Use table, p. 21-22 in Carlander 1977
Smallmouth bass	log wt(g) = -5.53 + 3.248 log TL(mm)	DEC - Hudson River
Striped bass	log wt(g) = -5.019 + 3.028 log TL(mm)	Hoff *et al.* 1988 - Hudson River
White catfish	log wt(g) = 5.46 + 3.24 log TL(mm)	Hughes & Carlson 1986 - Hudson River
White perch	log wt(g) = -4.9513 + 3.0249 log TL(mm)	Klauda *et al.* 1988 - Hudson River
White sucker	log wt(g) = -3.885 + 2.5914 log TL(mm)	Carlander 1969 - upstate New York

Table C-11. Reasons anglers gave for fishing.

Primary reason

	Area 1 n (%)	Area 2 n (%)	Area 3 n (%)	Total n (%)
1991-92 Survey				
Recreation	54 (96.4)	48 (96.0)	49 (81.7)	151 (91.0)
Food	1 (1.8)	1 (2.0)	9 (15.0)	11 (6.6)
Other	1 (1.8)	1 (2.0)	1 (1.7)	3 (1.8)
No response	-	-	1 (1.7)	1 (0.6)
TOTAL	56 (33.7)	50 (30.1)	60 (36.1)	166 (100)
1996 Survey				
Recreation	74 (97.4)	72 (94.7)	120 (84.5)	266 (90.5)
Food	-	-	18 (12.7)	18 (6.1)
Other	2 (2.6)	4 (5.3)	4 (2.8)	10 (13.2)
TOTAL	76 (25.9)	76 (25.9)	142 (48.3)	294 (100)

Any reason

	Area 1 n (%)	Area 2 n (%)	Area 3 n (%)	Total n (%)
1991-92 Survey				
Recreation	56 (100)	49 (98.0)	57 (95.0)	162 (97.6)
Food	4 (7.1)	3 (6.0)	21 (35.0)	28 (16.9)
Other	2 (3.6)	2 (4.0)	3 (5.0)	7 (4.2)
TOTAL	56 (33.7)	50 (30.1)	60 (36.1)	166 (100)
1996 Survey				
Recreation	75 (98.7)	73 (96.1)	129 (90.8)	277 (94.2)
Food	-	-	67 (47.2)	67 (22.8)
Other	5 (6.6)	6 (7.9)	9 (6.3)	20 (6.8)
TOTAL	76 (25.9)	76 (25.9)	142 (48.3)	294 (100)

Recreation included: recreation, socialize, be alone, enjoy outdoors.
Food included: food, get fish for friends.
Other included: get bait, reward for tags, other.

Table C-12. Use of fish caught by anglers.

Use	1991-1992 Survey				1996 Survey			
	Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Tossback								
Often	51 (91.1)	43 (86.0)	34 (57.6)	128 (77.6)	76 (100)	73 (96.1)	54 (38.0)	203 (69.0)
Sometimes	4 (7.1)	7 (14.0)	26 (44.1)	37 (22.4)	-	3 (3.9)	66 (46.5)	69 (23.5)
Rarely	-	-	-	-	-	-	16 (11.3)	16 (5.4)
Never	1 (1.8)	-	-	1 (0.6)	-	-	6 (4.2)	6 (2.0)
Eat								
Often	3 (5.4)	5 (10.0)	19 (32.2)	27 (16.4)	-	2 (2.6)	61 (43.0)	63 (21.4)
Sometimes	3 (5.4)	6 (12.0)	14 (23.7)	23 (13.9)	-	5 (6.6)	38 (26.8)	43 (14.6)
Rarely	5 (8.9)	3 (6.0)	8 (13.6)	16 (9.7)	-	8 (10.5)	19 (13.4)	27 (9.2)
Never	45 (80.4)	36 (72.0)	19 (32.2)	100 (60.6)	76 (100)	61 (80.3)	24 (16.9)	161 (54.8)
Give away								
Often	4 (7.1)	3 (6.0)	7 (11.9)	14 (8.5)	-	-	7 (4.9)	7 (2.4)
Sometimes	7 (12.5)	11 (22.0)	35 (59.3)	53 (32.1)	-	4 (5.3)	58 (40.8)	62 (21.1)
Rarely	3 (5.4)	4 (8.0)	8 (13.6)	15 (9.1)	-	6 (7.9)	28 (19.7)	34 (11.6)
Never	42 (75.0)	32 (64.0)	10 (16.9)	84 (50.9)	76 (100)	66 (86.8)	49 (34.5)	191 (65.0)
Sell								
Often	-	-	1 (1.7)	1 (0.6)	-	1 (1.3)	1 (0.7)	2 (0.7)
Sometimes	-	-	3 (5.1)	3 (1.8)	-	-	-	-
Rarely	-	-	3 (5.1)	3 (1.8)	-	1 (1.3)	3 (2.1)	4 (1.4)
Never	50 (89.3)	50 (100)	47 (79.7)	147 (89.1)	76 (100)	74 (97.4)	134 (94.4)	284 (96.6)
No Response	6 (10.7)	-	6 (10.2)	12 (7.3)	-	-	4 (2.8)	4 (1.4)
Fertilizer								
Often	-	-	1 (1.7)	1 (0.6)	-	-	-	-
Sometimes	2 (3.6)	1 (2.0)	2 (3.4)	5 (3.0)	-	2 (2.6)	1 (0.7)	3 (1.0)
Rarely	-	-	2 (3.4)	2 (1.2)	-	1 (1.3)	1 (0.7)	2 (0.7)
Never	54 (96.4)	49 (98.0)	55 (93.2)	158 (95.8)	76 (100)	73 (96.1)	139 (97.9)	288 (98.0)
No Response	-	-	-	-	-	-	1 (0.7)	1 (0.3)
Bait								
Often	4 (7.1)	3 (6.0)	2 (3.4)	9 (5.5)	-	3 (3.9)	8 (5.6)	11 (3.7)
Sometimes	15 (26.8)	12 (24.0)	23 (39.0)	50 (30.3)	-	11 (14.5)	41 (28.9)	52 (17.7)
Rarely	6 (10.7)	2 (4.0)	6 (10.2)	14 (8.5)	-	13 (17.1)	13 (9.2)	26 (8.8)
Never	31 (55.4)	33 (66.0)	29 (49.2)	93 (56.4)	76 (100)	49 (64.5)	79 (55.6)	204 (69.4)
No Response	-	-	-	-	-	-	1 (0.7)	1 (0.3)
Trash								
Often	-	-	-	-	-	-	-	-
Sometimes	-	-	1 (1.7)	1 (0.6)	1 (1.3)	1 (1.3)	3 (2.1)	5 (1.7)
Rarely	1 (1.8)	-	2 (3.3)	3 (1.8)	1 (1.3)	1 (1.3)	2 (1.4)	4 (1.4)
Never	55 (98.2)	50 (100)	57 (95.0)	161 (97.6)	74 (97.4)	74 (97.4)	137 (96.5)	285 (96.9)
TOTAL	56 (33.7)	50 (30.1)	60 (36.1)	166 (100)	76 (25.9)	76 (25.9)	142 (48.3)	294 (100)

Table C-13. How often anglers ate fish.

Meals in last month	1991-1992 Survey				1996 Survey			
	Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
0	9 (81.8)	12 (85.7)	13 (31.7)	34 (51.5)	-	9 (60.0)	25 (21.2)	34 (25.6)
1	-	1 (7.1)	8 (19.5)	9 (13.6)	-	2 (13.3)	17 (14.4)	19 (14.3)
2	-	1 (7.1)	8 (19.5)	9 (13.6)	-	3 (20.0)	18 (15.3)	21 (15.8)
3	-	-	2 (4.9)	2 (3.0)	-	-	12 (10.2)	12 (9.0)
4	1 (9.1)	-	2 (4.9)	3 (4.5)	-	-	6 (5.1)	6 (4.5)
5	1 (9.1)	-	-	-	-	-	6 (5.1)	6 (4.5)
6	-	-	-	-	-	-	4 (3.4)	4 (3.0)
7	-	-	1 (2.4)	1 (1.5)	-	-	3 (2.5)	3 (2.3)
8	-	-	2 (4.9)	2 (3.0)	-	-	5 (4.2)	5 (3.8)
10	-	-	4 (9.8)	4 (6.1)	-	1 (6.7)	9 (7.6)	10 (7.5)
12	-	-	-	-	-	-	7 (5.9)	7 (5.3)
15	-	-	-	-	-	-	2 (1.7)	2 (1.5)
20	-	-	-	-	-	-	4 (3.4)	4 (3.0)
30	-	-	1 (2.4)	1 (1.5)	-	-	-	-
0-1	9 (81.8)	13 (92.9)	21 (51.2)	43 (65.2)	-	11 (73.3)	42 (35.6)	53 (39.8)
>1	2 (18.2)	1 (7.1)	20 (48.8)	23 (34.8)	-	4 (26.7)	76 (64.4)	80 (60.2)
TOTAL	11 (16.7)	14 (21.2)	41 (62.1)	66 (100)	-	15 (11.3)	118 (88.7)	133 (100)

1991-92 Median consumer = 0 meals per month
1991-92 95th percentile consumer = 10 meals per month

1996 median consumer = 2 meals per month.
1996 95th percentile consumer = 12 meals per month

Meals in last week	1991-1992 Survey				1996 Survey			
	Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
0	9 (81.8)	12 (85.7)	22 (53.7)	43 (65.2)	-	13 (86.7)	63 (53.4)	76 (57.1)
1	1 (9.1)	1 (7.1)	8 (19.5)	10 (15.2)	-	-	30 (25.4)	30 (22.6)
2	1 (9.1)	1 (7.1)	5 (12.2)	7 (10.6)	-	1 (6.7)	19 (16.1)	20 (15.0)
3	-	-	3 (7.3)	3 (4.5)	-	1 (6.7)	2 (1.7)	3 (2.3)
4	-	-	1 (2.4)	1 (1.5)	-	-	3 (2.5)	3 (2.3)
5	-	-	1 (2.4)	-	-	-	-	-
6	-	-	-	-	-	-	1 (0.8)	1 (0.8)
7	-	-	1 (2.4)	1 (1.5)	-	-	-	-
0-1	10 (90.9)	13 (92.9)	30 (73.2)	53 (80.3)	-	13 (86.7)	93 (78.8)	106 (79.7)
>1	1 (9.1)	1 (7.1)	11 (26.8)	13 (19.7)	-	2 (13.3)	25 (21.2)	27 (20.3)
TOTAL	11 (16.7)	14 (21.2)	41 (62.1)	66 (100)	-	15 (11.3)	118 (88.7)	133 (100)

1991-92 Median consumer = 0 meals per week
1991-92 95th percentile consumer = 3 meals per week

1996 median consumer = 0 meals per week
1996 95th percentile consumer = 3 meals per week

Table C-14. What anglers think recipients do with fish given to them.

Expected use	1991-1992 Survey				1996 Survey			
	Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Eat fish	8 (57.1)	15 (83.3)	47 (94.0)	70 (85.4)	-	10 (76.9)	85 (91.4)	95 (89.6)
Use as bait	1 (7.1)	-	-	1 (1.2)	-	-	1 (1.1)	1 (0.9)
Fertilizer	-	-	-		-	-	1 (1.1)	1 (0.9)
DK / NR[1]	5 (35.7)	3 (16.7)	3 (6.0)	11 (13.4)	-	3 (23.1)	6 (6.5)	6 (5.7)
TOTAL	14 (17.1)	18 (22.0)	50 (61.0)	82 (100)	-	13 (12.3)	93 (87.7)	106 (100)

[1] *DK / NR* - Don't know / No response.

Table C-15 Anglers who said they gave fish away but did not themselves eat the fish.

Eat fish	1991-1992 Survey				1996 Survey			
	Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Yes	5 (35.7)	10 (55.6)	35 (70.0)	50 (61.0)	-	6 (46.2)	89 (95.7)	95 (89.6)
No	9 (64.3)	8 (44.4)	15 (30.0)	32 (39.0)	-	4 (30.8)	4 (4.3)	8 (7.5)
TOTAL	14 (17.1)	18 (22.0)	50 (61.0)	82 (100)	-	10 (9.7)	93 (90.3)	103 (100)

Table C-16. Distribution of relatives and friends with whom anglers shared fish.

Group	1991-1992 Survey				1996 Survey			
	Area 1	Area 2	Area 3	Total	Area 1	Area 2	Area 3	Total
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Anglers								
Shared fish	4 (36.4)	8 (57.1)	30 (73.2)	42 (63.6)	-	8 (53.3)	82 (69.5)	90 (67.7)
Total	11 (16.7)	14 (21.2)	41 (62.1)	66 (100)	-	15 (11.3)	118 (88.7)	133 (100)
Recipients								
Women <15	2 (15.4)	3 (13.0)	1 (1.7)	6 (6.3)	-	-	22 (22.4)	22 (20.4)
Women 15-49	2 (15.4)	4 (17.4)	18 (30.0)	24 (25.0)	-	3 (30.0)	19 (19.4)	22 (20.4)
Women >49	1 (7.7)	1 (4.3)	8 (13.3)	10 (10.4)	-	3 (30.0)	9 (9.2)	12 (11.1)
Men <15	4 (30.8)	2 (8.7)	6 (10.0)	12 (12.5)	-	2 (20.0)	24 (24.5)	26 (24.1)
Men >=15	2 (15.4)	8 (34.8)	18 (30.0)	28 (29.2)	-	1 (10.0)	16 (16.3)	17 (15.7)
Others[1]	2 (15.4)	5 (21.7)	9 (15.0)	16 (16.7)	-	1 (10.0)	8 (8.2)	9 (8.3)
High risk group[2]	8 (61.5)	9 (39.1)	25 (41.7)	42 (43.8)	-	5 (50.0)	65 (66.3)	70 (64.8)
TOTAL	13 (13.5)	23 (24.0)	60 (62.5)	96 (100)	-	10 (9.3)	98 (90.7)	108 (100)

[1] *Others* are those for whom the gender was not specified in the responses.

[2] *High risk group* is women of childbearing age (age 15-49) and children less than age 15 (<15).

Table C-17. PCB levels in selected fish species from Hudson River from 1992-96.

Location/Fish Species	1992	1993	1994	1995	1996	Average
Hudson Falls to Troy (RM 154-196)						
American eel	33.70(1)	-	-	-	-	33.70(1)
Black crappie	24.77(17)	21.81(40)	-	-	-	22.69(57)
Brown bullhead	13.35(44)	14.85(17)	14.05(34)	10.81(39)	7.04(9)	12.61(143)
Carp/Goldfish	68.11(18)	73.28(4)	49.74(7)	35.64(12)	108.78(4)	60.67(45)
Largemouth bass	11.94(52)	25.41(40)	13.58(39)	15.81(40)	10.08(28)	15.49(199)
Northern pike	7.23(17)	25.82(5)	3.20(1)	-	-	11.10(23)
Pumpkinseed	7.75(37)	21.29(6)	-	5.13(4)	-	9.26(47)
Pumpkinseed (yearling)	-	12.37(51)	13.25(60)	7.59(40)	8.06(61)	10.48(212)
Rock bass	3.70(10)	-	-	-	-	3.70(10)
Smallmouth bass	8.02(27)	-	-	-	-	8.02(27)
Walleye	22.35(6)	3.50(2)	-	-	-	17.64(8)
White perch	6.31(21)	-	-	-	-	6.31(21)
Yellow bullhead	-	9.58(4)	7.16(5)	-	10.73(2)	8.69(11)
Yellow perch	7.19(34)	40.52(24)	-	-	-	20.98(58)
Troy to Catskill (RM 110-154)						
American eel	5.92(20)	6.95(21)	-	-	-	6.45(41)
Atlantic shad	0.62(15)	0.67(7)	-	-	-	0.64(22)
Black crappie	2.20(13)	2.70(5)	-	-	-	2.34(18)
Blue crab (hepatopancreas)	5.01(3)	-	-	-	-	5.01(3)
Blue crab (muscle)	0.05(6)	-	-	-	-	0.05(6)
Blue crab (whole)	0.73(3)	-	-	-	-	0.73(3)
Blueback herring	1.25(14)	-	-	-	-	1.25(14)
Brown bullhead	3.11(2)	4.99(5)	-	3.78(20)	3.15(4)	3.85(31)
Carp/Goldfish	9.21(6)	3.92(4)	-	-	-	7.09(10)
Largemouth bass	4.16(19)	6.93(18)	7.15(20)	3.95(40)	3.71(23)	4.92(120)
Pumpkinseed	1.85(15)	-	-	-	-	1.85(15)
Pumpkinseed (yearling)	-	3.07(3)	3.11(10)	3.65(16)	2.00(12)	2.99(41)
Red-breasted sunfish	2.85(9)	-	-	-	-	2.85(9)
Rock bass	1.09(2)	0.10(1)	-	-	-	0.76(3)
Smallmouth bass	5.53(27)	11.81(17)	10.79(20)	-	3.57(20)	7.59(84)
Striped bass	7.91(30)	7.83(38)	5.00(50)	5.32(81)	3.13(34)	5.68(233)
Tiger muskellunge	-	4.42(9)	-	-	-	4.42(9)
Walleye	4.66(2)	8.46(2)	-	-	-	6.56(4)
White catfish	8.84(10)	8.80(1)	-	-	-	8.84(11)
White perch	7.05(20)	2.77(40)	3.08(39)	1.89(20)	4.20(39)	3.63(158)
Yellow perch	2.36(13)	1.27(18)	0.56(10)	-	-	1.44(41)
Catskill to Tappan Zee (RM 24-110)						
American eel	4.69(18)	-	-	-	-	4.69(18)
Atlantic shad	0.43(10)	0.51(2)	-	-	-	0.44(12)
Atlantic sturgeon	-	2.64(1)	2.72(5)	-	-	2.71(6)
Atlantic tomcod	0.30(10)	-	-	-	-	0.30(10)
Black crappie	1.34(2)	-	-	-	-	1.34(2)
Blue crab (hepatopancreas)	7.03(21)	11.31(8)	-	-	-	8.21(29)
Blue crab (muscle)	0.09(21)	0.06(8)	-	-	-	0.08(29)
Blueback herring	0.80(19)	-	-	-	-	0.80(19)
Bluefish	6.07(5)	-	-	-	-	6.07(5)
Pumpkinseed	-	1.48(3)	-	-	-	1.48(3)
Pumpkinseed (yearling)	0.84(21)	0.98(14)	2.22(23)	-	1.19(12)	1.38(70)
Striped bass	2.66(157)	2.89(171)	1.91(225)	1.74(174)	1.84(132)	2.20(859)
White catfish	7.96(23)	-	-	-	-	7.96(23)
White perch	3.85(22)	-	-	-	-	3.85(22)
Yellow perch	-	1.14(20)	-	-	-	1.14(20)

Data from Ron Sloan, NYSDEC, on January 20, 1999. The data files are checked and updated periodically for completeness and accuracy. Values are PCB concentrations as micrograms per gram wet weight (parts per million or ppm) with the number of individual fish analyzed in parenthesis. Except where noted, PCB analyses were performed on standard fillets, quantified as Aroclors and summed. The average PCB is sample-weighted. Fish that exceed the US Food and Drug Administration tolerance of 2 ppm cannot be sold in the marketplace.

Appendix D - Chronology of PCB Actions Regarding Hudson River Fisheries

Date	Action
3/18/72	Proposed PCB tolerance level of 5.0 ppm in fish flesh - US Food and Drug Administration (FDA) action. [37 FR 5705-5707]
12/6/74	Temporary tolerance level of 5.0 ppm adopted by FDA for fish. [39 FR 42746-42748]
Fall 1975	Finding of elevated PCB levels in Hudson River fish.
2/25/76	New York State Department of Environmental Conservation (NYS DEC) Regulation (6 NYCRR 12.19) adopted prohibiting taking and possession of fish in the Hudson River and its tributaries to the first impassable falls from Fort Edward to the Federal dam at Troy. Taking of American eel prohibited throughout Hudson River. Health advisories issued concurrently.
2/26/76	Amended 6 NYCRR 12.19 to permit sale of commercially taken Atlantic sturgeon over four feet in length, goldfish and American shad.
7/14/76	The taking and sale of bait-fish in the estuarine portion of the Hudson River permitted; includes bait fish as defined in regulation plus anchovies, killifish and silversides.
7/19/76	Menhaden added as bait fish in 6 NYCRR 12.19.
4/1/77	FDA proposed lowering the temporary tolerance level for PCB in fish from 5.0 ppm to 2.0 ppm. [42 FR 17487-17494]
10/14/77	Taking of American eel in Harlem and East Rivers prohibited (6 NYCRR 12.15). Renumbered to 6 NYCRR 11.2 on 8/22/78. Renumbered and divided regulations on Hudson River fisheries 6 NYCRR 12.19 becomes 6 NYCRR 11.4 for striped bass and 6 NYCRR 11.2 for other Hudson fisheries.
3/10/78	PCB "hotspot" dredging proposal announced by DEC.
7/25/78	Data for collections of fish before and after termination of PCB discharges publically released by DEC. No significant change noted. PCB levels found to be up to 50 times the temporary tolerance level of 5.0 ppm.
11/10/78	Blueback herring, alewife, Atlantic tomcod and blue crab removed from commercial fishing closure. Striped bass commercial closure reaffirmed.

Date	Action
6/29/79	FDA announced final rule of 2.0 ppm PCB in fish effective 9/28/79. [44 FR 38330]
10/5/79	FDA confirmed date of final rule for PCBs in foods, but stayed final tolerance for fish and shellfish pending hearings. [44 FR 57389]
5/1/81	FDA announced hearings on "magnitude of the human food loss" from reduction of tolerance. [46 FR 24551-24553]
6/10/81	NYS DOH issued health advisory for blue crabs due to cadmium and PCB contamination.
10/9/81	Striped bass commercial fishing closure reaffirmed based on 1981 spring collections.
2/23/82	Emergency regulation enacted to permit taking and sale of American eel to foreign countries. Strict limitations placed on sales and foreign certification of acceptance. Commercial fishing regulation (6 NYCRR 11.2) restructured to allow all species except white catfish, white perch, carp (except as bait), and goldfish (except as ornamentals). Commercial fishing for striped bass remains prohibited under 6 NYCRR 11.4.
3/9/82	US FDA announced availability of initial decision (issued 2/8/82) regarding reduction of PCB tolerance for fish and shellfish. [47 FR 10079-10080]
4/29/82	Emergency regulation which permitted the taking and sale of American eel to foreign countries expired. All certifications were found to be unacceptable. Remainder of the restructured regulation (6 NYCRR 11.2) made permanent.
10/15/82	Striped bass commercial fishing closure reaffirmed based on PCB data from spring 1982. Findings of dibenzofurans (a contaminant of PCB) and 2,3,7,8-TCDD (dioxin) in striped bass also announced.
12/13/82	Commercial fishing restrictions and health advisories announced by New Jersey for New Jersey portions of Hudson River and New York Harbor. Restrictions essentially echo New York restrictions.
12/30/82	EPA Administrator Anne Gorsuch announced withholding $20 million allocated by Congress from New York for PCB dredging project. Commissioner Robert Flacke denounced action.

Date	Action
10/11/83	Contaminant study of waterfowl from Hudson River and other state waters announced. Study to be completed in 1985.
11/15/83	Striped bass commercial fishing closure in Hudson River again reaffirmed. Future fishing regulations for striped bass discussed with commercial fishermen. PCB levels close to the PCB temporary tolerance level of 5.0 ppm.
5/10/84	Federal funds for PCB cleanup released in an agreement that ends state lawsuit against EPA. Agreement signed in US District Court in Manhattan. Another suit for same cause had been filed by several environmental groups and the Hudson River Fishermen's Association.
5/22/84	FDA announced adoption of new PCB tolerance level for fish of 2.0 ppm, effective August 20, 1984. [49 FR 21514-21529]
6/25/84	Based on elevated PCB levels, NYS DOH added advisory to EAT NONE for carp and goldfish taken from the Hudson between Troy and Catskill.
11/15/84	NYS DOH added advisories for several Hudson River species, based on reduction of the PCB tolerance to 2 ppm.
11/30/84	Emergency regulation (6 NYCRR 36.1) prohibited use of any gill nets in Hudson River during striped bass closed season (12/1-3/14). Regulation made permanent on 2/15/85.
2/8/85	6 NYCRR 11.2 amended to prohibit commercial fishing for several additional minor fisheries (black crappie, brown bullhead, and pumpkinseed).
3/31/85	Governor Mario Cuomo announced several actions to be taken regarding striped bass in Marine District. They include: closing commercial fisheries in New York Harbor, the New York Bight and waters off western Long Island, a certification and tagging program for striped bass caught off eastern Long Island, restrictive health advisories for all striped bass, and a program of financial assistance to affected commercial fishermen.
5/1/85	Emergency regulations (6 NYCRR 11.5, 11.6 and 43.1) filed effective 5/8/85 to implement regulation of commercial harvest and sale of striped bass. Refilled on 7/12/85, 9/10/85 and 11/7/85.
5/2/85	Details of the striped bass certification and tagging program announced for eastern Long Island commercial striped bass fishery. New intensive PCB study formalized for striped bass.

Date	Action
2/18/86	New Policy on Contaminants in Fish adopted by NYS DEC. Policy formalizes NYS DEC procedures when contaminants are found in recreational and commercial fisheries.
4/18/86	Public meetings announced to discuss new findings of PCB in striped bass in the Marine District and potential regulatory alternatives.
5/5/86	Emergency regulation filed, effective 5/8/86, to prohibit all possession and sale of striped bass in New York. 6 NYCRR 11.3, 11.4, 11.5 and 11.6 consolidated into 6 NYCRR 11.3. 6 NYCRR 43.1 amended. Emergency regulation extended on 7/3/86.
7/15/86	Permanent regulation adopted prohibiting commercial and recreational taking, possession and sale of striped bass statewide. 6 NYCRR 11.3 and 43.1 consolidated into 6 NYCRR 11.3.
7/13/87	NYS DOH added advisories to EAT NONE for walleye and striped bass and EAT NO MORE THAN A MEAL PER MONTH for bluefish, northern pike taken from Troy to and including the New York Harbor.
6/21/88	NYS DOH added advisory to EAT NO MORE THAN A MEAL PER MONTH for bluefish from marine waters.
4/16/92	NYS DOH revised advisories for the Hudson south of the Troy dam for (1) black crappie, brown bullhead and pumpkinseed to EAT NO MORE THAN ONE MEAL PER WEEK from eat no more than one meal per month and (2) walleye and largemouth bass to EAT NO MORE THAN ONE MEAL PER MONTH from eat none. An advisory for smallmouth bass was added to EAT NO MORE THAN ONE MEAL PER MONTH. The advisory was revised for striped bass from the Tappan Zee Bridge south to and including the New York Harbor to EAT NO MORE THAN ONE MEAL PER MONTH from eat none. These changes were in response to new data which showed decreases in PCB levels.
7/20/93	Renumbered 6 NYCRR 36.1 which prohibited use of any gill nets in Hudson River during striped bass closed season (12/1-3/14) to 6 NYCRR 36.3.
4/21/94	NYS DOH revised advisories for the Hudson River between Troy and Catskill to EAT NONE for all species except American shad and to EAT NO MORE THAN A MEAL PER MONTH for most species from the Hudson south of Catskill. New data showed that PCB levels had increased in most species. Simplified advisory format was adopted to more clearly describe the advisories.
4/19/95	NYS DEC Commissioner Zagata requested that NYS DOH review the public health implications of allowing catch-and-release fishing in the Hudson River between Hudson Falls and Troy.

Date	Action
5/1/95	NYS DOH Commissioner DeBuono certified that there is "no compelling public health reason for keeping the Upper Hudson River closed to recreational fishing."
5/18/95	NYS DOH changed advisory for Hudson River south of Catskill from "all species" to species-by-species advisory to remove confusion regarding several salt water fish that are found in these waters. Added a clear definition for the waters of the New York Harbor where advisories apply.
5/31/95	Governor Pataki announced NYS DEC proposal to amend 6 NYCRR 10.3 and 11.2 to permit catch-and-release fishing in the Hudson River between Hudson Falls and Troy.
6/14/95	NYS DEC proposed to amend 6 NYCRR 10.3 and 11.2 to permit catch-and-release fishing in the Hudson River between Hudson Falls and Troy.
7/31/95	Comment period on the proposed amendment closed after two public meetings and hearings on 7/17 and 7/24.
8/30/95	Effective date of 6 NYCRR 10.3 and 11.2 to permit recreational catch-and-release fishing on the Hudson River between Hudson Falls and Troy.
4/4/96	NYS DOH revised advisory for striped bass taken from Jamaica Bay from eat no more than one meal per month to EAT NO MORE THAN ONE MEAL PER WEEK.

-modified from an original by
Lawrence C. Skinner
and Edward G. Horn
of August 11, 1986
November, 1998

Appendix E - Interim Public Health Hazard Categories

CATEGORY / DEFINITION	DATA SUFFICIENCY	CRITERIA
A. Urgent Public Health Hazard This category is used for sites where short-term exposures (< 1 yr) to hazardous substances or conditions could result in adverse health effects that require rapid intervention.	This determination represents a professional judgement based on critical data which ATSDR has judged sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.	Evaluation of available relevant information* indicates that site-specific conditions or likely exposures have had, are having, or are likely to have in the future, an adverse impact on human health that requires immediate action or intervention. Such site-specific conditions or exposures may include the presence of serious physical or safety hazards.
B. Public Health Hazard This category is used for sites that pose a public health hazard due to the existence of long-term exposures (> 1 yr) to hazardous substance or conditions that could result in adverse health effects.	This determination represents a professional judgement based on critical data which ATSDR has judged sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.	Evaluation of available relevant information* suggests that, under site-specific conditions of exposure, long-term exposures to site-specific contaminants (including radionuclides) have had, are having, or are likely to have in the future, an adverse impact on human health that requires one or more public health interventions. Such site-specific exposures may include the presence of serious physical or safety hazards.
C. Indeterminate Public Health Hazard This category is used for sites in which "*critical*" data are *insufficient* with regard to extent of exposure and/or toxicologic properties at estimated exposure levels.	This determination represents a professional judgement that critical data are missing and ATSDR has judged the data are insufficient to support a decision. This does not necessarily imply all data are incomplete; but that some additional data are required to support a decision.	The health assessor must determine, using professional judgement, the "criticality" of such data and the likelihood that the data can be obtained and will be obtained in a timely manner. Where some data are available, even limited data, the health assessor is encouraged to the extent possible to select other hazard categories and to support their decision with clear narrative that explains the limits of the data and the rationale for the decision.
D. No Apparent Public Health Hazard This category is used for sites where human exposure to contaminated media may be occurring, may have occurred in the past, and/or may occur in the future, but the exposure is not expected to cause any adverse health effects.	This determination represents a professional judgement based on critical data which ATSDR considers sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.	Evaluation of available relevant information* indicates that, under site-specific conditions of exposure, exposures to site-specific contaminants in the past, present, or future are not likely to result in any adverse impact on human health.
E: No Public Health Hazard This category is used for sites that, because of the absence of exposure, do NOT pose a public health hazard.	Sufficient evidence indicates that no human exposures to contaminated media have occurred, none are now occurring, and none are likely to occur in the future	

**Such as environmental and demographic data; health outcome data; exposure data; community health concerns information; toxicologic, medical, and epidemiologic data; monitoring and management plans.*

Appendix F - Summary of Public Comments and Responses

This summary was prepared to address comments and questions on the public comment draft of the Hudson River Health Consultation. The public was invited to review the draft during the public comment period, which ran from March 19, 1999 to May 1, 1999. We received two written comments, one from an angler and the other from a public agency. Similar comments were consolidated or grouped together and some statements reworded to clarify the comment. If you have any questions about this summary, you may contact the New York State Department of Health's (NYS DOH) Outreach Unit at the toll free number: 1-800-458-1158, extension 27530.

Comment #1 - Why does this report extend into other bodies of water? It appears that you are trying "guilt by association" as a scare tactic.
Response #1 - This report commented on findings from a previous, similar study that surveyed anglers in the same part of the Hudson River but also interviewed anglers further south to the Battery and into New York City Harbor waters. The health advisories for eating fish from the Hudson River south the Tappan Zee Bridge and the New York City Harbor waters are the same as for the Hudson north of the Tappan Zee Bridge to Catskill because PCB contamination of fish is similar throughout this part of the Hudson and the New York City Harbor.

Comment #2 - Presentation of statistical analysis makes the report less readable.
Response #2 - The report was prepared for an audience with a variety of backgrounds. The summary avoided statistical jargon to improve readability for the general public.

Comment #3 -The NYS DOH is using old and outdated data. Table C-17 displays PCB data for selected fish species from 1992-1996. Newer data would show that fish are safe to eat.
Response #3 - The survey was conducted in 1996. The most recent PCB data available when the data were being analyzed were also from 1996. Because many species were not sampled in 1994-1996, we included data from earlier years to show the PCB levels in the variety of species that were sampled (see Table C-17 in the report).

Comment #4 - PCB levels in crab meat were last measured at 0.06 ppm PCB and the advisory is to eat no more than six crabs per week. Atlantic shad were 0.51 ppm yet the advice is to eat as much as you want. How is this possible?
Response #4 - Blue crabs are also contaminated with cadmium. The advisory for blue crabs is based primarily on the cadmium contamination. The health advisory notes that no one should eat more than one meal per week of fish from any fresh water, the Hudson estuary and a number of other waters around New York City. Atlantic shad are covered by this advice. Women of childbearing age, infants and children under the age of 15 are advised to not eat any fish from the Hudson estuary, including Atlantic shad. However, the advisory does note that

> "[a] few meals of Hudson River shad meat and roe, especially using cooking and trimming methods that minimize PCB content, would not pose an unacceptable health risk for women of childbearing age and children, assuming this is their only significant exposure to PCBs."

Comment #5 - Table C-17 should be expanded greatly by species, parts of fish and section of river (including Tappan Zee to the Battery) so fishermen can judge the validity of the NYS DOH health advisories.

Response #5 - For 1992-1996 and the portion of the Hudson surveyed, no other fish species were sampled than those listed in Table C-17. For most of the species listed, fish were collected from only one or two locations within each of the three reaches presented in the Table. For some individual locations within the areas that were combined for Table C-17, the number of analyses are limited and therefore the data may not represent the actual levels of PCB contamination. This table was included to show that many fish from the Hudson are significantly contaminated with PCBs. As noted in the footnote to Table C-17, the samples were standard filets (edible portions) of individual fish. With the exception of blue crabs, other parts of the fish have not been analyzed and few data are available. Samples that were analyzed as whole fish were not included in the Table as most people do not eat the whole fish.

Comment #6 - It would be helpful to clarify that this Health Consultation is not a health risk assessment and to move the objectives section prior to the background section.
Response #6 - The Environmental Contamination section has been renamed to "Environmental Contamination and Health Concerns" and a statement has been added to clearly state that a quantitative health risk assessment is beyond the scope of this report. The background discussion and description of community health concerns are presented first to provide the rationale for the study objectives.

Comment #7 - Comparison values should be incorporated in the appendices to aid in interpretation of the sampling results.
Response #7 - A newly renamed section "Environmental Contamination and Health Concerns" now includes a brief discussion of health concerns associated with exposure to PCBs such as from eating fish from the Hudson River. Reference to the United States Food and Drug Administration (FDA) tolerance level for PCBs in fish has been added to this newly renamed section and to the footnote in Table C-17. Appendix D already includes the administrative history of the FDA tolerance level (see p. 44 [3/18/72, 12/6/74, 4/1/77], p. 45 [6/29/79, 10/5/79, 3/9/82] and p. 46 [5/22/84]).

Comment #8 - A summary of PCB standards, monitoring activities and when PCB fish sampling was performed for the Hudson River and by whom should be added to the report.
Response #8 - As explained in Response #7, reference to the FDA tolerance level has been added to the report. The newly renamed section "Environmental Contamination and Health Concerns" explains in somewhat more detail the nature and extent of fish monitoring efforts on the Hudson River.

Comment #9 - Have there been any outbreaks of food-borne diseases from eating fish from the Hudson River?
Response #9 - Outbreaks of food-borne diseases from seafood are generally caused by eating inadequately cooked shellfish contaminated with bacteria, viruses or other microorganisms. Fish are rarely a source of these outbreaks.

Comment #10 - Information about background levels of PCBs in fish in the Hudson River, how often levels are monitored, clean-up efforts and health effects associated with eating PCB-contaminated fish at various levels should be available in a Fact Sheet format for the public.

Response #10 - Numerous fact sheets describing the clean-up efforts have been prepared by the US Environmental Protection Agency and NYS Department of Environmental Conservation. The health advisory booklet issued by NYS DOH includes a section describing potential health concerns associated with eating fish contaminated with PCB and other contaminants. The ATSDR has published a Toxicological Profile for PCBs that includes a Fact Sheet format discussion of health concerns related to PCB exposure including exposure from eating PCB-contaminated fish.

Comment #11 - Are there any plans to conduct a similar study in the Hudson River south of the Tappan Zee Bridge and the New York City metropolitan area?
Response #11 - At this time, the NYS DOH is not planning to conduct a similar systematic survey of anglers in the Hudson River south of the Tappan Zee Bridge and the New York City metropolitan area. However, we are focusing on educating people who may be eating fish from the Hudson and New York City metropolitan area waters about the fish advisory.

In an effort to reduce the costs of printing and postage, please notify us if you wish your name to be deleted from our mailing list or if your address has changed.

New York State Department of Health
Center for Environmental Health
Outreach Unit
547 River Street, Room 316
Troy, NY 12180-2216
1-800-458-1158, Ext. 27530
(518) 402-7530

September, 1 2017

Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Subject: Comments on EPA's Second Five Year Review of the Hudson River Superfund Site

Dear Mr. Klawinski:

As chief executives of counties along the Hudson River Superfund Site, we have a shared interest in restoring the river's health as soon as possible. As long as unacceptable amounts of toxic PCBs remain in the Hudson, they pose a major threat to public health and economic revitalization in our waterfront communities.

The Hudson River is the keystone of the Hudson Valley's multibillion-dollar tourism economy. Plans for future economic development—including resumption of a once-vibrant commercial fishing industry and marine transport on the Champlain Canal—await completion of remediation that would fulfill the project's federally mandated goal to be "protective of human health and the environment." Dredging undertaken to date has failed to factor in decisive evidence that two to five times more contaminated sediment exist in the river than assumed at the time the EPA cleanup plan was established in 2002.

Accordingly, we call on the EPA to:

1. Declare in your Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as your draft review explicitly states.

2. Eliminate the draft review's finding that the remediation "will be protective." The EPA forecasts the cleanup "will be protective" in 53 years despite admitting eight additional years of research are needed to verify this claim.

3. Conduct a comprehensive cleanup of the Upper Hudson. The draft review fails to incorporate

any analysis by the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation showing that remaining contamination in the Upper Hudson is equivalent to (in NOAA's words) "a series of Superfund-caliber sites." Both NOAA and the DEC have concluded that additional dredging is essential. An "is not protective" determination will pave the way for this to occur.

4. Undertake a remedial investigation of the Lower Hudson. The draft review admits that PCB levels in fish and sediment in the Lower Hudson remain higher than expected—that, in fact, dredging conducted to date has had little or no beneficial impact on a 160-mile portion of the Superfund site, from the Troy Dam to the Battery in Manhattan. Even more alarming, the draft review provides no plan to investigate and remove downriver contamination.

Our counties have invested considerable time, money and effort to secure a bright future for residents. Credible data indicate that additional dredging may be needed to restore the Hudson as soon as possible—safeguarding public health in communities along it, making its fish safe to eat and allowing us to realize its full economic potential. Concluding that the remedy for the entire Hudson River Superfund site is "not protective" is absolutely essential to assure the Hudson receives the cleanup it deserves.

Sincerely,

Daniel P. McCoy
Albany County Executive

Marcus J. Molinaro
Dutchess County Executive

Steven M. Neuhaus
Orange County Executive

Edwin J. Day
Rockland County Executive

Michael P. Hein
Ulster County Executive

Robert P. Astorino
Westchester County Executive

22 Market Street
Poughkeepsie, NY 12601

ALBANY
NY 120
01 SEP '17
PM 1 L

Director Gary Klawinski
U.S. Environmental Protection Agency
Hudson River Field Office
187 Wolf Road Suite 303
Albany, NY 12205

12205-113878

FW: Draft Second Five Year Review of the Hudson River Superfund Cleanup

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Wed 9/6/2017 9:53 AM

Inbox

To: 'epahrfo@outlook.com' <epahrfo@outlook.com>;

1 attachments (117 KB)

Gary Klawinski, Draft Second Five Year Review of the Hudson River Cleanup, 8-31-17.pdf;

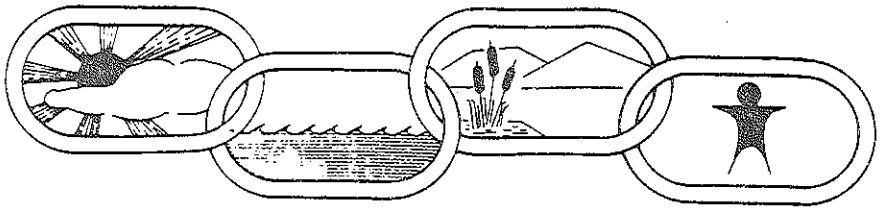
From: Gardner, Maureen [mailto:maureen.gardner@columbiacountyny.com]
Sent: Thursday, August 31, 2017 11:23 AM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>
Cc: Ed Simonsen <kinderhooked@earthlink.net>; Ellen Jouret-Epstein <jouretnoir@gtel.net>; Patrice Perry <patrice.perry@columbiacountyny.com>; Flood, Kenneth <kenneth.flood@columbiacountyny.com>; Kelly Baccaro <kelly.baccaro@columbiacountyny.com>
Subject: Draft Second Five Year Review of the Hudson River Superfund Cleanup

On behalf of the Columbia County Environmental Management Council, I have attached a letter addressed to your attention regarding the Draft Second Five Year Review of the Hudson River Superfund Cleanup. A hard copy has also been mailed to your office this morning.

Thank you for the opportunity to comment on this matter.

Sincerely,

Maureen R. Gardner, Clerk Typist
Columbia County Planning Department
401 State Street
Hudson, NY 12534
518-828-3375, extension 2382
Fax 518-828-2825
maureen.gardner@columbiacountyny.com



Columbia County
Environmental Management Council
401 State Street
Hudson, New York 12534
518-828-3375

August 31, 2017

Mr. Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Re: Draft Second Five Year Review of the Hudson River Superfund Cleanup

Dear Mr. Klawinski:

The Columbia County Environmental Management Council (EMC) consists of representatives from the County's eighteen towns and the City of Hudson, and was created under the State's Environmental Conservation Law to advise local, state and the federal government on issues related to the use, protection, and conservation of the environment for the benefit of all people.

The health of the Hudson River is inextricably linked to the environmental and economic health of all our communities. It has consequences for the air we breathe, the food we eat and sell, our drinking water, and the way we recreate. And each of these consequences has a significant impact for an economy that is heavily reliant on tourism and agriculture.

This is why we are so concerned about the conclusions stated in the Environmental Protection Agency's (EPA) Draft Second Five Year Review (FYR) of the Hudson River Superfund cleanup, mandated due to the still unknown quantities of PCB's dumped in the river by General Electric. While the EPA is claiming success for this cleanup, even the agency's own analysis shows that the effort has not reached its targeted goals and timelines, as laid out in the 2002 Record of Decision. We call upon the EPA to reconsider its conclusion, and state in the final report that the cleanup is in fact "not protective."

By law, this review is intended "to assure that human health and the environment are being protected by the remedial action being implemented." In other words, the cleanup should ensure that enough PCB 's have been removed to both protect people and help the recovery of the river's ecosystems. But the EPA's own review is actually unclear. For the upper 40 miles of the river to the Troy dam, we are told that the cleanup will, in the future, accomplish its goal. At the same time, however, it states that "more years of post-dredging data" are needed to assure long term recovery. And as for the downriver

impacts, including where Columbia County is located, the review states that it "did not predict significant impacts or major improvements from remedy implementation." In other words, there is and will be no remediation.

We knew from the outset that not all PCB's deposited by GE would be removed through this effort, but it was supposed to significantly reduce the levels of PCB's flowing downriver. Clear goals and targets were laid out for this. But one of the identified problems is that the contamination has proved to be much greater than originally estimated, in fact as much as two to four or five times greater. Thus there is much more contamination *remaining* than expected when the original goals were set. As a result, our communities will be exposed to greater levels of contaminants through the air, water, sediment, and fish for *at least a century.* What does it mean for the economic recovery counted on by local governments?

The EPA already has heard from other governmental agencies as well as independent scientists – the National Oceanic and Atmospheric Administration, the New York State Department of Environmental Conservation, the New York State Attorney General, and scientists convened by the Hudson River Foundation, among others – that the cleanup results are inadequate, *even by EPA's own measure.*

PCB contamination in the Hudson River is still a very significant threat to public health and economic prosperity. It's especially concerning that the magnitude of the threat is still not fully understood due to inadequate study. Thus it is just not credible that the EPA can state the cleanup "will be protective" in five or six decades. It is alarming that EPA considers it adequate to post fish consumption advisories. Many of the downriver communities most at risk have populations that rely on river catches for their food.

The Hudson River has been known as "America's River." It is one of our most scenic and historic American landscapes as well as a recreational and economic asset beyond measure. The Hudson River cleanup has implications for generations to come. We call upon the EPA to correct the determination reached in the Five-Year Review to say that the cleanup "is not protective" and to call for new goals and timelines.

Thank you for the opportunity to comment on this important matter.

Sincerely,

Edwin Simonsen | mtg

Edwin Simonsen, Chair
Columbia County Environmental Management Council

Cc: Columbia County Board of Supervisors

COUNTY OF DUTCHESS
MARCUS J. MOLINARO
COUNTY EXECUTIVE

June 28, 2017

RECEIVED
JUN 28 2017

Hand delivered @ Poughkeepsie

Mr. Gary Klawinski
EPA Region 2
Hudson River Office
Suite 303
187 Wolf Road
Albany, N.Y. 12205

RE: Testimony for the Environmental Protection Agency's Public Informational Meeting about the Second, Five–Year Review of the project to remove toxic PCBs from the Hudson River.

Dear Mr. Klawinski:

The Hudson River, with its beautiful waterfronts, irreplaceable ecosystems, and rich history, is a tremendous asset to Dutchess County, and is part of what makes us "Distinctly Dutchess." Since the passage of the Clean Water Act and the advent of the environmental movement born in the Hudson Valley, the Hudson River has made a tremendous comeback; and riverfront communities that once turned their backs on this polluted waterway now look to the renewed river with waterfront plans and increased access opportunities driving private and public investments. One aspect of the river's industrial age that still holds back the Hudson's revitalization is the PCB contamination hidden in its sediments that are being continually released into the river.

The Hudson River is an irreplaceable part of our communities. The EPA has already recognized the importance of the Hudson River as one of the first rivers designated as an American Heritage River, and it remains one of the nation's most beautiful and historic. The **American Heritage Rivers** receive special attention to further three objectives: natural resource and environmental protection, economic revitalization, and historic and cultural preservation. The Hudson's ability to achieve these objectives depends on the successful completion of the PCB cleanup, which is needed to improve the economy, health and quality of life of citizens in Dutchess County and throughout the Hudson Valley.

We join Scenic Hudson and others in expressing concerns regarding the conclusions reached in the Draft Second, Five-Year Review of the PCB removal project. Terms like "short-term protective" and "will be protective" do not ensure confidence in the report's conclusions, especially when it details the lack of available after-dredging test data. Terms such as the project to date "is not protective" of human health and the environment or, at the least, "protectiveness cannot be determined" would be better supported by the report. The data and analysis in the review support such a designation:

- It explicitly admits the cleanup currently is not protective—that it will take more than 50 years of natural attenuation to achieve this.

22 Market Street, Poughkeepsie New York 12601 • (845) 486-2000 • Fax: (845) 486-2021
mmolinaro@dutchessny.gov

- The report details the portions of this project that did not go as planned including: the discovery of significantly more PCBs than anticipated; inability to progress dredging from north to south; use of a single processing site; as well as lack of reduction in PCBs in Lower Hudson. It is also states that adequate post-dredging testing has not yet occurred to determine the impact of these deviations.

- While acknowledging the need for more data, the report predicts that decades in the future, some people in the upper Hudson will be able to eat one fish meal per month. This forecast is contradicted by expert scientists at the EPA's sister federal agencies, the New York State Department of Environmental Conservation and the Hudson River Foundation. All maintain it will take a century or more to reach this target.

- Finally, the EPA concedes the lower Hudson section of the Superfund site has received no health or environmental benefits from the upriver dredging. This is the most concerning to all of the Dutchess County riverfront communities. To conclude the active remediation component of this Superfund site without even a clear understanding of the sources and causes of the PCB contamination in the Lower Hudson River is unthinkable.

Fulfilling the project's federally mandated goal—and restoring the Hudson's health—is of critical importance for sustaining Dutchess County's $528-million tourism economy responsible for nearly 10,000 jobs. It's also necessary for creating jobs based on a healthy river, including the resumption of the Hudson's once-lucrative commercial fishing industry and the annual community festivals that celebrated their catch. Communities like Beacon, Poughkeepsie and Rhinebeck have made significant investments in their waterfronts to bring people and development back to the river, so it is critical these communities and the public in general understand how the future of this superfund project may or may not impact them.

The extended comment period and public informational meetings are an important step. As the EPA considers the future of this PCB cleanup project, it is vital these concerns are addressed and responded to as part of an open and transparent process.

We will never be given this opportunity again - for our generation and those that follow - do not limit the restoration of our Hudson River. Our economy, ecology, livelihoods and future depend on it."

Sincerely,

Marcus J. Molinaro
Dutchess County Executive

DUTCHESS COUNTY
Regional CHAMBER of COMMERCE

August 16, 2017

Gary Klawinski, Director Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

RE: Hudson River Superfund site Final Five-Year Review

Dear Director Klawinski:

On behalf of The Dutchess County Regional Chamber of Commerce, its board of directors and the more than 1,600 member businesses and organizations, I am writing to urge your agency to continue efforts to remediate and improve the water quality along the 200-mile span of the Hudson River Superfund site.

Established in 1907, the Dutchess County Regional Chamber of Commerce is dedicated to being an advocate for employers, the authoritative information source on matters affecting business, and the unwavering champion for the region's economic health. Our objective is to be the singular leader in the Dutchess County and larger regional business community by providing positive direction and universal support in creating a dynamic and vibrant economic environment.

Dutchess County tourism represents a vital economic sector, supporting a number of local businesses. In fact, visitors to the county spent over half a billion dollars in 2016, supporting over 10,000 jobs. We encourage a high-quality standard of living for our residents and visitors and this is fueled, in large part, by the beauty of our region, especially the Hudson River and the myriad parks along it.

Building upon this momentum depends on a clean, healthy Hudson River. As long as unacceptable levels of PCBs pollute its water, sediment and fish, they hinder economic gains—both the continuation of once-lucrative industries dependent on the river and long-stalled development opportunities along it. More importantly, they continue to pose a threat to the health of people living in riverfront communities.

For 70 years, the economic, recreational, cultural and scenic values of the Hudson River have been compromised by PCB contamination. This pollution has hampered the operation of local marinas, prevented ambitious economic development opportunities, and barred generations of residents and visitors from full enjoyment of this American Heritage River.

For these reasons, we call on the EPA to:

Declare in your Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as your draft review explicitly states.

Delete the draft review's forecast that the remediation "will be protective" in 53 years. This assumption is made despite your admission to needing eight additional years of research to verify.

Conduct additional cleanup of the Upper Hudson. Both the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation have concluded that without more dredging, it will take a century or longer for the Superfund project to achieve its goals. An "is not protective" determination will pave the way for the cleanup to continue.

Undertake a remedial investigation of the Lower Hudson. The draft review admits upriver dredging has had no effect on PCB contamination in the Lower Hudson—in fact, it is significantly higher than expected. The final review must layout a plan for investigating and removing this contamination.

Research confirms time and nature won't fix this project's shortcomings, as your draft review would lead us to believe. Only additional dredging will make the Hudson healthy as soon as possible. Therefore, we strongly urge the EPA to conclude that the remedy for the entire Hudson River Superfund site is "not protective." Then, and only then, can we provide the bright future our children and grandchildren deserve.

I appreciate your consideration and if I can help answer any questions or concerns, please do not hesitate to contact me at (845) 454-1700, ext. 1008.

Sincerely,

Frank M. Castella, Jr.
President & CEO

Hudson River Superfund Five-Year Review Comments Kingston CAC

Noble, Julie <JulieLNoble@kingston-ny.gov>

Thu 8/31/2017 3:08 PM

To:epahrfo@outlook.com <epahrfo@outlook.com>;

1 attachments (266 KB)

Kingston CAC Comments on EPA 5 year Superfund review.pdf;

Dear Mr. Klawinski,

Please find the comments from the Kingston Conservation Advisory Council regarding the EPA's Draft Five-Year Review of the Hudson River Superfund cleanup.

Thank you,

Julie Noble
Kingston CAC Chair

Julie L. Noble
Environmental Education and Sustainability Coordinator
Climate Smart Community Coordinator
City of Kingston Parks and Recreation
467 Broadway
Kingston, NY 12401
(845) 481-7339
JulieLNoble@kingston-ny.gov

Go Green! Print this email only when necessary.
Thank you for helping the City of Kingston be environmentally responsible.

City of Kingston
Conservation Advisory Council
420 Broadway
Kingston, NY 12401
(845) 481-7339

August 31, 2017

Gary Klawinski, Director
EPA Region 2
Hudson River Office
187 Wolf Road, Suite 303
Albany, NY 12205

Via email epahrfo@outlook.com

Re: Hudson River Superfund Five-Year Review

Dear Mr. Klawinski:
The Kingston Conservation Advisory Council (CAC) would like take this opportunity to comment on the EPA draft Five-Year Review for the Hudson River Superfund cleanup.

The Kingston CAC is an advisory body to the City of Kingston. The CAC's mission is to ensure the conservation of the City of Kingston's natural resources and the enhancement and protection of its environment while fostering unified action on environmental matters.

The City of Kingston has a population of 23,000 on the Hudson River, 100 miles south of Fort Edwards. The City has 2 miles of waterfront on the Hudson as well as 3 miles of tidal Rondout Creek waterfront. The City has one of the only public swimming beaches on the Hudson at Kingston Point. The Hudson River and Rondout Creek are important assets for water-based recreation, waterfront and economic development.

PCB contamination in the river remains a significant threat to public health and prosperity.

The EPA is to be commended for its role in the extensive removal of PCBs from the upper Hudson River, however, we ask that the report state that the remedy is NOT protective to human health and the environment. Phrases that "the remedy will be protective" should be removed.

As stakeholders directly impacted by contamination in the Hudson River, our concern is that the cleanup remedy did not work as intended, and is not protective of human health and the environment. Given that two to four times more PCBs were present than was assumed when the cleanup was designed, it is clear that more dredging needs to be carried out. This conclusion is supported by NOAA and NYSDEC; without more dredging, it will take a century or longer for the Superfund project to achieve its goals. The report must call for additional dredging of PCBs in the upper Hudson. Without additional dredging, more PCB contamination will spread further

down river threatening public health, wildlife and the economies of lower Hudson River communities.

We agree with the NYSDEC recommendation, informed by an independent evaluation of the information and data available for the site, "that investigation of the site be expanded to include performance of a Remedial Investigation and Feasibility Study for the lower Hudson, the portion of the site between the Federal Dam at Troy and the Battery in New York City." We concur with the NYSDEC conclusion that "this work is necessary to determine the nature and extent of PCB contamination in the sediments, water, and biota of the lower Hudson, and to evaluate remedial alternatives to address the currently uncontrolled, unacceptable risks to human health and the environment."

We thank you for considering our comments, and in the public interest, we urge the EPA to declare the Superfund cleanup not protective of human health and the environment, that additional dredging is needed in the upper Hudson, and that there be an investigation of contamination in the lower Hudson.

Sincerely,

Julie L. Noble

Chair
Kingston Conservation Advisory Council

Elizabeth Broad
Lorraine Farina
Emilie Hauser
Lynn Johnson
Kevin McEvoy
Julie Noble
Casey Schwarz

Cc: Commissioner Basil Seggos, New York State Department of Environmental Conservation 625 Broadway Albany, NY 12233-1010
Congressman John Faso, Kingston District Office 721 Broadway Kingston, NY 12401
The Honorable Andrew M. Cuomo, Governor of New York State NYS State Capitol Building Albany, NY 12224

TOWN of SARATOGA
12 SPRING STREET
SCHUYLERVILLE, NY 12871

TELEPHONE # (518) 695-3644
FAX # (518) 695-6782

August 21, 2017

Gary Klawinski
Hudson River Field Office
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Gary;

I am a life-long resident of the Town of Saratoga and am completing my 14th year as supervisor of the Town of Saratoga. I am an avid bass fisherman and regularly fish the Hudson River between Fort Edward and Stillwater (catch and release only). During the past 14 years I have regularly read reports on the dredging progress and occasionally attended CAG meetings. This past year I was officially appointed a representative of Saratoga County on the CAG group. Our old Town Hall used to be located at 30 Ferry Street on the banks of the Old Champlain Canal in Schuylerville and I have personally witnessed the overflow of the old canal onto Fort Hardy Park on numerous occasions. Prior to becoming Town Supervisor I served as Historian for the Town of Saratoga and the Village of Schuylerville and documented on numerous occasions the flooding of the Beach area and Fort Hardy Park.

I am not a scientist and often have not understood the metrics described by the EPA and GE as the dredging and backfilling operations have proceeded. However, I can assure the EPA that the river did not recover immediately after stirring up the sediments as observed by me of the numerous areas where there are currently few fish and little to no vegetation. It is clear that the remedy has not been protective of the long term or even immediate health of our river or its inhabitants.

In response to the Five-Year Review Questions I submit the following:

1. Is the remedy functioning as intended by the decision documents?
   a. It is clear from the initial information on habitat and fish samples that it is taking longer for recovery than anticipated. PCB's were far deeper and more dispersed than the ROD anticipated.
   b. Habitat reconstruction has not resulted in repopulation of species within the parameters that the ROD anticipated.

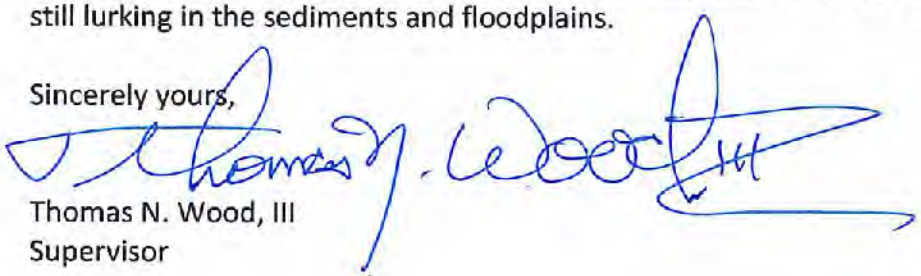
c. Re-suspension and down river re-deposition of sediments into the flood plains has not been addressed.

2. Are the exposure assumptions, toxicity, data, cleanup levels, and remedial actions objections used at the time of the remedy selection still valid?
   a. The variability of testing methods has tainted the results to date.
   b. The ROD left behind significant deposits throughout the upper Hudson that are not part of the cleanup. Those deposits are in excess of standards used in other PCB cleanup projects and leave our river subject to additional cleanup costs every time we attempt a project – whether residential or public.

3. Has any other information come to light that could call into question the protectiveness of the remedy?
   a. The original Champlain Canal was not included in the remedy and it is hydro-logically part of the Hudson River. Significant PCB concentrations were found and partially removed from the canal north of Lock 5, yet the original canal was ignored. The original canal is now so silted in with blocked culverts and dead fall that it is often stagnant and overflows the banks during heavy storms.
   b. The ROD ignored the industrial and recreational use of the river when it required dredging only to the depth of the contamination – ignoring the fact that New York State has been unable to dredge to required depths for decades. Additionally, the EPA (with the ROD as an excuse) refilled areas that had silted in over the decades – impeding industrial and recreational use.
   c. The ROD focused on river sections closer to Fort Edward, ignoring contamination of the same toxicity in river sections below Lock 5. Those areas will continue to redeposit PCB's in the upper river, the flood plains and the lower river.

For these reasons – I urge the EPA to recognize that the remedy as designed is not protective. Additional dredging is required if those of us in the upper Hudson are to have a clean river. We cannot undertake projects and use of our river with the knowledge that the legacy of PCB's is still lurking in the sediments and floodplains.

Sincerely yours,



Thomas N. Wood, III
Supervisor

Town of Saugerties pcbs Hudson River

Terri Wood <twood@saugerties.ny.us>

Tue 8/29/2017 4:52 PM

To: epahrfo@outlook.com <epahrfo@outlook.com>;

Importance: High

1 attachments (18 KB)

pcbs.pdf;

Dear Mr. Klawinski,

Please see the attached in regards to the above referenced subject.

Please confirm receipt of this e-mail-thanks.

Terri Wood
Secretary to the Supervisor
Town of Saugerties
(845) 246-2800 x345

TOWN OF SAUGERTIES

4 HIGH STREET, TOWN HALL
SAUGERTIES, NEW YORK 12477

TEL. (845) 246-2800 FAX. (845) 247-0355

GREG L. HELSMOORTEL
SUPERVISOR
JAMES J. BRUNO
DEPUTY SUPERVISOR

MEMBERS OF TOWN BOARD
FRED COSTELLO, JR.
WILLIAM M. SCHIRMER
LEEANNE THORNTON

August 29, 2017

Gary Klawinski, Director
EPA Region 2
Hudson River Office
187 Wolf Rd., Suite 303
Albany, NY 12205

Dear Mr. Klawinski,

This is to let you know that the Saugerties Town Board and myself, per the recommendation of the town's Conservation Advisory Commission (CAC), supports the NYSDEC recommendations to the EPA regarding its Five-Year Review Report on the cleanup of PCBs in the Hudson River.

Sincerely,

Greg Helsmoortel, Supervisor

cc: Town Board Members
Conservation Advisory Commission

Town of Stuyvesant

RESOLUTION OF 2017 IN SUPPORT OF ADDITIONAL REMEDIATION AND INVESTIGATION OF PCB CONTAMINATION IN THE HUDSON RIVER

WHEREAS, on June 1, 2017, the U.S. Environmental Protection Agency (EPA) released its Proposed Second Five-Year Review Report for Hudson River PCBs Superfund site, determining that the remedy in the Upper Hudson River — a 40-mile stretch above the Federal Dam at Troy, NY — "will be protective of human health and the environment upon completion;" and

WHEREAS, EPA did not reach a protectiveness determination for the Lower Hudson River — a 150 mile stretch below the Federal Dam; and

WHEREAS, nearly 200 miles of the Hudson River — from Hudson Falls to New York City — is a federal Superfund site because General Electric (GE) discharged millions of pounds of polychlorinated biphenyls (PCBs) into the River for thirty years, between 1947 and 1977; and

WHEREAS, PCBs are manmade, bioaccumulative, persistent pollutants that have been linked to adverse health effects including, among others: cancer, liver disorders, reduced birth weight and conception rates, persistent and significant deficits in neurological development; and

WHEREAS, the continued presence of PCBs in the Hudson River has significantly diminished economic activity and recreational use and enjoyment of the River; and

WHEREAS, New York State Department of Health has in place fishing restrictions and advisories against consumption of fish to control human exposure pathways that could result in unacceptable risks; and

WHEREAS, some may believe that the EPA's Proposed Second Five-Year Review Report does not adequately address scientific data and analyses presented by the National Atmospheric Administration and the New York State Department of Environmental Conservation that PCBs remain widespread and in greater amounts than expected throughout the Hudson River and that there is much more contamination left behind than originally estimated; and

WHEREAS, EPA determined dredging was needed to accelerate the timetable to reach remedial targets and deemed that delays of twenty years or more in reaching these targets were unacceptable;

WHEREAS, a peer-reviewed study published by the National Oceanic and Atmospheric Administration found that the remedial targets for Lower Hudson River may be reached decades longer than anticipated, since original model projections may have greatly underestimated the time to reach risk thresholds in the Lower Hudson River fish;

WHEREAS, the New York State Department of Environmental Conservation has concluded that the remedy may not be protective of human health and the environment, and that target fish tissue concentrations may not be met within time frames that EPA set out in the Record of Decision for the cleanup; and

WHEREAS, the dredging conducted by GE, which was completed years behind schedule, may not adequately allow for restoration of the Hudson River, and

WHEREAS, EPA admits that the remedy is not yet protective of human health and the environment in the Upper Hudson River, and that it will need eight or more years of fish tissue data to determine whether the remedy is effective; and

WHEREAS, EPA admits that the Lower Hudson River is not recovering as anticipated and that it needs more needs more information about PCB contamination in that stretch of the River; and

WHEREAS, the presence of toxic PCBs in the Hudson River will continue to threaten the health and well-being of Hudson Valley residents and the environment for years to come.

NOW THEREFORE BE IT RESOLVED, that the
Town of Stuyvesant hereby urges EPA to review its Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site and (1) officially declare that the cleanup may not be protective of human health and the environment;
(2) require GE to consider additional dredging in the Upper Hudson River; (3) require GE to perform any additional necessary removal of contaminated soils and sediments, including those in the Champlain Canal, to restore economic opportunities; (4) require GE to conduct a full remedial investigation of the Lower Hudson River; and (5) declare EPA's firm commitment to the restoration of the Hudson River, a significant natural and economic resource of New York State.

And further, BE IT RESOLVED that a copy of this resolution will be sent to:

- US Environmental Protection Agency – Region 2 and Hudson River Field Office
- John Flannery, Chairman and CEO, General Electric Corporation
- New York State Governor Andrew Cuomo
- NYS Office of the Attorney General
- NYS Department of Environmental Conservation
- NYS Canal Corporation
- National Oceanic and Atmospheric Agency
- US Department of Interior Fish and Wildlife Service
- Hudson River Congressional Delegation

Motion made at a regular meeting of the Stuyvesant town board, on August 10th 2017 by Ed Scott and seconded by Tom Burrall

VOTING MEMBERS:

Councilman Ed Scott	Yea
Councilman Tom Burrall	Yea
Councilman Brian Chittenden	Yea
Councilwoman Kelley Williams	Absent
Supervisor Ron Knott	Yea

Melissa A. Naegeli, RMC
Town Clerk
Dated: August 10, 2017
[*SEAL*]

Ulster County EMC response to Hudson River Superfund Review

Amanda Wolfson <awol@co.ulster.ny.us>

Thu 8/31/2017 7:45 AM

To:epahrfo@outlook.com <epahrfo@outlook.com>;

Cc:Dave Haldeman <davehaldeman48@gmail.com>;

1 attachments (1005 KB)

UC EMC Hudson River Superfund review response.pdf;

Please find attached a response from the Ulster County Environmental Council concerning the EPA Hudson River Superfund Five-year review.
Thank you

Ulster County EMC
Ulster County Department of the Environment
17 Pearl St.- PO Box 1800
Kingston, NY 12402
(845) 338-7287

Ulster County Environmental Management Council

August 30, 2017

Gary Klawinski, Director
EPA Region 2
Hudson River Office
187 Wolf Road, Suite 303
Albany, NY 12205

Via email epahrfo@outlook.com

Re: Hudson River Superfund Five-Year Review

Dear Mr. Klawinski:

I appreciate the opportunity to comment on behalf of New York's Ulster County Environmental Management Council regarding the EPA draft Five-Year Review for the Hudson River Superfund cleanup. The Ulster County Environmental Management Council is comprised of appointed representatives from the county's conservation advisory councils, commissions and boards in addition to at-large members.

There are twenty-four local governments in Ulster County which have a total population of about 182,000. Ulster County communities share many miles of shoreline with the lower Hudson River and two of our communities draw drinking water from the Hudson River. These factors give our council a unique interest in the health of the river. The Hudson River is an important asset for drinking water, water-based recreation, waterfront and economic development.

The EPA is to be commended for its role in the extensive removal of PCBs from the Upper Hudson River; however, we take exception to the conclusion in the draft Five-Year Review that the remedy for cleanup has resulted in protecting human health and the environment.

When considering remedies to address PCB contamination in the Hudson, the EPA determined that cancer and non-cancer health risks were well above the acceptable risk range for people who ate fish from both the upper and lower Hudson. The Superfund cleanup remedy was intended to address the risks, and the Five-Year Review is intended to ensure the risks have been adequately addressed

As stakeholders directly impacted by contamination in the Hudson River, our concern is that the cleanup remedy is not working as intended, and is not protective of human health and the environment. Given that two-to-three times as many PCBs remain in the river than expected, it is clear that more cleanup is needed. Without additional cleanup, PCB contamination will spread further down river threatening public health, wildlife and the much needed economic development of the lower Hudson River communities.

In its Hudson River PCB Superfund Summary Fact Sheet, EPA acknowledges PCB concentrations in fish in the Upper Hudson have not yet reached protective levels, though average PCB concentrations are

declining. EPA states, it "believes it is likely that improvement will occur gradually over several decades, at least."

The National Oceanic and Atmospheric Administration, the New York State Department of Environmental Conservation (NYSDEC) and the Hudson River Foundation have compiled data and have concluded the river will not recover for many decades beyond the cleanup goals established by EPA. This means the public who use the Hudson River will continue to be exposed to high levels of PCBs through air, water, sediment, and fish for possibly - another century. For those who use the river for recreation as well as those who live and work along the river, continued exposure to high levels of PCBs through air, water, sediment and fish for many decades is unacceptable.

We agree with the NYSDEC recommendation, informed by an independent evaluation of the information and data available for the site, "that investigation of the site be expanded to include performance of a Remedial Investigation and Feasibility Study for the lower Hudson, the portion of the site between the Federal Dam at Troy and the Battery in New York City." We concur with the NYSDEC conclusion that "this work is necessary to determine the nature and extent of PCB contamination in the sediments, water, and biota of the lower Hudson, and to evaluate remedial alternatives to address the currently uncontrolled, unacceptable risks to human health and the environment."

We thank you for considering our comments, and in the public interest, we urge the EPA to declare the Superfund cleanup not protective of human health and the environment, that additional dredging is needed, and more must be done to ensure the health of the river.

Yours truly,

Dave Haldeman, Chair
Ulster County Environmental Management Council

Cc: Commissioner Basil Seggos,
Chairman Kenneth Ronk, Jr.
Congressman John Faso
Governor Andrew Cuomo

Dan Carpenter
104 Green Street
Schuylerville, N.Y.
(Village of Schuylerville--dpcarpenter78@gmail.com)

September 1, 2017

Gary Klawinski
Hudson River Field Office
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski,

For the last 4 years I have represented the village of Schuylerville, first as a trustee and now as Mayor. For nineteen years I have experienced the high flows, the dredging and the moods of the river in all seasons and in all weather. I am not a scientist and quite often have not understood the metrics described by the EPA and GE as the dredging and backfilling operations have proceeded. However, I can assure the EPA that the river did not recover immediately after stirring up the sediments. It is clear that the remedy has not been protective of the long term or even immediate health of our river or its inhabitants.

Of particular concern is repeated flooding of the original Champlain Canal now plugged with sediment and un able to fulfill its 100+ year role as the collector of run off water from the Village streets and storm sewers. The recent overflows of the canal's banks threaten a serious breach which would cause the canal water to flow directly into Ft. Hardy Park over its many playing fields and possibly contaminate the critical location of the well heads of the municipal water system. Since the original canal is hydrologically part of the River at the level ABOVE lock C5 such a breach could prove catastrophic. We cannot even touch the sediments now trapped in the original canal for fear of violating the disposal protocols of the recent dredging operations. We desperately need an intervention in this threatening situation.

Further, In response to the Five-Year Review Questions I repeat the conclusions of our local working group:

1. Is the remedy functioning as intended by the decision documents? NO
   a. It is clear from the initial information on habitat and fish samples that it is taking longer for recovery than anticipated. PCB's were far deeper and more dispersed than the ROD anticipated.
   b. Habitat reconstruction has not resulted in repopulation of species within the parameters that the ROD anticipated.
   c. Resuspension and down river redisposition of sediments into the flood plains has not been addressed.

2. Are the exposure assumptions, toxicity, data, cleanup levels, and remedial actions objections used at the time of the remedy selection still valid? NO
   a. The variability of testing methods has tainted the results to date.
   b. The ROD left behind significant deposits throughout the upper Hudson that are not part of the cleanup. Those deposits are in excess of standards used in other PCB cleanup projects and leave our river subject to additional cleanup costs every time we attempt a project – whether residential or public.

3. Has any other information come to light that could call into question the protectiveness of the remedy? MOST DEFINITELY YES
   a. The original Champlain Canal was not included in the remedy and it is hydrologically part of the Hudson River. Significant PCB concentrations were found and partially removed from the canal north of Lock 5, yet the original canal was ignored. The original canal is now so silted in with blocked culverts and dead fall that it is often stagnant and overflows the banks during heavy storms.
   b. The ROD ignored the industrial and recreational use of the river when it required dredging only to the depth of the contamination – ignoring the fact that New York State has been unable to dredge to required depths for decades. Additionally, the EPA (with the ROD as an excuse) refilled areas that had silted in over the decades – impeding industrial and recreational use.
   c. The ROD focused on river sections closer to Fort Edward, ignoring contamination of the same toxicity in river sections below Lock 5. Those areas will continue to redeposit PCB's in the upper river, the flood plains and the lower river.

For these reasons – I urge the EPA to recognize that the remedy as designed is not protective. Additional dredging is required if those of us in the upper Hudson are to have a clean river. We cannot undertake projects and use of our river with the knowledge that the legacy of PCB's is still lurking in the sediments and floodplains.

Sincerely yours,

Dan Carpenter
Mayor, Village of Schuylerville

104 Green St.
12471

SEP - 1 1997

Gary Klaunski
Hudson River Field Office
187 Wolf Road Suite 303
Albany NY
12205

12205$1138 col2 2 0 5

Dan Carpenter
104 Green Street
Schuylerville, N.Y.
(Village of Schuylerville--dpcarpenter78@gmail.com)

September 1, 2017

Gary Klawinski
Hudson River Field Office
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Gary;

I am a resident of the Town of Saratoga and walk along the Hudson River in the Towns of Saratoga and Northumberland nearly every day. For the last 4 years I have represented the village of Schuylerville, first as a trustee and now as mayor. For nineteen years I have experienced the high flows, the dredging and the moods of the river in all seasons and in all weather.

I am not a scientist and quite often have not understood the metrics described by the EPA and GE as the dredging and backfilling operations have proceeded. However, I can assure the EPA that the river did not recover immediately after stirring up the sediments. It is clear that the remedy has not been protective of the long term or even immediate health of our river or its inhabitants.

In response to the Five-Year Review Questions:

Is the remedy functioning as intended by the decision documents? NO
It is clear from the initial information on habitat and fish samples that it is taking longer for recovery than anticipated. PCB's were far deeper and more dispersed than the ROD anticipated. Habitat reconstruction has not resulted in repopulation of species within the parameters that the ROD anticipated.
Resuspension and down river redisposition of sediments into the flood plains has not been addressed.

Are the exposure assumptions, toxicity, data, cleanup levels, and remedial actions objections used at the time of the remedy selection still valid? NO

The variability of testing methods has tainted the results to date.
The ROD left behind significant deposits throughout the upper Hudson that are not part of the cleanup. Those deposits are in excess of standards used in other PCB cleanup projects and leave our river subject to additional cleanup costs every time we attempt a project – whether residential or public.

Has any other information come to light that could call into question the protectiveness of the remedy? MOST DEFINITELY YES
The original Champlain Canal was not included in the remedy and it is hydrologically part of the Hudson River. Significant PCB concentrations were found and partially removed from the canal

north of Lock 5, yet the original canal was ignored. The original canal is now so silted in with blocked culverts and dead fall that it is often stagnant and overflows the banks during heavy storms.

The ROD ignored the industrial and recreational use of the river when it required dredging only to the depth of the contamination – ignoring the fact that New York State has been unable to dredge to required depths for decades. Additionally, the EPA (with the ROD as an excuse) refilled areas that had silted in over the decades – impeding industrial and recreational use.

The ROD focused on river sections closer to Fort Edward, ignoring contamination of the same toxicity in river sections below Lock 5. Those areas will continue to redeposit PCB's in the upper river, the flood plains and the lower river.

For these reasons – I urge the EPA to recognize that the remedy as designed is not protective. Additional dredging is required if those of us in the upper Hudson are to have a clean river. We cannot undertake projects and use of our river with the knowledge that the legacy of PCB's is still lurking in the sediments and floodplains.

Sincerely yours,

Dan Carpenter

Dan Carpenter
Mayor, Village of Schuylerville

Dan Carpenter
104 Green St
Schuylerville N.Y 12871

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01 SEP 2017 PM 1 L

Gary Klawinski
Hudson River Field office
187 Wolf Rd. Suite 303
Albany N.Y. 12205

12205-113878

Westchester gov.com

Robert P. Astorino
County Executive

August 28, 2017

Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Subject: Comments on EPA's Second Five Year Review of the Hudson River Superfund Site

Dear Mr. Klawinski:

As chief executives of counties along the Hudson River Superfund Site, we have a shared interest in restoring the river's health as soon as possible. As long as unacceptable amounts of toxic PCBs remain in the Hudson, they pose a major threat to public health and economic revitalization in our waterfront communities.

The Hudson River is the keystone of the Hudson Valley's multibillion-dollar tourism economy. Plans for future economic development—including resumption of a once-vibrant commercial fishing industry and marine transport on the Champlain Canal—await completion of remediation that would fulfill the project's federally mandated goal to be "protective of human health and the environment." Dredging undertaken to date has failed to factor in decisive evidence that two to five times more contaminated sediment exist in the river than assumed at the time the EPA cleanup plan was established in 2002.

Accordingly, we call on the EPA to:

1. Declare in your Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as your draft review explicitly states.

2. Eliminate the draft review's finding that the remediation "will be protective." The EPA forecasts the cleanup "will be protective" in 53 years despite admitting eight additional years of research are needed to verify this claim.

Office of the County Executive

Michaelian Office Building
White Plains, New York 10601 Telephone: (914) 995-2900 E-mail: ce@westchestergov.com

3. Conduct a comprehensive cleanup of the Upper Hudson. The draft review fails to incorporate any analysis by the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation showing that remaining contamination in the Upper Hudson is equivalent to (in NOAA's words) "a series of Superfund-caliber sites." Both NOAA and the DEC have concluded that additional dredging is essential. An "is not protective" determination will pave the way for this to occur.

4. Undertake a remedial investigation of the Lower Hudson. The draft review admits that PCB levels in fish and sediment in the Lower Hudson remain higher than expected—that, in fact, dredging conducted to date has had little or no beneficial impact on a 160-mile portion of the Superfund site, from the Troy Dam to the Battery in Manhattan. Even more alarming, the draft review provides no plan to investigate and remove downriver contamination.

Our counties have invested considerable time, money and effort to secure a bright future for our residents. Credible data indicates that additional dredging may be needed to restore the Hudson as soon as possible—safeguarding public health in communities along it, making its fish safe to eat and allowing us to realize its full economic potential. Concluding that the remedy for the entire Hudson River Superfund site is "not protective" is absolutely essential to assure the Hudson receives the cleanup it deserves.

Sincerely,

Robert P. Astorino
County Executive

Westchester gov.com
Office of the County Executive
148 Martine Avenue, 9th Floor
White Plains, New York 10601

WESTCHESTER
NY 365
SECURITY
FINISH

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Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

12205-113878

Comment on Hudson River Cleanup from Catskill Mountainkeeper

CMK - Woodstock <kathy@catskillmountainkeeper.org>

Fri 9/1/2017 4:52 PM

To: EPA - Hudson River Fisheries Office <epahrfo@outlook.com>;

1 attachments (251 KB)

CMK-CommenttoEPA-HudsonRiverPCBs-20170901.pdf;

Gary Klawinski, Director

EPA Region 2, Hudson River Office

187 Wolf Road, Suite 303

Albany, NY 12205

via email: *epahrfo@outlook.com*

Dear Director Klawinski:

Please accept Catskill Mountainkeeper's attached comment on the Hudson River Cleanup.

Sincerely,
Kathleen Nolan, MD, MSL

Kathleen Nolan, MD, MSL
Senior Research Director
Catskill Mountainkeeper
kathy@catskillmountainkeeper.org
845-417-6489 (mobile)

www.catskillmountainkeeper.org

"Working together to protect the Catskills"

September 1, 2017

Gary Klawinski, Director
EPA Region 2, Hudson River Office
187 Wolf Road, Suite 303
Albany, NY 12205

via email: epahrfo@outlook.com

RE: Hudson River Cleanup (https://www3.epa.gov/hudson/)

Dear Director Klawinski:

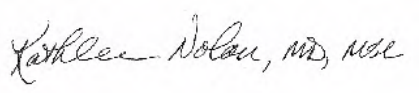
Catskill Mountainkeeper is a nationally recognized advocate for the Hudson Valley and Catskill region. Working with a network of more than 40,000 concerned citizens and strategic partners, Mountainkeeper's programs protect and promote our region's extraordinary natural heritage, while promoting smart development that supports local communities and grows our economy in a sustainable way.

Catskill Mountainkeeper urges the Environmental Protection Agency to insist that cleanup of polychlorinated biphenyls from the Hudson River be comprehensive. PCBs are toxic to humans and wildlife, and the river does not have any mechanism to rid itself of these contaminants. Instead, PCBs linger and gradually disperse into the environment, contaminating water and air and accumulating over time to dangerous levels. Exposure to aerosolized PCBs has been studied in sites near the Hudson River and has been shown to increase our risk of cancer, hypertension, heart disease, diabetes, and cancer. High-level accumulations of PCBs in fish make them too toxic for humans to eat.

General Electric dumped high concentrations of PCBs into the Hudson River, and if General Electric does not remove more of them, we are left with a 200-mile long Superfund site that is a danger to the health of anyone who breathes the air along the river or who boats on it or fishes in it.

Our clean-up efforts now are our best opportunity to return the Hudson River toward its original pristine and life-giving conditions. We know how this problem occurred (PCBs were introduced by humans into the river) and we know how to fix it (we go into the river with specialized equipment and remove them). We must demand that General Electric do its clean-up job as completely as possible, giving us back a safe and healthy river that is ready for us to enjoy now and into the future.

Sincerely,



Kathleen Nolan, MD, MSL
Senior Research Director
Catskill Mountainkeeper

kathy@catskillmountainkeeper.org
845-417-6489 (mobile)

September 1, 2017

Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski:

We represent businesses along the 200-mile span of the Hudson River Superfund site. The river is the bedrock of the Hudson Valley's current and future economic vitality. It drives the region's multibillion-dollar tourism industry and is in large part responsible for the ongoing recovery of the real estate market in the Lower Hudson since the great recession. The beauty of the river and the myriad parks along it contribute significantly to residents' quality of life and serve as catalysts for attracting visitors and new jobs.

Building upon this momentum depends on a clean, healthy Hudson River. As long as unacceptable levels of PCBs pollute its water, sediment and fish, they hinder lasting economic gains—both the resumption of once-lucrative industries dependent on the river and long-stalled development opportunities along it. More important, they continue to pose a threat to the health of people living in riverfront communities.

For 70 years, the economic, recreational, cultural and scenic values of the Hudson River have been compromised by PCB contamination. This pollution has destroyed a once-vibrant commercial fishing industry, hampered the operation of marinas, led to a severe curtailment of marine transport on the Champlain Canal, tripled the costs of dredging the NY-NJ Harbor, prevented ambitious economic development opportunities on the Upper Hudson similar to those being realized along the Mohawk River, and barred generations of residents and visitors from full enjoyment of this American Heritage River.

For these reasons, we call on the EPA to:

Declare in your Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as your draft review explicitly states.

Delete the draft review's forecast that the remediation "will be protective" in 53 years. You make this assumption despite your own admission that it will take eight additional years of research to verify.

Conduct additional cleanup of the Upper Hudson. Both the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation have concluded that without more dredging, it will take a century or longer for the Superfund project to achieve its goals. An "is not protective" determination will pave the way for the cleanup to continue.

Undertake a remedial investigation of the Lower Hudson. The draft review admits that upriver dredging has had no effect on PCB contamination in the Lower Hudson—in fact, it is significantly higher than expected. The final review must lay out a plan for investigating and removing this contamination.

Data confirm that time and nature won't fix this project's shortcomings, as your draft review would lead us to believe. Only additional dredging will make the Hudson healthy as soon as possible. Therefore, we strongly urge the EPA to conclude that the remedy for the entire Hudson River

Superfund site is "not protective." Then and only then can we begin to plan for the bright future our children and grandchildren deserve.

Sincerely,

Chambers of Commerce and Business Development Agencies

Barbara A. Corsale, President
Mechanicville-Stillwater Chamber of Commerce

Frank M. Castella, Jr., President & CEO
Dutchess County Regional Chamber of Commerce

Marla Hodge, President
Schuylerville Area Chamber of Commerce

Pete Bardunias, President & CEO
The Chamber of Southern Saratoga

Unions

Ron Diaz, Business Agent
Local Union 21

Thomas Carey, Business Agent
Plumbers and Steamfitters HVACR

Not For Profit Organizations and Institutions

Elizabeth Waldstein-Hart, Executive Director
Walkway Over the Hudson

Paul Calogerakis, Chairman
Poughkeepsie Alliance

Businesses

Wiley Harrison, Owner
The Business of your Business

Victor Mendolia, Associate Real Estate Broker
Beach & Bartolo Realtors

John Hamilton, Vice President, Finance & Corporate Development
Omnicom Group

James Cahill, President
NYS Building & Construction Trades Council

Robert Baxter, Owner
R.L. Baxter Building Corporation

Roger E. Grout, President
McDonald's REAAL, Inc.

Elyse D. Harney, Principal Broker/Owner
Elyse Harney Real Estate

Sheena Salvino, Executive Director
Hudson Development Corporation

Ted Buerger, Chairman
American Towns

Pamela Edington, Ed.D., President
Dutchess Community College

Kim Taylor, Of Counsel
Bryant Rabbino LLP

Patrick Landewe, Keeper
Saugerties Lighthouse

Joe Bonura, Principal
Bonura Hospitality Group

James Sullivan, President & Managing Director
IKOR - Life Care Management Solutions

Gregory Burns, President
Consigli Construction NY

Christian W. Meyer, President
Meyer Contracting Corporation

Catharine Hamilton, President
Putnam Market

Alex Reese, Owner
Obercreek Farm LLC

Ariel Pagan, Owner
Ugly Rooster Café

Chris Morrow, Co-owner
Northshire Bookstore

Dan Lundtquist, Owner
Five Porch Farms

Eli Lesser-Goldsmith, Co-owner and General Manger
Healthy Living

Harry Hill, Principal Broker
H H Hill Realty Services, Inc.

Joseph Cotter, CEO
National Resources, Inc

Karen Totino, Licensed Real Estate Salesperson
Green Conscience Home & Garden

Kellie McGuire, Owner
Peak Magazine

Kevin Buckel, General Manager
Hudson River Cruises

Kevin Spath, Owner
Spath Counseling Service

Kim Mathews, RLA, FASLA, Principal
Landscape Architects, P.C.

Kit Burke-Smith, Owner
Kit Burke-Smith Jewelry

Kris Seiz, Owner
Storm King Adventure Tour

Marla Hodge & Maria Saavedra, Owners
Mohawk Maiden Cruises, LLC

Mary Kay Verba, President and CEO
Dutchess Tourism Inc

Michael Oates, Managing Partner
Bellefield Development Partners, LLC

Alon Koppel, Partner
FusionLab, Inc.

Jeffrey Russell Werner Esq., Attorney
Jeffrey Russell Werner, LLC

Jaime McMillian, Founder
Spatial Dynamics

Jaime McMillian, Founder
Arts Center on Hudson

Mike Fitzgerald, Owner
Growler and Grill

Nathan Darrow, Owner
Saratoga Apple Inc.

Peggy Fusco, Owner
Gardening Angels

Leading Citizens of the Hudson Valley

Alison Spear
Alison Spear AIA

Chip Lowenson

Daniel Kramer

David H. Mortimer, President
Mary W. Harriman Foundation

David Redden, Director
David Redden LLC

Evan Mason and Garrard Beeney, Principals
Deco Works Ltd

Gary Glynn

Hoke Slaughter

Jay Saunders

James Goodfellow

Julia Widdowson

Kristin Flood

Leigh Seippel

Marjorie Hart, Acting CEO
United Catalyst, LLC.

Michael P. Dowling, Immediate Past Chair
Land Trust Alliance

Ned Whitney, Retired Managing Director
Dillon, Read & Co. Inc

Richard Klapper

Richard Krupp, Managing Partner
Pierpoint Capital

Sarah A. W. Fitts, Partner
Debevoise & Plimpton LLP

The Chamber of Southern Saratoga
County
Clinfton Country Rd #102
Clifton Park, NY 12065

ALBANY
NY 120
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PM 1 L

Director Gary Klawinski
U.S. Environmental Protection Agency
Hudson River Field Office
187 Wolf Road Suite 303
Albany, NY 12205

12205-113878

FW: FYR Team presentation

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Tue 8/15/2017 3:16 PM

To:Public Comment Hudson 2nd FYR (epahrfo@outlook.com) <epahrfo@outlook.com>;

Cc:Romanowski, Larisa <Romanowski.Larisa@epa.gov>;

From: Gil Hawkins [mailto:gilhawkins@verizon.net]
Sent: Thursday, June 15, 2017 1:16 PM
To: Fazzolari, John <JFazzolari@ene.com>
Cc: Althea Mullarkey <amullarkey@scenichudson.org>; Amy Bracewell <amy_bracewell@nps.gov>; Bridget Boyd <bridget.boyd@health.ny.gov>; Nace, Charles <Nace.Charles@epa.gov>; Metz, Chloe <Metz.Chloe@epa.gov>; Chris Debolt <cdebolt@co.washington.ny.us>; King, David <King.David@epa.gov>; Kluesner, Dave <kluesner.dave@epa.gov>; David Mathis <othroff2@aol.com>; David Tromp <david.tromp@dec.ny.gov>; Donna Davies <Donna_davies@nps.gov>; Fischer, Douglas <Fischer.Douglas@epa.gov>; Erin Doran <edoran@riverkeeper.org>; Klawinski, Gary J <Klawinski.Gary@epa.gov>; Henry, Richard (US FWS) <Richard_Henry@fws.gov>; James Candiloro <james.candiloro@canals.ny.gov>; Jay Field <jay.field@noaa.gov>; Edwards, Jennifer <Edwards.Jennifer@epa.gov>; Battipaglia, Joseph <Battipaglia.Joseph@epa.gov>; John Davis - NYS Office of the Attorney General <John.Davis@ag.ny.gov>; Joseph Savoie <joe.savoie@canals.ny.gov>; jssunit1@aol.com; Justin Deming <justin.deming@health.ny.gov>; Garufi, Katherine <Garufi.Katherine@epa.gov>; Kathryn Jahn <Kathryn_Jahn@fws.gov>; Kevin Farrar - NYS Department of Environmental Conservation (kevin.farrar@dec.ny.gov) <kevin.farrar@dec.ny.gov>; LaPoma, Jennifer <LaPoma.Jennifer@epa.gov>; Romanowski, Larisa <Romanowski.Larisa@epa.gov>; Rosman, Lisa (NOAA) <Lisa.Rosman@noaa.gov>; mannajo@clearwater.org; Greenberg, Marc <Greenberg.Marc@epa.gov>; Margaret Byrne <Margaret_Byrne@fws.gov>; Olsen, Marian <Olsen.Marian@epa.gov>; McCloe, Deepali <DMcCloe@ene.com>; Merrilyn Pulver Moulthrop <merrilyn@capital.net>; Cheplowitz, Michael <Cheplowitz.Michael@epa.gov>; Ona Ferguson <oferguson@cbuilding.org>; Patrick Field <pfield@cbuilding.org>; PE BCEE Bruce Fidler <bfidler@louisberger.com>; Peter Defur PhD (environsc@gmail.com) <environsc@gmail.com>; Peter Goutos <pgoutos@casmithllc.com>; Tom Brosnan <tom.brosnan@noaa.gov>; Lieber, Thomas <Lieber.Thomas@epa.gov>; Traynor, Michael (mtraynor@louisberger.com) <mtraynor@louisberger.com>; William Shaw <william.shaw@dec.ny.gov>
Subject: Re: FYR Team presentation

Its okay John, I have been asking the same first question for 16+ years..... I would also like to point out that in Question A, 62%-72% of what? The mass in the ROD of 3.2 million lbs. is quite different than the later discovered 5 million lbs. Which number is correct? It's a tale of two rivers! Though fish are important, the plight of the marinas in the lower river who cannot dispose of contaminated dredge spoils will go on much longer.
Best!
Gil

Vice President
Hudson River Fishermen's Association
201-446-2652
"Fight for the Hudson!"

Gil,
I am sorry I didn't see these questions during the meeting as my computer screen was committed to displaying the presentation at the EPA office. I will pass this question on to EPA.

Thanks,
John

From: G. HAWKINS [mailto:gilhawkins@verizon.net]
Sent: Thursday, June 15, 2017 11:09 AM
To: Fazzolari, John <JFazzolari@ene.com>
Subject: Re: FYR Team presentation

What monitors (baselines) are in place for sediments and fish samples below the Troy Dam? Is monitoring (baselines) ongoing at specific active projects like the Tappan Zee Bridge and the Quanta Resources in Edgewater, NJ?
~~~<^><... Gil Hawkins
sent from my iPhone

On Jun 15, 2017, at 9:20 AM, Fazzolari, John wrote:

> Five-Year Review Team,
> Please find the slides for today's meeting attached.
>
> Thanks,
> John
> <Second FYR Team Presentation.pdf>
~~~

FW: Requesting Senator Schumer's Assistance on Hudson River PCBs in New York City

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Tue 8/15/2017 3:02 PM

To: Public Comment Hudson 2nd FYR (epahrfo@outlook.com) <epahrfo@outlook.com>;

Cc: Romanowski, Larisa <Romanowski.Larisa@epa.gov>;

1 attachments (160 KB)

2017_07_07RvkLetterReFYRReportNYCMeeting_Schumer.pdf;

From: Jeremy Cherson [mailto:jcherson@riverkeeper.org]
Sent: Friday, July 07, 2017 9:32 AM
To: Kelsey_LaFreniere@schumer.senate.gov; allison_biasotti@schumer.senate.gov
Cc: Klawinski, Gary J <Klawinski.Gary@epa.gov>; Richard Webster <rwebster@riverkeeper.org>
Subject: Requesting Senator Schumer's Assistance on Hudson River PCBs in New York City

Dear Allison and Kelsey,

Please see attached a formal request from Riverkeeper for assistance from Senator Schumer in securing additional public engagement and outreach on EPA's Hudson River Superfund Five Year Review in New York City. We have also reached out to your colleagues in Senator Gillibrand's office. We look forward to hearing back from your office shortly.

Sincerely,

Jeremy Cherson
Campaign Advocacy Coordinator
Riverkeeper
20 Secor Rd. Ossining, NY
C: 770.630.6790 W. 914.478.4501 x.257
jcherson@riverkeeper.org

RIVERKEEPER
NY's clean water advocate

July 7, 2017

The Honorable Charles Schumer
Peekskill Regional Office,
One Park Place, Suite 100
Peekskill, NY 10566

Dear Senator Schumer:

I am writing on behalf of Riverkeeper, Inc.—a member-supported environmental watchdog organization dedicated to defending the Hudson River and its tributaries and to protecting the drinking water supply of nine million New York City and Hudson Valley residents—to urge you to ask the Environmental Protection Agency ("EPA") to hold a public information meeting in New York City regarding the Proposed Second Five-Year Review of the General Electric ("GE") PCB Superfund Site ("FYR Report"). The recently released FYR Report includes a misguided determination that the cleanup to date "will be protective of human health and the environment" in the Upper Hudson River, a 40-mile stretch above the Federal Dam in Troy, NY, despite the agency's admission that it needs at least eight more years of data to understand whether or not the cleanup is working. EPA also concedes that the Lower Hudson River, a 150-mile stretch below the Troy Dam to Manhattan, is not responding to the cleanup as anticipated.

Riverkeeper is deeply concerned that EPA has not adequately recognized the environmental justice implications of the PCB contamination in the Hudson River. Many New Yorkers, especially people in New York City from low-income and minority communities, either rely on subsistence fishing from the Hudson River as an important source of food or would like to do so. As such, EPA should ensure that the communities that are most interested in using the Hudson for subsistence fishing are adequately informed and have a meaningful opportunity to participate in the public comment process for the FYR Report. Unfortunately, EPA has not undertaken sufficient outreach to such communities anywhere along the Hudson River.

Ingestion of contaminated fish from the Hudson River remains a major health concern for New Yorkers, despite longstanding New York Department of Health ("NYSDOH") fish consumption advisories. EPA's own research in 2009 showed that despite state fish consumption advisories, people along the Hudson

continue to eat the fish they catch and bring them home to their families.[1] NYSDOH has also found that awareness of fish consumption advisories among anglers in New York City is about half that of anglers in the Mid and Upper Hudson, with awareness in the Lower Hudson falling between the two.[2] Recent media stories show that this lack of awareness is still problematic. For example, on June 29, 2017, a radio piece on WNYC mistakenly described the fish in New York City waters as "edible, with some exceptions" and dismissed the advisories as, "really a matter of perspective" and "quite subjective."[3] The same day, an article in New York Magazine also downplayed the health risks associated with consuming fish from New York City waterways.[4] In reality, the fish consumption advisories recommend extremely limited consumption generally, and *no* consumption for women under 50 and children under 15, for most fish species from New York City water bodies due to continued PCB contamination.[5]

Despite this significant public health concern, EPA is holding only two public information meetings along the entire 197-mile stretch of the Hudson River Superfund Site, neither of which are located in or near New York City. Moreover, it was clear from the first public information meeting in Poughkeepsie on June 28, 2017 that EPA failed to do sufficient outreach to subsistence fishing communities. When Riverkeeper asked who in the crowd of over 300 people was a subsistence fisher, not a single person raised their hand. The second meeting will take place in Saratoga Springs on July 19, 2017—180 miles from New York City. EPA did not schedule any FYR Report meetings in the low-income and minority communities downriver, which make up much of the subsistence fishing community affected by this toxic contamination.

In other places, EPA is conducting vastly more extensive community outreach at similar Superfund sites. For example, EPA Region 10 has held more than *eighty* community outreach and engagement activities since 2012 regarding the Portland Harbor Superfund Site, which is also contaminated with PCBs.[6] There, EPA identified strategies for reaching out to underrepresented communities in the region, had translators present at meetings, and attended cultural events to promote greater community engagement. Region 10 also studied fish consumption patterns among different ethnic, tribal, and immigrant groups to determine consumption rates and ensure that the risk assessments were contextualized by that consumption.[7]

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[1] U.S. Envtl. Prot. Agency, Region 2, Hudson River PCBs Superfund Site Cmty. Involvement Plan (June 2009) at 3-2, *available at* https://www3.epa.gov/hudson/pdf/hudson_river_cip_2009_update.pdf.
[2] N.Y. State Dep't of Health, PCBs Superfund Site Cmty. Advisory Grp., Hudson River Fish Advisory Outreach Project Update (Sept. 19, 2013), http://www.hudsoncag.ene.com/files/Hudson%20Fish%20Health%20Advice%20Outreach%20091913.pdf. Note that NYSDOH in its survey defined the Mid and Upper Hudson as north of the Village of Catskill and the Lower Hudson as between New York City and the Village of Catskill.
[3] New Yorkers Are Fishing For Lunch And You Can Too, The Brian Lehrer Show (June 29, 2017), http://www.wnyc.org/story/fish-your-lunch-nyc/.
[4] Alex Vadukul, *The Everything Guide to Catching Your Lunch*, N.Y. Magazine, June 29, 2017, http://nymag.com/guides/everything/everything-guide-fishing-in-nyc/.
[5] N.Y. State Dep't of Health, N.Y.C. Region Fish Advisories, (last visited July 6, 2017), https://www.health.ny.gov/environmental/outdoors/fish/health_advisories/regional/new_york_city.htm#table.
[6] U.S. Envtl. Prot. Agency, Region 10, Portland Harbor Cmty. Involvement Plan (2016), at A-2-7, *available at* https://www3.epa.gov/region10/pdf/ph/sitewide/community_involvement_plan_june2016.pdf.
[7] U.S. Envtl. Prot. Agency, Region 10, Record of Decision: Portland Harbor Superfund Site (Jan. 2017) at 35, *avaliable at* https://www3.epa.gov/region10/pdf/ph/sitewide/record-of-decision-jan2017.pdf.

In response to repeated requests from Riverkeeper and others that EPA hold a downriver meeting to provide information on the FYR Report to communities south of Poughkeepsie, and particularly to communities known to consume PCB-contaminated fish regularly or for subsistence, the agency has stated only that implementation of "institutional controls," such as riverside signs warning about consumption risks, is the responsibility of New York State. However, even if New York State is acting concurrently, EPA should still reach out to communities that rely on fish from the Hudson or would like to do so. Under Executive Order 12898, federal agencies are explicitly tasked with "identifying the need for ensuring protection of populations with differential patterns of subsistence consumption of fish and wildlife," and "communicat[ing] to the public the risks of those consumption patterns."[8]

All of your constituents who fish along the Hudson River Superfund Site deserve a meaningful opportunity to participate in the public process for the FYR Report. Riverkeeper respectfully requests that you urge EPA to ensure that the communities most interested in subsistence fishing, particularly those in the New York City area, have a meaningful chance to engage with EPA as the agency decides how to proceed with the PCB cleanup in the Hudson River. Such downriver public information meetings must be convened as soon as possible, and, at the very latest, before the September 1, 2017 deadline for submission of public comments on the FYR Report.

Please feel free to contact our Campaign Advocacy Coordinator, Jeremy Cherson, at (914) 478-4501 ext. 257 or jcherson@riverkeeper.org with any questions. I appreciate your continued involvement in this important issue.

Sincerely,

Richard Webster
Legal Director
Riverkeeper, Inc.
20 Secor Road
Ossining, NY 10562
P: (914) 478-4501
F: (914) 478-4527

[8] Exec. Order No.12,898, Fed. Actions To Address Envtl. Justice in Minority Populations and Low-Income Populations, 59 Fed. Reg. 7629 (Feb. 16, 1994), at § 4-4, *avaliable at* https://www.archives.gov/files/federal-register/executive-orders/pdf/12898.pdf.

FW: Senator Schumer and Gillibrand Letter on NYC Meeting

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Tue 8/15/2017 2:58 PM

To:Public Comment Hudson 2nd FYR (epahrfo@outlook.com) <epahrfo@outlook.com>;

Cc:Romanowski, Larisa <Romanowski.Larisa@epa.gov>;

1 attachments (237 KB)

Hudson River superfund NYC mtg 7-18-17.pdf;

From: Jeremy Cherson [mailto:jcherson@riverkeeper.org]
Sent: Wednesday, July 19, 2017 10:11 AM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>
Cc: Richard Webster <rwebster@riverkeeper.org>; Erin Doran <edoran@riverkeeper.org>
Subject: Senator Schumer and Gillibrand Letter on NYC Meeting

Mr. Klawinski,

Please see below a letter from Senators Schumer and Gillibrand requesting your office arrange a New York City meeting on the second five-year review. Given the short time frame remaining in the comment period, we request your office acts as soon as possible to give the public ample time to digest the information before submitting comments.

Please also note that due to the diversity of New York City that your office should arrange for translators and present the information in multiple languages that correspond to the communities most likely to consume fish contaminated with PCBs.

Sincerely,

Jeremy Cherson
Campaign Advocacy Coordinator
Riverkeeper
20 Secor Rd. Ossining, NY
C: 770.630.6790 W. 914.478.4501 x.257
jcherson@riverkeeper.org

RIVERKEEPER
NY's clean water advocate

United States Senate
WASHINGTON, DC 20510

July 18, 2017

Administrator Scott Pruitt
Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Dear Administrator Pruitt:

We write to urge the Environmental Protection Agency (EPA) to hold a public information meeting in New York City regarding the Proposed Second Five-Year Review Report for the Hudson River Superfund Site. It is crucial that the local community, including those along the Lower Hudson River, have the opportunity to hear directly from the EPA on this proposed report and to have their own voices be heard.

We appreciate that the EPA has already held a public information meeting in Poughkeepsie, and has another meeting planned in Saratoga Springs on July 19. However, we believe it is important that the EPA hold an additional meeting in New York City, as well, to give the maximum number of New Yorkers who live adjacent to the Hudson River an opportunity to participate in the public comment process. There is a significant population of residents in New York City who may have an interest in the status of the cleanup of the Hudson River PCBs Superfund Site and they should have maximum ability to learn about the status of this endeavor and to offer their views on current and future plans in this regard. With Poughkeepsie located more than 80 miles away from New York City, and Saratoga Springs more than 180 miles away, EPA should do more to ensure that potentially interested communities have the opportunity to hear directly from the agency and participate in the public comment process, particularly since some elderly, disabled, low-income, and other residents who have hectic schedules, yet who wish to engage in the process, may have a difficult time attending a meeting so far from home. It is for this reason that we urge the EPA to hold a public information meeting in New York City on this Proposed Second Five-Year Review Report as soon as possible.

Thank you in advance for your consideration of this request. Should you have any additional questions, please do not hesitate to contact our offices.

Sincerely,

Charles E. Schumer
United States Senator

Kirsten Gillibrand
United States Senator

Hudson River Sloop Clearwater
Hudson River Fishermen's Association
Natural Resources Defense Council
Riverkeeper
Scenic Hudson
Sierra Club

June 5, 2017

Sent Via First Class Mail and E-Mail

Gary Klawinski, Director
EPA Hudson River Field Office
187 Wolf Road, Suite 303
Albany, NY 12205
Gary.Klawinski@epa.gov

Re: Request for Extension of Public Comment Period for Hudson River PCB Superfund Five Year Review Report

Dear Mr. Klawinski:

We write to request a 90-day extension of the public comment period on the Draft Second Five Year Review (FYR) Report for the Hudson River PCB Superfund Site issued by the Environmental Protection Agency (EPA) Region 2 office on June 1. The current 30-day timeframe is woefully inadequate and would forestall meaningful public participation, which the EPA maintains is an important part of the FYR process.

The extension is necessary for riverfront communities, the public, and state and federal agencies to provide input on the EPA's highly technical review of the protectiveness of the in-river dredging remedy for the Hudson River Superfund Site. The Second FYR is a complex document containing nearly 1,000 pages of detailed information. The public deserves adequate time to understand what the report actually means— especially in regards to the significant public health threats, prohibited access and stymied economic activity this PCB-polluted waterway will inflict on people and communities for generations to come.

This extension is fully within the EPA's authority; in fact, the agency extended the comment period in 2012 for the Hudson's First FYR, a significantly shorter report with much less technical analysis. We urge the EPA to develop a more adequate timeline for this process to allow for effective public participation and foster a clearer understanding of the report's polemical conclusions. At the very least, the EPA should extend the Hudson River PCB Superfund Site Five Year Review Report public comment period until September 29, 2017.

Sincerely,

Richard Webster, Esq.
Riverkeeper, Inc.

Althea Mullarkey
Scenic Hudson, Inc.

Gil Hawkins
Hudson Fishermen's Association

Manna Jo Greene
Hudson River Sloop Clearwater, Inc.

Daniel Raichel, Esq.
Natural Resources Defense Council

Cc: Administrator Scott Pruitt, EPA HQ
Governor Andrew Cuomo, NY
Commissioner Basil Seggos, NYS DEC
Senator Kirsten Gillibrand, US Senate
Acting Administrator Cathy McCabe, EPA Region 2
Acting Deputy Administrator Walter Mugden, EPA Region 2
Representative Yvette Clark, US Congress
Representative Joe Crowley, US Congress
Representative Eliot Engel, US Congress
Representative John Faso, US Congress
Representative Steve Israel, US Congress
Representative Hakeem Jeffries, US Congress
Representative Carolyn B. Maloney, US Congress
Representative Sean Patrick Maloney, US Congress
Representative Grace Meng, US Congress
Representative Jerrold Nadler, US Congress
Representative Kathleen M. Rice, US Congress
Representative José E. Serrano, US Congress
Representative Louise M. Slaughter, US Congress
Representative Elise Stefanik, US Congress
Representative M. Velázques, US Congress
Senator William Larkin, NYS Senate
Assemblywoman Carrie Woerner, NYS Assembly

Scenic Hudson, Inc.
One Civic Center Plaza, Suite 200
Poughkeepsie, NY 12601-3157

SCENIC HUDSON
land ▪ parks ▪ advocacy

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JUN 12 2017

Acting Administrator Cathy McCabe
EPA Region 2
William Jefferson Clinton Building
1200 Pennsylvania Avenue, N. W.
Washington, DC 20460

1101

20460-

FW: Five Year Review Public Meetings on Hudson River PCB clean up

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Tue 8/15/2017 3:13 PM

To:Public Comment Hudson 2nd FYR (epahrfo@outlook.com) <epahrfo@outlook.com>;

Cc:Romanowski, Larisa <Romanowski.Larisa@epa.gov>;

-----Original Message-----
From: Richard Webster [mailto:rwebster@riverkeeper.org]
Sent: Friday, June 16, 2017 3:41 PM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>
Cc: hudsonpcb@googlegroups.com
Subject: Fwd: Five Year Review Public Meetings on Hudson River PCB clean up

Gary:

We are getting communications from our members and others complaining about the lack of meetings downstate and in New York City regarding the Five Year Review. We believe that such meetings are essential to ensure full public participation in the Five Year Review process. This is particularly important because awareness of the PCB issue is lower in New York City, the fish advisory is more complex, and there are minority communities that cannot effectively participate in meetings held only in English a long way outside of the City.

We strongly urge EPA to arrange public participation meetings in NYC and to tailor some of the meetings to minorities, who may be most interested in subsistence fishing. I trust that you agree and expect to hear from you soon. However, because of the urgency and importance of this issue we are also going to reach out to federal legislative offices with this request.

I look forward to hearing from you.

Richard Webster
Legal Director, Riverkeeper

Hudson River Comment Sheet

Julie WASH <jwash232@comcast.net>

Thu 8/31/2017 4:14 PM

To:epahrfo@outlook.com <epahrfo@outlook.com>;

1 attachments (188 KB)

hudson.pdf;

Hudson River
PCBs SUPERFUND SITE

COMMENT SHEET — 2017 Five Year Review Report

Hudson River PCBs Superfund Site

Name *(Please Print)*: Julie A. Wash

Agency/Organization: Saratoga Unites Environmental Action Committee

Address: (home) 9 Make Your Own Way, Saratoga Springs NY 12866

Written comments must be postmarked by September 1, 2017

COMMENTS:

GE's 2016 headlines Re: profit moving forward under the Trump administration is "more optimistic." Revenue = $33.1 billion. Don't let GE tell you it can't afford to clean up the Hudson. The lack of environmental responsibility on the part of GE, and the capitulation of the EPA in light of the clear and present dangers (still actively investigated by the State DEC.) need to be addressed. Sure, take time to study what you've done, but move forward with a phase to use suction dredging north of Troy, N.Y. to contain PCBs lifted out of the Hudson. Make GE use the best technology, not the cheapest. They would not survive if they just went cheap — that's Wal-Mart, not GE. Hold them accountable, EPA, -- with a NEW phase, now that they are making more profit using cheaper labor in FL -- a state with fewer regulations. We need closure, but CLEAN CLOSURE with GE.

Written Comments can be sent by mail or email to:

Gary Klawinski, Director
EPA Region2, HudsonRiver Office
187 Wolf Road, Suite 303
Albany, NY 12205
email: epahrfo@outlook.com

Comments on Hudson River Draft Five Year Review

Hayley Carlock <hcarlock@scenichudson.org>

Fri 9/1/2017 3:35 PM

Inbox

To:epahrfo@outlook.com <epahrfo@outlook.com>;

2 attachments (1 MB)

9.1.17 FYR Cover Letter to EPA and Executive Summary FINAL.pdf; 2017_09_01 FINAL FYR Comments.pdf;

Dear Director Klawinski,

Please find attached a cover letter and comments on EPA's Proposed Second Five Year Review for the Hudson River Superfund Site on behalf of Riverkeeper, Scenic Hudson, Hudson Riverkeeper Fisherman's Association, Hudson River Sloop Clearwater, Natural Resources Defense Council, and Sierra Club Atlantic Chapter.

Please note that due to the large file size, we are unable to send Attachments A-AA to our comments via email. We have sent the attachments and a hard copy of our comments via First Class mail.

Best,

Hayley Carlock, Esq.
Director of Environmental Advocacy
Scenic Hudson, Inc.
Tel: 845 473 4440 Ext 210
Fax: 845 473 2648
hcarlock@scenichudson.org

Via Electronic and First Class Mail
September 1, 2017

Gary Klawinski, Director
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Re: Comments of Hudson River Environmental Groups and Cleaner Hudson Coalition on the Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site

Dear Director Klawinski:

On behalf of Hudson River Fisherman's Association, Hudson River Sloop Clearwater, the Natural Resources Defense Council, Riverkeeper, Inc., Scenic Hudson, Inc., and the Sierra Club Atlantic Chapter we submit the attached detailed comments on the Environmental Protection Agency's Proposed Second Five-Year Review for the Hudson River PCB Superfund Site—one of the largest Superfund Sites in the United States.[1]

Our full comments are attached hereto, but we offer a summary of our primary points below.

Executive Summary

The outcome of the EPA's 5 Year Review for the Hudson River Superfund Site will set an important precedent for other Superfund sites across the nation. EPA's determination that the in-river remedy for the Hudson River Superfund Site "will be protective" of human health and the environment is arbitrary and capricious, and not supported by current data or analysis by independent scientists and the Natural Resource Trustees for the Site, including New York State. EPA acknowledges in its Proposed Second Five Year Review ("FYR") that the in-river remedy is currently "not protective"; this must be the official finding of the final FYR. The FYR should outline next steps toward additional remediation of the Upper Hudson River and commit to a remedial investigation of the Lower Hudson River. A finding by EPA that the remedy is "not protective" will put the entire Hudson River on a speedier path to recovery, and will realize the Superfund statute's goal of protecting the health of the people and wildlife living in and around the River.

Additionally, we emphasize the following:

[1] The Hudson River PCBs Superfund Site is a nearly 200-mile stretch of the Hudson River in eastern New York State from Hudson Falls, New York to the Battery in New York City. It runs adjacent to fourteen counties in New York and two counties in New Jersey. The Site is divided into the Upper Hudson River, which runs from Hudson Falls to the Federal Dam at Troy (a distance of approximately 40 miles), and the Lower Hudson River, which runs from the Federal Dam at Troy to the southern tip of Manhattan at the Battery in New York City (a distance of approximately 150 miles).

- EPA has a non-discretionary duty to ensure Superfund cleanups protect human health and the environment. Demonstrable accomplishment of the remediation goals set forth in the 2002 Record of Decision ("ROD") principally drives whether a remedy is "protective" or "not protective."

- As a threshold matter, EPA's own guidance indicates that "will be protective" is not an appropriate determination for the Hudson River Site. "Will be protective" is only appropriate when a remedy is still "under construction" (i.e., active mobilization or dredging). Because construction of the Hudson River remedy is complete and EPA admits the remedy is currently not protective, the only appropriate protectiveness determination categories pursuant to EPA guidance are "protectiveness deferred" or "not protective."

- Since consumption of fish is the major exposure pathway of concern for both people and wildlife, EPA determined in the ROD that the time to reach target PCB concentrations in fish was the primary factor in selecting the remedy for the Hudson River. It also concluded that remedial alternatives that would take 10-20 years longer to achieve targeted reductions in fish tissue PCB concentrations were "not sufficiently protective." A rapid reduction in PCB concentrations in fish—and therefore a rapid reduction in risks to people and wildlife—was the principle that drove selection of the active dredging remedy.

- Testing undertaken subsequent to the issuance of the ROD found that surface sediment concentrations of PCBs, which drive PCB concentrations in fish, were 2-3 times higher than EPA had previously assumed. After dredging, 3-5 times more residual PCB contamination in surface sediments remained than was expected. Despite this finding, EPA has not reevaluated the appropriateness of the remedy.

- The first interim fish tissue goal for the Site is 0.4 mg/kg of PCBs in species-weighted Upper Hudson average. This goal was projected in the ROD to be reached within 5 years post-dredging. It is nearly certain that the cleanup will not meet the first interim target by 2020; as of 2016, one year after dredging, PCB levels in fish were measured at 1.3 mg/kg—more than 300% greater than the ROD goal. Fish tissue concentrations would have to decline at a rate of over 25% to reach this goal, a near impossibility. Even EPA's exaggerated and unsupported 8% rate of recovery would leave this goal unmet for 10 years beyond the ROD's projection. Independent scientific analyses indicate that more realistic decay rates are 3-5% and, based on this, that the first target goal will likely not be reached for 15-40 years beyond the dates set forth in the ROD. By EPA's own statements in the ROD, such a delay renders the remedy not protective.

- By dismissing the importance of the clearly defined interim fish tissue targets, EPA in effect contends that the cleanup will be protective if it achieves the ROD's numeric remediation goals at some unknown point more than 55 years in the future. This conclusion is unacceptable, as it accepts essentially the same performance standard of the ROD's passive remediation alternatives that were rejected as not sufficiently protective of human health or the environment.

- Because PCB levels continue to present unacceptable risks to human health and the environment, the only measure protecting the public are the institutional controls for the site—specifically, New York State Department of Health's fish consumption advisories. These advisories—which warn women under age fifty and children to eat no fish at all—are ineffective, especially among low-income and minority populations, which are most likely to subsist on Hudson River fish. People all along the Hudson are exposed to toxic levels of General Electric's PCBs, through consumption of fish and other exposure pathways, and will continue to be until the goals of the ROD are reached.

- EPA fails to issue any protectiveness determination at all for the Lower Hudson in its Proposed FYR, and admits that the Lower Hudson is not seeing any detectable reductions in PCB levels as a result of the dredging project as anticipated in the ROD. EPA acknowledges that, rather, localized sediments drive PCB concentrations far more than loads from the Upper Hudson. To ensure that the entirety of the site is protective of human health and the environment, as required by the Superfund Act, EPA must immediately order General Electric to conduct a remedial investigation of the Lower Hudson to evaluate whether additional cleanup is necessary.

Respectfully submitted,

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USEPA PROPOSED SECOND FIVE YEAR REVIEW OF THE HUDSON RIVER PCBS SUPERFUND SITE

COMMENTS OF HUDSON RIVER FISHERMEN'S ASSOCIATION, HUDSON RIVER SLOOP CLEARWATER, NATURAL RESOURCES DEFENSE COUNCIL, RIVERKEEPER, SCENIC HUDSON AND SIERRA CLUB – ATLANTIC CHAPTER

SEPTEMBER 1, 2017

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I. Introduction

Substantial amounts of General Electric s (GE) toxic polychlorinated biphenyls (PCBs) are still present in the Hudson River—including in sediments, water, and fish—and there is a significant possibility that the river will remain excessively contaminated for decades. Furthermore, EPA s remedy for Hudson River sediment removal (OU2 Remedy) to date is on track to fail to achieve rapid reductions of PCBs within the specific timeframes established to protect human health and the environment. Therefore, the U.S. Environmental Protection Agency (EPA) must issue a not protective determination in the Proposed Second Five Year Review of the Hudson River PCBs Superfund Site (the Proposed Second FYR).[1]

A not protective determination is the only appropriate conclusion consistent with EPA s own Five-Year Review Guidance.[2] In addition, this determination is supported by (1) independent analyses of the Site project data; (2) current and expected environmental conditions as compared to the goals and objectives (remedial action objectives or RAOs) laid out in the 2002 Record of Decision (the 2002 ROD) for the Hudson River PCBs Superfund Site (the Site); and (3) provisions in relevant statutes and regulations, including the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or the Superfund Act)[3] and the National Contingency Plan,[4] and in operable documents that govern the remediation project.

II. Background

A. General Electric Discharged Toxic PCBs into the Hudson River for Decades, Creating Unacceptable Risks to Human Health and the Environment.

From 1947 to 1977, GE discharged untold amounts of highly toxic PCB waste from two capacitor plants into the waters of the Upper Hudson River near Fort Edward and Glens Falls.[5] PCBs are extremely resistant to decay—destruction by chemical, thermal, and biochemical processes is incredibly difficult and costly. Once in the environment, PCBs travel among soil,

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[1] *See generally* U.S. Envtl. Prot. Agency, *Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site* (May 31, 2017) [hereinafter 2017 FYR] *available at* https://www.epa.gov/sites/production/files/2017-06/documents/hudson_second_five-year_review_report.pdf. (Attachment A)

[2] *See generally* U.S. Envtl. Prot. Agency, *Comprehensive Five-Year Review Guidance* (June 2001) [hereinafter EPA FYR Guidance] *available at* http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=2000IRKW.TXT; U.S. Envtl. Prot. Agency, *Supplement to the "Comprehensive Five-Year Review Guidance* (Sept. 2011) [hereinafter EPA FYR Guidance Supplement] *available at* https://semspub.epa.gov/work/HQ/175441.pdf.

[3] 42 U.S.C. □□9601-9628.

[4] 40 C.F.R. Part 300

[5] *See* Brendan Lyons, *Dredging Up the Truth*, Albany Times Union (March 8, 2014) *available at* http://www.timesunion.com/local/article/Dredging-up-the-truth-5294643.php; *see also id.* (providing documents, including a 1968 internal GE memo on misleading regulators) *direct link to memo available at* http://web.timesunion.com/ge_dredging/graphics/1968_memo_on_misleading_regulators.pdf (No one can accurately say how many pounds of PCBs ended up in the Hudson River or the bedrock under GE's capacitor plants. A GE spokesman said the company has not issued an estimate of the volume of PCBs that were discharged to the river.). (Attachment B)

water, and air. Through these exposure pathways, animals and humans bio-accumulate PCB toxins in their bodies, especially in fatty tissues.[6]

The cumulative impacts of PCB contamination on public health and the environmental wellbeing of the riverine ecosystem have been ongoing for 70 years. Since PCBs are bio-accumulative and slow to metabolize, exposure to even low amounts of PCB toxins can cause people and animals to accumulate a much higher body-burden concentration of PCBs than exist in the immediate environment.[7]

For people, PCBs are known carcinogens,[8] endocrine disrupters, and can damage the skin, liver, pancreas, and cardiovascular system. PCBs can also impair the development of the brain and neurological system.[9] Prenatal PCB exposure has been linked to low birth weight babies and, as these children age, to reproductive, developmental, and neurobehavioral disorders that continue for several years.[10] For animals—fish, invertebrates, birds, and mammals—PCB exposure can bring about reproductive failures, developmental impairments, and mortality, causing declines in wildlife populations.[11]

B. The 2002 Record of Decision for the Hudson River Superfund Site Contains Remedial Action Objectives Necessary to Protect Human Health and the Environment.

Because of the threat posed to human health and the environment, in September 1983, much of the Hudson River—nearly 200 miles between Hudson Falls and the Battery in New York City—was recommended for placement on EPA s National Priorities List (NPL). In 1984, a record of decision was issued for the Hudson Superfund Site with an Interim No-Action decision for PCB-contaminated sediment in the river bottom, and a limited in-place capping, containment and monitoring of exposed Remnant Deposits remedy for areas of former river bottom in the Upper Hudson that had been exposed by removal of the Fort Edward Dam.[12] In 1989, as part of the subsequent five-year review of the 1984 record of decision (as required by CERCLA), EPA ordered a reassessment of the no-action remedy. In 2002, the agency issued the another record of decision—the 2002 ROD—for sediment removal (the remedy or remedial action), requiring GE to dredge PCB-contaminated sediment in the most heavily polluted areas of the Upper

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[6] U.S. Dep t of Health and Human Servs. Agency for Toxic Substances and Disease Registry, *ATDSR Case Studies in Environmental Medicine Polychlorinated Biphenyls (PCBs) Toxicity*,22 (May 14, 2014) [hereinafter ATDSR PCBs Case Study] *available at* https://www.atsdr.cdc.gov/csem/pcb/docs/pcb.pdf. (Attachment C)

[7] *Id.*

[8] *See generally* World Health Org. Int l Agency for Research on Cancer, *Polychlorinated biphenyls and polybrominated biphenyls*, IARC Monograpohs on the Evaluation of Carcinogenic Risks to Humans Vol. 107 (June 29, 2015) *available at* http://monographs.iarc.fr/ENG/Monographs/vol107/mono107.pdf. (Attachment D)

[9] Johnathan Chevrier, et al., *Associations Between Prenatal Exposure to Polychlorinated Biphenyls and Neonatal Thyroid-Stimulating Hormone Levels in a Mexican-American Population, Salinas Valley, California*, Envtl. Health Perspectives Vol. 115, 10 (Oct. 2007) *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2022659/. (Attachment E)

[10] *Id.*

[11] Hudson River Natural Res. Trustees, *Hudson River Natural Resource Damage Assessment* at 1 (Jan. 2013) [hereinafter NRDA] *available at* https://darrp.noaa.gov/sites/default/files/case-documents/PCBContamincationOfTheHudsonRiverEcosystem.pdf.

[12] U.S. Envtl. Prot. Agency, *Superfund Record of Decision: Hudson River PCBs Site, NY* (Sept. 25, 1984) [hereinafter 1984 ROD] *available at* http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=9100PYDY.TXT.

Hudson. Contaminated sediments in these hot spots posed a serious, ongoing, and unacceptable risk to human health and the environment.[13]

EPA divided the Hudson River Superfund Site into separate parts or operable units for the purpose of developing a remedial plan for each distinct part. The focus of the Proposed Second FYR is the remedial plan for Operable Unit 2 (OU2), which targets contaminated sediments located in the Upper Hudson River.[14] EPA concluded that active remediation in the Hudson River was necessary to protect the public health or welfare and the environment due to the health hazards associated with human ingestion of fish, as well as the ecological risks associated with ingestion of [Hudson River] fish by birds, fish and mammals. [15]

The 2002 ROD includes five remedial action objectives for the protection of human health and the environment:

1) Reduce the cancer risks and non-cancer health hazards for people eating fish from the Hudson River by reducing the concentration of PCBs in fish;[16]
2) Reduce PCB levels in sediment in order to meet the applicable or relevant and appropriate requirements for surface water;[17]
3) Reduce the inventory (mass) of PCBs in sediments that are or may be bioavailable;[18]
4) Minimize the long-term flow of PCBs that run over the Federal Dam and downstream through the Lower Hudson River;[19] and
5) Reduce the risks to ecological receptors by reducing the concentration of PCBs in fish.[20]

EPA also set specific numeric PCB concentration targets to assure protectiveness, as discussed in detail below.[21]

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[13] *See generally* U.S. Envtl. Prot. Agency, *Hudson River PCBs Site New York Record of Decision* (Feb. 20, 2002) [hereinafter 2002 ROD] *available at* https://www3.epa.gov/hudson/RecordofDecision-text.pdf.

[14] Other operable units include: Operable Unit 1 (1984 ROD remedy for Remnant Deposits 2-5); Operable Unit 3 (1999 EPA removal of 4,400 tons of contaminated sediments from Rodger s Island); and Operable Unit 4 (yet to be determined remedy for remediation of floodplains). U.S. Envtl. Prot. Agency, *First Five-Year Review Report for Hudson River PCBs Superfund Site*, 1 (Jun. 1, 2012) [hereinafter 2012 FYR] *available at* http://www3.epa.gov/hudson/pdf/Hudson-River-FYR-6-2012.pdf.

[15] 2002 ROD at 49.

[16] *Id.* at 50.

[17] *Id.* at 50-51. For the Hudson River Superfund Site, the federal Applicable Requirements are: 0.5 g/L total PCBs for drinking water (maximum contaminant level under the Safe Drinking Water Act); 1 ng/L for the Ambient Water Quality Criterion; and 0.014 g/L for the criteria continuous concentration Federal Water Quality Criterion in freshwater and 0.03 g/L in saltwater. 2002 ROD at 50-51. The New York State Applicable Requirements are: 0.09 g/L total PCBs for protection of human health and drinking water sources; and 0.12 ng/L for protection of wildlife; 0.001 ng/L for the protection of the health of human consumers of fish. *Id.*

[18] 2002 ROD at 51.

[19] *Id.* at 51.

[20] *Id.* at 50. The selected remedy in the ROD will achieve this in three ways: by (1) a relative reduction in toxicity quotients for the river otter and the mink, measured in the same manner as was done for reduction in risk to human health; (2) reducing the time that it would take . . . to reach the Remediation Goal for protection of ecological receptors, which is a range of PCB concentrations in largemouth bass based on the river otter, and a target range of PCB concentrations in spottail shiner based on the mink ; and (3) [r]educ[ing] PCB loading from the Upper Hudson into the Lower Hudson [to] ultimately reduce the concentrations of PCBs in sediment, water and fish and thereby reduce risk to . . . ecological receptors in the Lower Hudson. *Id.* at 73-75.

[21] *Id.* at 71.

C. EPA Selected the Remedy for the Hudson River Superfund Site Primarily Due to the Expedited Timeframe to Meet Interim and Final Remedial Targets.

In order to accomplish the RAOs, the 2002 ROD evaluated five remedial alternatives—three active remedies and two non-active remedies.[22] The three active remedies involved capping and/or dredging of contaminated sediments, followed by natural attenuation,[23] but only as applied to the northernmost forty miles of the Superfund Site—from the plant sites to the Federal Dam (the Upper Hudson River). The roughly 150 miles of the Hudson Superfund Site below the Federal Dam (the Lower Hudson River) was not . . . identified for active remediation on the assumption that active remediation in the Upper Hudson River would sufficiently reduce[] risks to humans and ecological receptors living in and near the Lower Hudson River. [24]

All three active remedial alternatives outlined in the 2002 ROD divided the Upper Hudson into three distinct sections of unequal length with varying cleanup standards based on the amount of Tri+ PCBs [25] found in surface sediment.[26] The major animating principle behind all three active alternatives was simple: remove or sequester enough PCBs in surface sediments so that PCBs would no longer get into the water column or the food chain where they would harm people and wildlife.[27] By contrast, the non-active remedies included a no action alternative and a monitored natural attenuation (natural attenuation or MNA or MNR) alternative.[28]

The time to reach the interim and final RAOs and targets for fish tissue concentrations were the *primary factor* in EPA s decision to select an active remedy, and reject the non-active alternatives as not sufficiently protective of human health and the environment.[29] To understand and compare the remedial timeframes, EPA relied on computer modeling designed to predict the short-and-long-term concentrations of PCBs in Hudson River sediment, water, and fish.[30] The

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[22] 2002 ROD at 54-66.
[23] *Id.* at 56-62.
[24] *Id.* at 2.
[25] The remedial alternatives discussed in the ROD target Tri+ PCBs defined as PCB molecules with 3 to 10 chlorine atoms based upon the finding that that the Tri+ PCB concentration ranged from 98 to 100 percent of the total PCB concentration in fish collected. 2002 ROD at 24 n.1. Total PCB levels in the Upper Hudson, however, were roughly 2-4 times higher than the Tri+ PCBs levels. *See* Jay Field et al., *Hudson River Remedy: Unremediated PCBs and the Implications for Restoration* (2011) [hereinafter Unremediated PCBs Trustee Poster] *available at* https://www.fws.gov/northeast/ecologicalservices/HudsonRiver/docs/HRES%20Hudson%20River%20PCBs%20Remedy%20Implications.pdf.
[26] For example, the REM 3/10/Select alternative—which EPA ultimately selected—called for the dredging and removal of contaminated sediments: in areas in River Section 1 with a surface concentration of greater than 3 g/m^2 of Tri+ PCBs; in areas in River Section 2 with a surface concentration more than 10 g/m^2 of Tri+ PCBs; and in select hot spots in River Section 3. 2002 ROD at 58, 94. Similarly, the CAP 3/10/Select remedy called for capping of those same sediments respectively, and the REM 0/0/3 remedy called for removal of contaminated sediments in River Sections 1, 2, and 3 in areas with surface concentrations of Tri+ PCBs of greater than 0 g/m^2, 0 g/m^2, and 3 g/m^2, respectively. *Id.*
[27] *See id.* at 50-51 (discussing remedial action objectives).
[28] The MNA alternative assumed some future control of the PCBs, which at the time were still entering the Hudson ecosystem from the contaminated plant sites (i.e., source control). *Id.* at ii.
[29] *Id.* at 66 (emphasis added).
[30] *Id.*at 26. EPA predictions for PCB fish tissue reduction timeframes were the product of a series of interconnected modeling efforts. The backbone of these efforts was the Upper Hudson River Toxic Chemical Model (HUDTOX), which forecasted PCB concentrations in water and sediment in the Upper Hudson River. U.S. Envtl. Prot. Agency, *Revised Baseline Modeling Report*, ES-2 (Jan. 2000) *available at*

modeling results led EPA to conclude that the No Action and Natural Attenuation remedial alternatives were *not sufficiently protective* of human health and the environment because: (1) the Natural Attenuation alternative would *take at least twenty years longer than the selected remedy* to reach target levels in fish tissue in River Sections 1 and 2; and (2) both non-active alternatives would not sufficiently remedy the unacceptably elevated levels of PCBs in the Upper Hudson as well as the continued degradation of the sediments and surface water quality . . . *for at least several decades longer* than any of the active remedial alternatives. [31] EPA also predicted that it would take *at least 10 additional years* for MNA to reach the 0.2 mg/kg and 0.4 mg/kg PCB target levels, as compared to the active remediation alternatives. [32] In short, EPA determined that the unacceptable risk will continue for many decades without active remediation of the PCB-contaminated sediments and control of the upstream sources. [33]

EPA acknowledged the limited interim protection provided by longstanding New York State Department of Health (NYSDOH) fish consumption advisories.[34] However, the EPA also found that these controls do not protect ecological receptors, and [that] human health risk reduction relies on *knowledge of and voluntary compliance with* the consumption advisories and fishing restrictions, citing evidence that fish consumption advisories are not fully protective of human health due to gaps in compliance. [35] Accordingly, expeditious reduction of PCBs in fish was critical to selection of the remedy and in ensuring the protection of human health and the environment.

D. The OU2 Remedy Must Meet the Specific Targets Set in the 2002 Record of Decision.

In the 2002 ROD, while EPA found all three active remedies to be sufficiently protective, it ultimately selected the REM 3/10/Select alternative. The selected remedy involved removal of sediments with PCB surface concentrations of greater than 3 g/m^2 and10 g/m^2 in River Sections 1 and 2, respectively, and select hot spots in River Section 3.[36]

Although EPA recognized that the REM-0/0/3 Alternative would be *more* protective than the selected REM 3/10/Select option, other considerations including cost and feasibility weighed in favor of the lesser protective 3/10/Select remedy.[37] The fact that the 2002 ROD set a low bar with a remedial goal of 0.05 mg/kg (allowing consumption of one half-pound fish meal a week by men), which will purportedly be met only at some unknown point more than 55 years in the

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http://www3.epa.gov/hudson/rbmr-bk1&2-chpt1-5.pdf. Outputs from HUDTOX were used as inputs in a number of bioaccumulation models, including the FISHRAND model, which ultimately predicted long-term trends in PCB fish tissue concentrations under the various remedial alternatives. *Id.* at ES-2 to ES-3.

[31] 2002 ROD at 102, 108 (emphases added).

[32] *Id.* at 103 (emphasis added).

[33] *Id.* at 102.

[34] The New York State Department of Health advisories caution that all children under 15 and women under 50 should never eat any fish from any section of the river, and that no one should ever eat fish from the Upper Hudson. *See* New York State Dep t of Health, *Hudson River Health Advice on Eating Fish You Catch* (Oct. 2016) *available at* https://www.health.ny.gov/publications/2794.pdf. Men over 15 and women over 50 are advised that they may safely eat some select species of fish in the Mid and Lower Hudson on a occasional basis. *Id.* (Attachment F)

[35] 2002 ROD at 104 (emphasis added); *see also* U.S. Envtl. Prot. Agency, *Hudson River PCBs Reassessment RI/FS Phase 3 Report: Feasibility Study* (Dec. 2000) *available at* http://www3.epa.gov/hudson/fs000001.pdf.

[36] 2002 ROD at 94.

[37] *Id.* at 104.

future, makes it even more important for EPA to ensure that the remedy is on track to meet the interim and final remedial goals that were actually projected in the 2002 ROD.

The 2002 ROD includes the following specific fish tissue and sediment targets:

Fish Tissue Target PCB Concentrations

For human exposure through consumption:

- 0.05 mg/kg in fish fillet for a person eating one-half pound meal per week
- 0.2 mg/kg in fish fillet for a person eating one half-pound meal per month
- 0.4mg/kg in fish fillet for a person eating one-half pound meal every two months

The 2002 ROD provided specific timeframes for achieving these fish tissue targets, although it is worth noting that the document itself is not entirely consistent.[38] The 2002 ROD assumed that fish tissue concentrations would meet the first interim target of 0.4 mg/kg within five years of the completion of dredging and the second interim target of 0.2 mg/kg within sixteen years of the completion of dredging.[39] While EPA did not expect the entire Upper Hudson River to meet the final remedial goal of 0.05 mg/kg within the time period modeled by GE and EPA, it did expect River Section 3 to meet that goal within 43 years of the completion of dredging.[40] Consequently EPA also expected the majority of the Lower Hudson River to meet that goal within the same timeframe due to the lower initial concentration of Site-related PCBs in the Lower Hudson compared to the Upper Hudson. [41]

The short-term targets already allow for some variation from the modeling projections used as the basis for the 2002 ROD targets. According to that modeling, EPA actually anticipated that the REM 3/10/Select Remedy would meet the 0.4 mg/kg fish tissue target within two years of the completion of dredging and the 0.2 mg/kg target within 14 years of the completion of dredging.[42] Furthermore, EPA anticipated that River Section 3 would meet the 0.05 mg/kg target within 41 years of the completion of dredging.

For wildlife exposure through consumption: [43]

- A range of 0.3 to 0.03 mg/kg in largemouth bass (whole body) for river otter
- A range of 0.7 to 0.07 mg/kg in spottail shiner (whole body) for mink

[38] As discussed herein, the 2002 ROD includes timeframes for meeting the short- and long-term targets, as well as model projections. These timelines have slight differences of up to three years. For example, the 2002 ROD expects the remedy to meet the 0.4 mg/kg within five years of the completion of dredging; *id.* at 103, whereas the model projection indicates that the remedy would meet the 0.4 mg/kg target within two years of the completion of dredging. *See* 2002 ROD, Table 11-2. For the purposes of these comments, it is assumed that EPA used 2010 as the year that dredging would be completed in the model projections.
[39] *Id.* at 50.
[40] *Id.* at 103.
[41] *Id.* at 103.
[42] *Id.* at 73.
[43] *Id.* at 50. EPA recalculated the ranges in the Second Five Year Review to 0.2 mg/kg to 0.07 mg/kg for river otter and 0.34 mg/kg to 0.11 mg/kg for mink, both of which lie within the original ranges. 2017 FYR at 65.

EPA expected an active remedy (i.e., one including dredging) to meet the remediation goal range for river otter 30 to 40 years earlier than the No Action or MNA alternatives.[44] Similarly, the agency expected to meet the target range for mink 60 years earlier with an active remedy.[45] Using the dredging period to measure the timelines in the 2002 ROD, the cleanup was expected to achieve the range for river otter within approximately 23 years of the completion of dredging, whereas the range for mink would be met during the dredging period.[46]

Sediment Target PCB Levels

To achieve fish tissue remediation goals, target cleanup levels for sediment were established based on model results relating fish tissue PCB concentrations to sediment PCB concentrations.[47] Under this process, the 2002 ROD set standards for sediment removal including an overall target of removal of approximately 2.65 million cubic yards of PCB-contaminated sediment from the Upper Hudson River, which was estimated to contain 70,000 kg (about 150,000 lbs) of total PCBs or roughly 65% of the then-estimated total PCB mass present in the Upper Hudson River.[48]

River Section 1 (Thompson Island Pool) ~ 6 miles

- 3 g/m2 Tri+ PCBs MPA
- 10 mg/kg Tri+ PCBs in surface sediment (~ 25-30 mg/kg total PCBs in top 12 inches)

River Sections 2 & 3 (multiple reaches/pools) ~ 35 miles

- 10 g/m2 Tri+ PCBs MPA
- 30 mg/kg Tri+ PCBs in surface sediment (~ 60-90 mg/kg total PCBs in top 12 inches)

The sediment removal targets were set on a mass per unit area (MPA) basis predicated on the model-estimated reduction necessary to achieve target fish tissue concentrations.[49] However, the operative fact that will drive protectiveness is the *residual* PCBs left in sediment after dredging, not how much PCB was removed.

III. The Hudson River Five Year Review Process.

EPA is statutorily required to conduct a five-year review of a Superfund site whenever contamination remains on site at a level that does not allow for unlimited use and unrestricted exposure after cleanup. [50] For communities along the nearly 200 miles of the Hudson River contaminated by PCBs, these conditions will exists for the foreseeable future.

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[44] 2002 ROD at 74.
[45] *Id.*
[46] *Id.* at 75.
[47] *Id.*
[48] *Id.* at ii, 94.
[49] Id. at 64, 94.
[50] 42 U.S.C. 9621(c) (If the President [or his delegate, in this case the EPA Administrator] selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site, the President shall

Five-year reviews are intended to evaluate the implementation and performance of remedial actions. Through this process, EPA must determine whether the selected remedy is protective of human health and the environment —or, whether the cleanup is working and activities to date will achieve the RAOs. In a five-year review report, EPA should consider the human health and ecological risks as well as the general performance of the selected remedy in order to assess the protectiveness of the cleanup. EPA must then make a protectiveness determination.

Because remedial construction is complete at the Hudson River Superfund Site, as discussed below, EPA must make a site-wide protectiveness determination, which should generally be the same protectiveness determination as the least protective Operable Unit at the site. [51] In addition, because the OU2 remedy here includes the use of institutional controls by way of the NYSDOH fish consumption advisories, EPA must also evaluate the current and long-term effectiveness of the fish consumption advisories and include relevant information about the advisories as part of the protectiveness determination. [52]

In a five-year review, EPA is directed to answer three questions based on and sufficiently supported by data and observations and then make the most appropriate protectiveness determination as guided by the condition of the river and the best available data analysis. The questions and the topics to be included under each question include (but are not limited to) the following:[53]

Question A: Is the remedy functioning as intended by the decision documents? Topics include remedial action performance and monitoring results; system operations/operations and maintenance; costs of system operations/operations and maintenance; opportunities for optimization; early indicators of potential remedy problems; and implementation of institutional controls and other measures.

Question B: Are the exposure assumptions, toxicity data, and Remedial Action Objectives (RAOs) used at the time of the remedy still valid? Topics include changes in exposure pathways; changes in land use; new contaminants and/or contaminant sources; remedy byproducts; changes in standards, newly promulgated standards, and TBCs [To Be Considereds]; changes in toxicity and other contaminant characteristics; expected progress towards meeting RAOs; and risk recalculation/assessment (as applicable).

Question C: Has any other new information come to light that could call into question the protectiveness of the remedy? Topics include ecological risks; natural disaster impacts; and any other information that could call into question the protectiveness of the remedy.

The first five-year review (First FYR) for the Hudson River Superfund Site—which EPA started and completed in only 60 days—was released on June 1, 2012 with a conclusion that the

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review such remedial action no less often than each five years after the initiation of such remedial action to assure that human health and the environment are being protected by the remedial action being implemented.).

[51] EPA FYR Guidance Supplement at 2.

[52] *Id.*

[53] EPA FYR Guidance at 3-7 (Exhibit 3-3); *see also id.* at 4-1 to 4-2 (Exhibit 4-1).

OU2 remedial action will be protective. [54] As noted to EPA in comments on the First FYR by some of our organizations,[55] the will be protective statement was erroneous and not supported by a critical review of the project as intended by both statute and EPA s own guidance.[56] While the First FYR acknowledged that high levels of contamination in areas outside of the dredging footprint would delay reaching the 2002 ROD goals within the expected timeframes,[57] EPA offered no recommendations for appropriate action to achieve the protectiveness goals.

Due to EPA s failure to recognize and adaptively manage the predicted shortcomings of the remedy, and following the EPA s de facto approval of the termination of the GE dredging program,[58] some of our organizations filed a petition (the Petition) in December 2015. The Petition demanded that EPA conduct an immediate five year review of the remedy s protectiveness and take all additional necessary actions to ensure human health and environmental RAOs are in fact being achieved.[59] We note that EPA ignored all of the recommendations and concerns expressed in the Petition in the Proposed Second FYR and only gave a cursory written response.[60]

EPA incredibly repeats its erroneous will be protective conclusion in its Proposed Second FYR, issued on June 1, 2017. EPA does so despite acknowledging that remedy is currently not protective of human health and the environment. As discussed further below, the only appropriate determination that EPA can make for the OU2 remedial action in the Proposed Second FYR is not protective.

IV. EPA Has a Duty to Ensure the Remedial Objectives Are Met.

A. EPA Has a Non-Discretionary Duty to Ensure the Remedy Protects Human Health and the Environment.

CERCLA charges EPA with ensuring that toxic pollution in our nation s most contaminated areas is prevented from harming people or the natural environment. Specifically, at Superfund sites like the Hudson River, where EPA identifies pollution that may present an imminent and substantial danger to the public health and welfare, [61] the agency must select an appropriate remedy that will attain a degree of cleanup [that] . . . *at a minimum assures protection of human health and the environment*. [62]

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[54] 2012 FYR at vi.
[55] *See* Attachment G.
[56] EPA FYR Guidance at 3-7.
[57] *See* 2012 FYR at 33-34 (River Sediment Evaluation); *id.* at 39 (Issues, Recommendations and Follow-Up Actions).
[58] In November 2015, EPA approved of the decommissioning of GE s dewatering facility and other critical infrastructure that supported the active construction of the OU2 remedy.
[59] *See* Petition to US EPA for Evaluation and Expansion of Remedial Action Selected in the 2002 Record of Decision for the Hudson River PCBs Site (hereinafter, *Petition to USEPA*), Riverkeeper, Scenic Hudson, *et. al.,* December 17, 2015 (Attachment H).
[60] Letter from Judith Enck to Petitioners (Mar. 16, 2016) (Attachment I).
[61] 42 U.S.C. 9604(a)(1).
[62] *Id.* at 9621(d) (emphasis added).

This protectiveness standard is further defined through CERCLA and its implementing regulations, which mandate that EPA develop quantifiable cleanup goals designed to eliminate quantifiable risks. In order to identify and implement remedies that are protective of human health and the environment, CERCLA requires that EPA establish site-specific remedial action objectives, including concrete and quantifiable remediation goals.[63] All remedial actions selected by the agency must assure[] protection of human health and the environment. [64] Whether a remedy succeeds or fails under this standard is measured by its ability to meet the remedial action objectives and the remediation goals.[65]

Specifically, for EPA-led cleanups, the agency must establish remedial action objectives specifying . . . remediation goals which establish acceptable exposure levels that are protective of human health and the environment. [66] These exposure levels are numeric, taking into account any federal and state maximums as well as levels associated with quantifiable cancer and non-cancer risks.[67] Indeed, as EPA sediment cleanup guidance provides, it is important that [remedial action objectives], remediation goals, and cleanup levels are based on site-specific data *and are clearly defined*. [68]

These clearly defined goals—memorialized in a record of decision—are the heart of CERCLA. Without them, there is no measurable standard by which EPA can demonstrate satisfaction of its duty to protect human health and the environment—or, alternately, one by which the public can hold the agency accountable.

Quantifiable remediation goals are also the heart of the five-year review process, where EPA . . . is legally responsible for making [a] protectiveness determination for ongoing or completed remedies.[69] The first and most significant question asked in a five-year review is whether the remedy is functioning as intended, determined primarily by whether the relevant performance standards (e.g., cleanup levels, plume containment, pumping rates) are or will likely be met. [70]

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[63] *See* 40 C.F.R. □□300.430(a)(1)(i), (e)(2)(i); *see also* 42 U.S.C. □9621(b)(1).
[64] *Id*. □9621(d)(1).
[65] *See id*. □□9621(c), (d)(1); *see also* U.S. Envtl. Prot. Agency, *Interim Guidance for Evaluation of Federal Agency Demonstrations that Remedial Actions are Operating Properly and Successfully Under CERCLA Section 120(h)(3)* (Aug. 1996), *available at* http://www2.epa.gov/fedfac/guidance-evaluation-federal-agency-demonstrations-remedial-actions-are-operating-properly-and_intro (completion of a remedial action is defined by the attainment of specific cleanup levels or performance goals that are specified in a decision document, such as a Record of Decision). *See also, e.g.*, U.S. Dep t of Energy, *Guide to Ground Water Remediation at CERCLA Response Action and RCRA Corrective Action Sites*, 7-10 (Oct. 1995) [hereinafter DOE Groundwater Guidance] *available at* http://homer.ornl.gov/sesa/environment/guidance/gw/grndh2o.pdf (The suitability and performance of any completed or ongoing ground water remedial action should be evaluated with respect to the objectives of those actions (e.g., . . . attainment of cleanup levels).).
[66] 40 C.F.R. □300.430(e)(2)(i).
[67] *Id.* at □□300.340(e)(2)(i)(A), (B)-(E).
[68] U.S. Envtl. Prot. Agency, *Contaminated Sediment Remediation Guidance for Hazardous Waste Sites*, ii (Dec. 2005) *available at* http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1000R7F.TXT (emphasis added). (Attachment J)
[69] EPA FYR Guidance Supplement at 4.
[70] EPA FYR Guidance at 4-1 (Jun. 2001); *id.* at 3-3 (stating quantitative monitoring data are the primary bases of the technical analyses and subsequent protectiveness determination(s)).

In other words, demonstrable accomplishment of the remediation goals contained in the record of decision s remedial objectives principally drives whether a remedy is protective or not protective. [71] Where RAOs and/or remedial goals may not be met, EPA must determine what additional review or action is needed.[72]

In the present case, the threat posed by GE s PCBs in the Hudson River to the health of New Yorkers and the State s environment is clear. As EPA concluded in the 2002 ROD, the significant health and ecological risks associated with the ingestion of PCB-laden fish made active remediation necessary to protect the public health or welfare and the environment. [73] To eliminate this threat, EPA developed specific RAOs and remediation goals to be achieved by the cleanup. EPA s selection of the remedy was premised on its ability to meet these criteria within a reasonably prompt timeframe.[74] The agency now has a duty to ensure that the cleanup achieves those targets in order to protect human health and the environment.

B. EPA Set Clear Goals for Protection of Human Health and the Environment in the 2002 Record of Decision and Cannot Redefine the Measure of Success.

As explained above, setting clear, identifiable remediation goals by which success or failure of a remedy can be measured is at the heart of CERCLA. In the absence of these goals, EPA would be without a measurable standard by which to demonstrate satisfaction of its duty to protect human health and the environment. Moreover, there would be no measurable standard by which EPA and potentially responsible parties—here, GE—could be held accountable.

EPA cannot dismiss the chief remedial goals of the Hudson River remedy—the clearly defined interim fish tissue targets—at this key juncture as unimportant or meaningless. Although the remedy will not be protective until the ultimate fish tissue goal of 0.05 mg/kg is met, the interim targets of 0.4 mg/kg within five years post-dredging and 0.2 mg/kg within 16 years post-dredging are important benchmarks in evaluating whether the remedy is making adequate progress.

Over the course of the Proposed Second FYR process, EPA has repeatedly dismissed the importance of these interim targets. Distressingly, in a 2016 letter to the New York State Department of Environmental Conservation (DEC), EPA implied that numeric goals for PCB levels in fish established in the 2002 ROD are no longer mandatory targets for the cleanup, but merely interim milestones that, once achieved, might allow fish advisories to be relaxed somewhat. [75] EPA also stated that the goals of the selected remedy do not include specific

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[71] *See* EPA FYR Guidance at 3-4 (review should include [d]ata supporting the effectiveness of the remedy in meeting cleanup levels and remedial action objectives identified in ROD); DOE Groundwater Guidance at 7-10 (The suitability and performance of any completed or ongoing ground water remedial action should be evaluated with respect to the objectives of those actions (e.g., . . . attainment of cleanup levels).). Thus, where quantifiable remediation goals are not met, EPA may not determine that the remedy is protective.

[72] EPA FYR Guidance at 4-9, 4-12.

[73] 2002 ROD at 49.

[74] *See id.* at 102-05.

[75] Letter from Judith Enck, U.S. Envtl. Prot. Agency Region 2 Administrator to Basil Seggos, New York Department of Environmental Conservation Commissioner at3 (Dec. 16, 2016) *available at* http://bloximages.chicago2.vip.townnews.com/poststar.com/content/tncms/assets/v3/editorial/c/dd/cdd3e1d5-03bb-5ee6-849e-c7631462ddbf/585c4e9a3209d.pdf.pdf. (Attachment K)

years in which specified PCB levels need to be achieved in fish in order for EPA to deem the remedy protective. [76]

These statements are irresponsible and contradict the fundamental goals of the 2002 ROD, which found consumption of fish [to be] the major pathway of concern for exposure to and harm from PCBs.[77] Indeed, the primary factors EPA used to select an appropriate remedy were its ability to reduce PCB concentrations in fish and [t]he time to reach target PCB concentrations in fish. [78] These remain the touchstones of a successful and protective cleanup today, and to suggest otherwise ignores the current dangers posed by unaddressed PCBs in the Hudson.

While the Proposed Second FYR concludes that the remedies at the Hudson River PCBs Superfund Site *will be protective* of human health and the environment for the Upper Hudson River,[79] it does not provide any specific timeframe in which this will occur. However, the entire point of undergoing active remediation (i.e., dredging) in addition to MNA was to reach more protective fish tissue targets in the short-term.[80] Therefore, EPA s conclusion that the remedy will be protective at some unknown and undetermined point in the future is meaningless because that is the same result that would have occurred if EPA had undertaken *no active remediation at all*.

If EPA does not hold the remedy to the interim fish tissue targets, then it will be impossible to evaluate protectiveness until the MNA period is over, some 55 or more years into the future. This is entirely inconsistent with the purpose and requirements of CERCLA, and with the remedy set forth in the 2002 ROD. As discussed *infra*, it is all but certain that the cleanup will in fact miss the five-year, 0.4 mg/kg fish tissue target. Given the lengthy and uncertain timeline to reach the remedial goal of 0.05 mg/kg, EPA must be willing to measure the effectiveness of the cleanup against the interim targets, and, importantly, admit when the cleanup is falling short.

V. A "Will Be Protective" Determination is Inappropriate for the Hudson River Remedy According to EPA Guidance.

In the Proposed Second FYR, EPA concludes that the remedy will be protective of human health and the environment.[81] Significantly, while the agency claims the remedy will be protective at some unknown point in the future, it admits that the remedy is not yet protective of human health and the environment. [82]

There are five possible conclusions EPA may reach about the protectiveness of the remedy in a five-year review:

1) Protective;
2) Will be protective;

[76] *Id.*
[77] 2002 ROD at 54.
[78] *Id.* at 54, 66.
[79] 2017 FYR at 24 (emphasis added).
[80] 2002 ROD at 104.
[81] 2017 FYR at 8, 71.
[82] *Id.*

3) Short-term protective;
4) Protectiveness cannot be determined (or protectiveness deferred); or
5) Not protective.[83]

Based on the facts and status of the OU2 Remedy, the only protectiveness determinations even potentially available to EPA are (i) not protective or (ii) protectiveness cannot be determined. As discussed further herein, based on the currently available data, EPA must determine that the OU2 remedy is *not protective*.

EPA s Comprehensive Five Year Review Guidance (EPA FYR Guidance) and Guidance Clarifying the Use of Protectiveness Determination for Comprehensive Environmental Response, Compensation and Liability Act Five Year Reviews (EPA Protectiveness Determination Guidance) give clear direction to EPA regional offices in how they are to arrive at five-year review protectiveness determinations. The latter guidance was issued in 2012 specifically to address concerns by the Office of Inspector General that regional offices were not applying protectiveness definitions consistently and were issuing protectiveness determinations that were not fully supported by data.[84]

A. Construction of the OU2 Remedy is Complete.

The status of the remedy is an operating remedial action that has not yet achieved remedial action completion. [85] This initial classification is important as it limits which protectiveness determinations are applicable to the remedy. However, as a preliminary matter, EPA must clarify that the *construction* of the remedial action is in fact complete.[86]

EPA states in its Proposed Second Five Year Review Summary Form that the site has not achieved construction completion,[87] but simply stating this in a single place in the Proposed Second FYR, without any explanation or justification whatsoever, does not make it so. A review of the rest of the Proposed Second FYR report and appendices, the 2002 ROD, and relevant EPA guidance makes it abundantly clear that the OU2 remedy has reached the construction completion milestone.

As EPA repeats numerous times in the Proposed Second FYR, GE completed Phase 2 of the dredging of the Hudson River on October 3, 2015 and backfilling was completed on November

[83] EPA FYR Guidance, at 4-13; *see also* U.S. Envtl. Prot. Agency, *Guidance Clarifying the Use of Protectiveness Determination for Comprehensive Environmental Response, Compensation and Liability Act Five Year Review* (Sept. 13, 2012) [hereinafter EPA Protectiveness Determination Guidance] *available at* https://semspub.epa.gov/work/HQ/174829.pdf. (Attachment L)

[84] *See* U.S. Envtl. Prot. Agency, *Memo Clarifying the Use of Protectiveness Determinations for Comprehensive Environmental Response, Compensation, and Liability Act Five-Year Reviews* at 1 (Sept. 13, 2012) *available at* https://semspub.epa.gov/work/HQ/174829.pdf.

[85] Operating remedial actions are those actions that are ongoing, but where cleanup levels have not yet been achieved. Such actions typically have remedial components requiring several years to reach cleanup levels (e.g., . . . monitored natural attenuation. . . .). 2017 FYR at 4-2.

[86] The OU2 Remedy is clearly not under construction as the physical construction of the remedy, i.e., the in-river dredging and habitat reconstruction, has been completed. *See* EPA FYR Guidance, at 4-2. Additionally, the OU2 Remedy is not a completed remedial action as the cleanup levels have not yet been achieved. *See id.*

[87] 2017 FYR at 14.

5, 2015. Complete demobilization of GE s sediment processing facility—a necessary component to the dredging project—occurred in December 2016, and all other support facilities were demobilized earlier in 2016. The habitat reconstruction portion of the remedial action was completed on August 8, 2016.[88] Therefore, it is plainly clear that the active mobilization component of the remedy—that is, the dredging project—is complete. Only the MNA period and long-term operation, maintenance and monitoring (OM&M) remain.

Throughout the Proposed Second FYR, EPA clearly marks a distinction between the construction phase of the remedy (i.e., dredging and habitat reconstruction) and the subsequent MNA period. For example, in Appendix 8, EPA states that [r]emedial construction included dredging, backfill placement, capping and habitat reconstruction. [89] EPA continues to discuss remedial construction in the past tense throughout this Appendix.[90] Furthermore, EPA states construction of the remedy was scheduled to commence in 2005 and to be conducted over a five-year period. This construction, in addition to monitored natural attenuation of the remaining PCBs, would lead to reductions of PCB concentrations. . . . [91] This statement clearly delineates construction as active dredging not including the subsequent MNA period.

The 2002 ROD is also quite clear with regard to the meaning of construction of the OU2 remedy. The construction period is commensurate with active mobilization for the dredging project. It ends long before the MNA period ends and the ultimate remedial goals are reached. The 2002 ROD discusses specific remedial construction parameters: The construction timeframes represent the estimated time required for mobilization, operation and demobilization of the remedial work, but *do not include the time required for long-term monitoring or OM&M*. [92] When discussing REM 3/10/Select, the remedy ultimately chosen for OU2, EPA states in the 2002 ROD: *After construction is completed*, this alternative relies of institutional controls and MNA until RAOs are achieved. [93] Finally, in the context of the potential for adverse environmental impacts during construction, the 2002 ROD defines construction as dredging and cap placement. [94]

EPA s FYR Guidance explicitly contemplates MNA remedies, like the OU2 remedy, where construction may be complete although cleanup levels have not yet been achieved. The EPA s FYR Guidance consistently defines remedial actions under construction as those where physical construction is not yet complete, as opposed to operating remedial actions, in which construction may be complete but cleanup levels have not yet been achieved.[95]

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[88] *Id.* at 20; *see also id.* App x 9.
[89] *Id.* App x 8 at 2-3.
[90] *Id.* (As a result, construction of the selected remedy *was executed* in accordance with . . .) (emphasis added).
[91] 2017 FYR at 30.
[92] 2002 ROD, at 56 (emphasis added)
[93] *Id.* at 60 (emphasis added). *See also id.* at 81 (After construction of the remedy is completed, the natural attenuation process would provide additional reductions.).
[94] 2002 ROD at 85.
[95] EPA FYR Guidance at 4-2 (MNA remedies cited as specific example); *see also* U.S. Envtl. Prot. Agency, *Close Out Procedures for National Priorities List Sites*, 1-2 (May 2011) [hereinafter EPA NPL Close Out Procedures] *available at* https://semspub.epa.gov/work/HQ/176076.pdf.

EPA does mention, in other areas of the Proposed Second FYR, that it will not consider the OU2 *remedy* to be complete until the natural attenuation component has also been completed and the RAOs have been achieved.[96] EPA is correct that remedial action completion will not occur until the MNA period has ended and all cleanup levels have been reached.[97] However, completion of the remedial action is an entirely distinct milestone from construction completion. [98] It is the completion of *construction* that is relevant in determining which protectiveness determinations are available to EPA, as discussed above.

Therefore, EPA cannot credibly argue that construction of the OU2 remedy is ongoing, and any assertions to that effect are arbitrary and capricious.

B. The Only Protectiveness Determinations Available for the OU2 Remedy are "Not Protective" and "Protectiveness Deferred."

EPA s determination that the remedy will be protective is inappropriate for the remedy according to the agency s FYR Guidance and Protectiveness Determination Guidance. According to the Protectiveness Determination Guidance, a will be protective determination is only appropriate when remedial construction activities are ongoing but the remedy is anticipated to be protective upon completion and no remedy implementation or performance issues have been identified.[99] Therefore, will be protective is not an available option for the OU2 remedy because, as explained above, construction of the remedy is complete—the physical and engineering components of the remedial action were completed in 2015 and 2016, respectively. Moreover, regardless of the status of the construction of the remedy, exposures are not currently under control and unacceptable risks are occurring, as explained further throughout these comments.

EPA admits in the Proposed Second FYR that the remedy is not currently protective of human health and the environment.[100] The human and ecological risks remain well above EPA s acceptable risk range,[101] and the institutional controls (fish consumption advisories) are not even close to completely effective in preventing actual exposures to these unacceptable risks.[102]

For all of these reasons, EPA s determination that the remedy will be protective is inconsistent with agency guidance and inappropriate for the OU2 remedy.[103] The only protectiveness

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[96] *See* 2017 FYR at 8, 20.
[97] *See* EPA NPL Close Out Procedures at 1-2, 2-4; *see also* EPA FYR Guidance at 4-2.
[98] EPA FYR Guidance at 4-2.
[99] EPA Protectiveness Determination Guidance at 3.
[100] 2017 FYR at 8.
[101] The risk-based RAO for the protection of human health is 0.05 mg/kg in fish fillet based on cancer and non-cancer hazard indices for the RME adult fish consumption rate of one half-pound meal per week. Current average fish tissue levels are many times that amount (1.3 mg/kg in 2016). 2017 FYR at 17.
[102] *See id.* at 62 (EPA acknowledged in the 2002 ROD that the consumption advisories are not fully effective in preventing or limiting fish consumption.).
[103] Indeed, as DEC pointed out in its December 2016 Recommendations to EPA for the Five Year Review Report for Hudson River PCBs Site, EPA s 2012 Five Year Review determination that the remedy will be protective may not have been in compliance with EPA guidance because EPA acknowledged in 2012 that human and ecological risks were not under control and that the risks remained unacceptable. *See* New York State Dep t of Envtl. Conservation, *Recommendations to EPA for the 'Five Year Review Report' for Hudson River PCBs Site* at 18-

determinations potentially available to EPA in the Proposed Second FYR are not protective and protectiveness deferred. As discussed throughout this comment, based on current data, EPA must find the remedy not protective.

VI. Current Data Indicate the Remedy is Not Protective of Human Health and the Environment.

A. Fish Tissue PCB Concentrations

1. The 2002 ROD Established Clear Interim Remedial Targets for Fish Tissue Concentrations.

The remedial objective in the 2002 ROD specific to fish tissue concentrations and human health is to [r]educe the cancer risks and non-cancer health hazards for people eating fish from the Hudson River by reducing the concentration of PCBs in fish. [104] In furtherance of this RAO, the 2002 ROD contains three target fish tissue concentrations for the cleanup: 0.4 mg/kg (safe to consume one half-pound fish meal every two months); 0.2 mg/kg (safe to consume one half-pound fish meal per month); and 0.05 mg/kg (safe to consume one half-pound meal every week). [105] The final target of 0.05 mg/kg is the remedial goal of the cleanup for the protection of human health. [106]

As discussed above, EPA evaluated five remedial alternatives in the 2002 ROD. In doing so, EPA stated that [t]he time to reach target PCB concentrations in fish was a *primary factor* in comparing remedial alternatives. [107] Alternatives that included active remediation (i.e., dredging or capping) met the interim and final targets more quickly than the No Action and MNA alternatives. [108] The table below, reproduced from the 2002 ROD, [109] illustrates the differences among the alternatives in meeting the targets.

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19 (Dec. 2016) [hereinafter DEC Report] *available at* http://www.dec.ny.gov/docs/fish_marine_pdf/hudsondredging5yr.pdf (Attachment M)

[104] 2002 ROD at 50.

[105] *Id.*

[106] *Id.*

[107] *Id.* at 66 (emphasis added).

[108] *Id.* at 66-67, 71-72.

[109] *Id.* at 73.

TABLE 1

Year to Reach Human Health Risk-based PCB Concentrations in Species-weighted Fish Fillet Upper Hudson River[1]

Alternative	Remediation Goal (0.05 mg/kg)	0.2 mg/kg	0.4 mg/kg
No Action[2]	> 2067	> 2067	> 2067
MNA[3]	> 2067	2035 to > 2067	2024 to > 2067
CAP-3/10/Select	> 2067	2024	2013
REM-3/10/Select	> 2067	2024	2012
REM-0/0/3	> 2067	2018	2010

1 Upper Hudson River average is weighted by river section length. River Section 1: 6.3 miles = 15.4%; River Sect ion 2: 5.1 miles = 12 .5%; and River Sect ion 3: 29 .5 miles = 72 .1%.

2 > 2067 means that the level will not be achieved within the model forecast period (*i.e.*, by 2067).

3 Higher value is upper bound.

The modeling for the 2002 ROD projected that the interim targets would be met, on a river section average basis, in 2012 and 2024, respectively.[110] Considering that the dredging was not completed until 2015, it stands to reason that the remedy would now be projected to meet the 0.4 mg/kg by 2017 (within two years of the completion of dredging) and the interim target of 0.2 mg/kg by 2029 (within 14 years of the completion of dredging). The interim targets in the 2002 ROD, which expected fish recovery to occur more slowly than model projections, would now allow for the 0.04 mg/kg target to be met by 2020 (within five years of the completion of dredging) and the 0.2 mg/kg target by 2031 (within 16 years of the completion of dredging).[111]

As discussed above, the time to reach the interim and final targets was a key component in EPA s selection of the remedy. Based on EPA s own rationale for selecting an active remedy, it is clear that delays of ten or more years in reaching the interim and final targets are not protective of human health.

2. Fish Tissue Concentrations Have Declined Since the Dredging Period, but There is Variation Among Species and Location.

It is undisputed that current fish tissue concentrations in the Upper Hudson River threaten both human health and the environment.[112] Although still hazardous, limited post-dredging data

[110] *See* 2002 ROD at Table 11-2.
[111] *See id.* at 103.
[112] *See, e.g.,* 2017 FYR at 71 (stating that as of the date of this five-year review, EPA recognizes the remedy at OU2 to be not yet protective of human health and the environment.); DEC Report at 28 (stating that the current fish

indicates that fish tissue concentrations in the Upper Hudson River have declined since the dredging period.[113] According to EPA, the 2016 data suggests that fish have begun to recover from dredging impacts and are generally back to pre-dredging levels.[114] Specifically, in 2009, prior to the start of dredging, the species weighted, wet weight average was 1.4 mg/kg.[115] In 2016, one year after the completion of dredging, the species weighted, wet weight average was 1.3 mg/kg.[116] EPA also claims that certain species are at or near the 0.4 mg/kg target in the Upper Hudson River, including largemouth bass and yellow perch.[117]

The Hudson River Foundation s (HRF) June 2017 Report, *An Independent Evaluation of the PCB Dredging Program on the Upper Hudson and Lower Hudson River*, also indicates that fish tissue concentrations in the Upper Hudson River have declined since the dredging period.[118] However, HRF found that those declines vary by location and by species. For example, in Thompson Island Pool in River Section 1, post-dredging TPCB concentrations in pumpkinseed and small forage fish were three to six times lower than observed pre-dredging levels.[119] Further downstream, the results are mixed.[120] At sampling locations in River Sections 2 and 3, concentrations in pumpkinseed were only two times lower than pre-dredging levels, and concentrations in small forage fish had declined very little, if at all.[121] In fact, at some locations, concentrations in small forage fish were *higher* than pre-dredging levels.[122]

This variability shows that fish tissue concentrations are closely tied to localized remedial activity and sediment contamination in the Upper Hudson River. HRF concluded that dredging in the Upper Hudson River was most effective in Thompson Island Pool, where a significant amount of sediment removal occurred.[123] The lack of response in small forage fish downstream reflects the linkages of TPCB concentrations in forage fish to localized sediment contamination levels and the limited areas that were targeted for dredging between Schuylerville and Waterford.[124] HRF s analysis should prompt EPA to reevaluate the relationship between fish tissue concentrations and localized sediments in the Upper Hudson River.

In short, while certain fish tissue concentrations have declined to some extent in the Upper Hudson River, the variation among species and locations requires additional investigation.

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tissue concentrations in the Upper Hudson River continue to result in exposures to both human and ecological receptors which are above EPA s acceptable risk range.); Hudson River Found., *An Independent Evaluation of the PCB Dredging Program on the Upper Hudson and Lower Hudson River*, 17 (June 2017) [hereinafter HRF Report] *available at* http://www.hudsonriver.org/download/2017-06-01Report-HRFDredgingProgramEvaluationFinal.pdf (stating that [b]ased on the 2016 post-dredging monitoring, TPCB concentrations in fish throughout the Upper and Lower Hudson remain above interim target levels and remediation goal specified in the ROD.).(Attachment N)

[113] *See* 2017 FYR Appx. 3 at 6-1, 6-2.

[114] 2017 FYR at 33.

[115] *Id.*

[116] *Id.*

[117] *Id,* at 45.

[118] *See generally* HRF Report.

[119] *Id.* at ii, 11.

[120] *Id.* at ii.

[121] *Id.* at ii, 11-12.

[122] *Id.* at 11.

[123] *Id.* at 17.

[124] *Id.*

3. Although There is a Significant Amount of Uncertainty and Variability Involved in Fish Tissue Recovery Rates, it is Clear That EPA Has Overstated the Recovery Rate.

At the August 9, 2017 Five Year Review Team Meeting, EPA stated that it continues to expect fish tissue recovery rates to be approximately 8% per year. However, as further detailed in an independent expert analysis, *Hudson River PCBs Site Proposed Second Five Year Review – Technical Review* prepared by S.S. Papadopulos & Associates, Inc. (SSPA), there is considerable uncertainty and variability in fish tissue recovery rates.[125] EPA s conversion of Aroclor data into homologue equivalent data is among the sources of uncertainty introduced.[126] Specifically, the method used for Aroclor data (M8082) is known to result in inaccuracy and increased uncertainty; the process of converting Aroclor data to homologue equivalent data involves a large amount of uncertainty that EPA failed to take into account; and extrapolating from one data set to another added even more uncertainty.[127] The uncertainty involved—and unaccounted for—in the data conversion process is particularly troubling, as EPA used those data to support an 8% recovery rate in fish tissue.[128]

EPA s inconsistent use of rib-out data is also problematic. EPA stated that rib-out data could be used [i]f the margin of error between rib-on and rib-off measurements [was] less than 20% of the average of lipid normalized PCB concentrations with a 95% confidence level .[129] Consequently, rib-out data were excluded from wet weight trends, as they differed by a factor of two or more, but the data were included in lipid normalized trends, as they differed by less than 20%.[130] However, the difference between individual paired rib-in and rib-out samples could be much greater—up to 75%.[131] Although there were significant discrepancies among some individual paired samples, EPA still utilized the suspect 2007 and 2008 data in calculating an 8% average recovery rate.[132]

In addition to these uncertainties, SSPA determined that fish tissue recovery rates are highly variable and misleading in the context of a protectiveness determination.[133] SSPA plotted several variations to demonstrate the uncertainty in EPA s anticipated recovery rate of 8%.[134] The variations on the use of rib-out data or Aroclor based measurement data show the potential for significantly different fish tissue recovery rates.[135] Those different rates, which ranged from 4% to 8%, could add decades onto recovery times.[136] For example, using the current average fish tissue concentration of 1.3 mg/kg, and assuming a 4% recovery rate, fish in the Upper Hudson River would not reach the 0.4 mg/kg five-year target for 27 years and the 0.2 mg/kg 16-year

[125] *See generally* S.S. Papadopulos & Associates, Inc., *Hudson River PCBs Site Proposed Second Five Year Review – Technical Review* (August 2017) (Attachment O) [hereinafter SSPA].
[126] SSPA at 7.
[127] *Id.*
[128] *See id.*
[129] *Id.* at 10.
[130] *Id.*
[131] *Id.*
[132] *See id.*
[133] *Id.* at 2.
[134] *See id.* at 12.
[135] *Id.* at 10-11.
[136] *See* SSPA at 18 (Table 3).

target for 43 years.[137] Even if EPA s unsupported and optimistic recovery rate of 8% actually occurs, it would take 15 years to get below five-year target.[138]

SSPA made several significant observations regarding the uncertainty and variability as it relates to EPA s anticipated 8% recovery rate:

> The average 8% rate is shown to be uncertain when it is not reproducible with slight variations in data inclusion. In fact, the variations consistently produced average rates of recovery lower than the rate calculated using EPA s approach. *EPA's approach therefore results in recovery rates that are biased high*; the EPA rate is at the fastest end of the range of recovery rates found by applying slight changes to method.
>
> Furthermore, □ the individual rates of recovery vary drastically by species and river section □. Importantly, the use of an average rate, while useful in representing the central tendency of recovery rates, *is deceptive in determining EPA's protectiveness statement for the Site*, because those fish populations with slow recovery rates or slightly increasing trends have half-lives several decades longer than the 8 years suggested by the 8% rate. These populations will continue to be an exposure risk for human health beyond the timeframe suggested by the 2017 Proposed FYR.[139]

In short, EPA s conclusion that the remedy will be protective based on an 8% recovery rate fails to account for significant uncertainty and variability. Furthermore, SSPA s analysis shows that EPA s anticipated recovery rate is overly optimistic, and that slower recovery rates will add decades to the 2002 ROD timeframes.

4. <u>The Cleanup is Not on Track to Meet the First Interim Target Within Five Years of Completion of Dredging.</u>

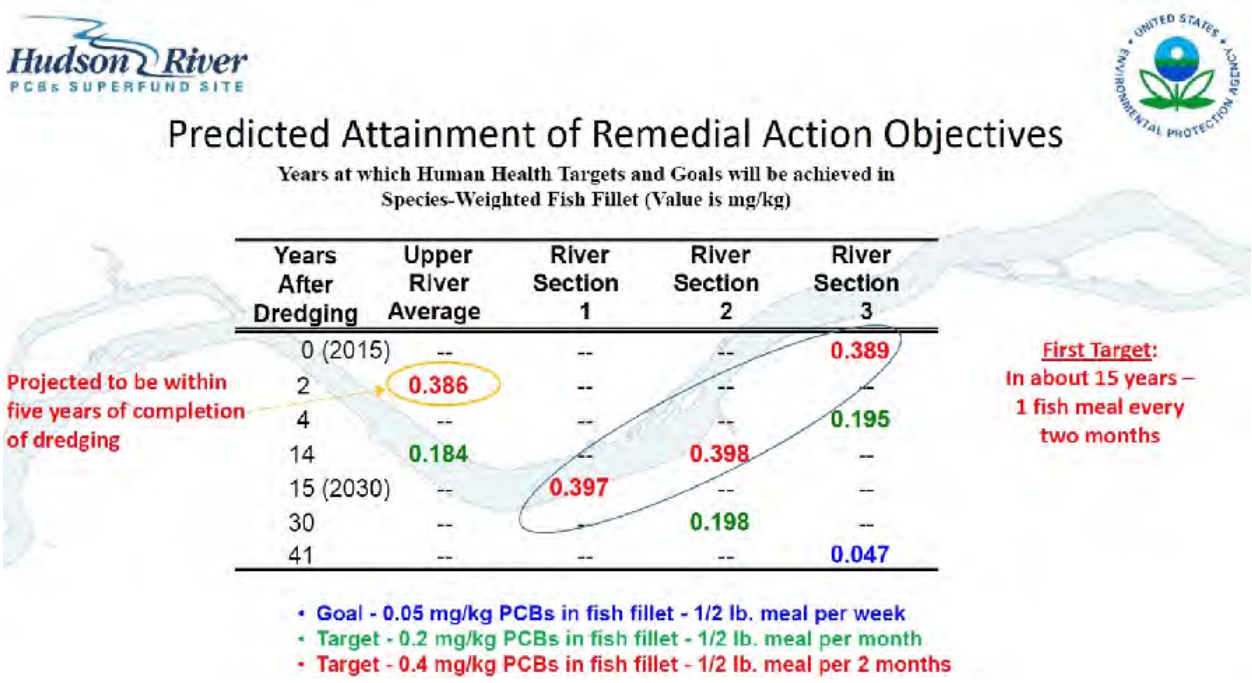
EPA recognizes that the remedy is not yet protective of human health and the environment in the Upper Hudson River, and points to the fact that the 2002 ROD did not anticipate the remedy to be protective by this time.[140] While it is true that the 2002 ROD did not expect the remedy to be protective two years after the completion of dredging, EPA ignores what the 2002 ROD *did* expect in the near term—that within five years of dredging, average fish tissue concentrations would be at or below 0.4 mg/kg. While more data is necessary to fully understand the timeline for reaching the interim and final targets, the cleanup will *not* meet the five-year target of 0.4 mg/kg for more than 15 years after the completion of dredging if its current expectations about recovery rate are correct.

According to EPA, the 2016 data indicates that the species weighted, wet weight fish tissue concentration in the Upper Hudson River is 1.3 mg/kg. With fish tissue concentrations at that

[137] *Id.*
[138] *Id.*
[139] *Id.* at 12-13 (emphases added).
[140] 2017 FYR at 8 (stating that [a]s expected in the [ROD], average PCB concentrations in fish in the Upper Hudson are declining but have not yet reached protective levels.).

level, concentrations would have to decline at a very high rate of over 25% in order to meet the 0.4 mg/kg goal within five years of the completion of dredging, or by 2020, which is all but impossible. Even assuming an 8% decay rate, which is optimistic, the Upper Hudson Average would miss the 2020 target by more than ten years. As discussed *supra*, EPA has already determined that delays of ten years or more in meeting the interim and final targets are unacceptable. Because the data show that the recovery will occur at a rate that the 2002 ROD found was not protective in the context of other remedial alternatives, EPA has no basis whatsoever to find that the remedy will be protective for the Upper Hudson.

At the July 20, 2017 Community Advisory Group meeting, EPA itself admitted that the cleanup will not meet the five-year target on an Upper Hudson Average basis.[141] However, EPA has not made that clear in the Proposed Second FYR or in its public presentations to date. Rather, EPA created a PowerPoint slide based on Table 11-2 from the 2002 ROD, which shows the specific years in which the cleanup was expected to meet the interim and final remedial goals on an Upper Hudson Average and River Section basis.[142] The slide from EPA s presentation to the CAG is reproduced below.



EPA s use of this slide is misleading. The information is merely a recitation of the 2002 ROD modeling, but EPA uses it to suggest that the First Target is 15 years from the date of completion of dredging, not five years, as is actually the case. Moreover, the slide continues to suggest that the Upper Hudson Average will meet the 0.4 mg/kg target within five years of dredging (potentially even within two years), even though it will almost certainly miss that target. Finally, if the Upper Hudson Average will not meet the 0.4 mg/kg target until 2031 (as is

[141] EPA walked back this admission at the August 9, 2017 Public Information Meeting in NYC, suggesting that the target may not be met for seven years or more, and again at the August 16, 2017 Five Year Review Team meeting, insisting that it did not know whether the target would be met within five years.
[142] *See* 2002 ROD Table 11-2 at p.3 of 4.

the case with current conditions, assuming an 8% decay rate) one or more river sections will lag behind, meaning that in about 15 years, it is very unlikely that it will be safe to eat one fish meal every two months from each of the river sections.[143]

Although there is a significant amount of uncertainty and variability regarding decay rates, EPA must recognize that the cleanup will not meet the five-year target as set out in the 2002 ROD. In fact, it is likely that meeting the 0.4 mg/kg interim target will occur at a rate that EPA already determined was not protective for the other remedial alternatives considered in the 2002 ROD. Therefore, EPA should find that the remedy is a not protective and require GE to undertake for further investigation and remediation to get the cleanup back on track.

5. EPA Lacks Necessary Information to Make Long Term Predictions About Whether the Remedy Will Be Protective in the Future.

EPA asserts that it does not have sufficient data to predict future trends in fish tissue concentrations.[144] In fact, EPA repeatedly states that it needs *at least* eight more years of data to draw statistically based conclusion about trends with a high degree of confidence. [145] However, despite lacking key information necessary to evaluate the effectiveness of the cleanup, EPA insists that declines in fish tissue concentrations are generally consistent with ROD predictions and that the system is responding as anticipated. [146] In fact, the absolute level of PCBs in fish in the Upper Hudson is much higher than EPA anticipated at two years post-dredging, and the rates of decline observed are lower than EPA predicted. EPA cannot have it both ways. It should either make no prediction about the future if it thinks uncertainty is too high, or it should find that the first target will not be met if its current expectation about the rate of decline in fish tissue concentrations is correct. Both approaches lead to the same finding—that the remedy is not protective.

Essentially, EPA contends that the cleanup is consistent with the 2002 ROD as long as it will be protective at some unknown point in the future, following an undefined period of monitored natural attenuation.[147] EPA either cannot or will not provide specific timeframes in the Proposed Second FYR for when it expects the cleanup to meet the 2002 ROD targets.[148] Instead, EPA merely states that it does not expect to meet the remedial goal of 0.05 mg/kg for decades, and that it expects to meet the interim targets of 0.4 and 0.2 much sooner. [149]

EPA s statements in the Proposed Second FYR regarding the 2002 ROD expectations are inconsistent with its statements at recent public events. For example, in the Proposed Second

[143] *See id.* (showing that River Sections 1 and 2 were not expected to meet the 0.4 mg/kg target until 12 to 13 years after the Upper River Average).
[144] *See e.g.*, 2017 FYR at 5 (stating that [f]ish, sediment, and water data at this early time are not sufficient to identify post-dredging trends with a high degree of confidence.).
[145] *Id.* at 6; *see also id.* at 33, 69, 70; *id.* App x 3 at 1-2; *id.* App x 3 at 6-2, 6-3.
[146] 2017 FYR at 58.
[147] *See id.* at 8 (stating that EPA expects that continued natural attenuation following the completion of dredging will achieve the long term remediation goal . . . . As EPA indicated in the [ROD], EPA believes it likely that improvement will occur gradually over several decades at least.); *see also id*. at 24.
[148] *See* 2017 FYR at 58 (stating that [a]s additional post dredging data are collected, EPA will be able to further assess the specific timeframes to achieve the 0.2 mg/kg and 0.4 mg/kg target levels.).
[149] 2017 FYR at 33.

FYR, EPA asserts that declines in tissue concentrations consistent with 2002 ROD predictions. Although further monitoring will be required to verify that RAOs are being achieved, the lines of evidence to this point indicate that the system is responding as anticipated and that target levels will be achieved within the timeframes predicted in the ROD. [150] However, as discussed above, EPA admitted at the July 20, 2017 CAG meeting that the cleanup will miss the five-year target.

In sum, fish tissue concentrations throughout the Hudson River Superfund Site continue to pose a threat to human health and the environment. In the Upper Hudson River, evidence suggests that declines in fish tissue concentrations associated with dredging vary by species and location. Furthermore, there is considerable uncertainty and variability with regard to fish tissue recovery rates. However, it is nearly certain that the cleanup will miss the five-year interim target of 0.4 mg/kg. Finally, EPA admits that it cannot predict when fish tissue concentrations will meet the 2002 ROD targets with any confidence. Therefore, EPA cannot support its finding that the cleanup will be protective of human health.

6. The Lack of Response in Fish Tissue Concentrations in the Lower Hudson River Demonstrates the Need for a Full Remedial Investigation and Feasibility Study.

EPA expressly admits that fish tissue concentrations in the Lower Hudson River are not responding as anticipated. EPA recognizes that it is clear that [t]he rate of decline of fish tissue PCB concentrations generally decreases with distance downstream. As a result, there is a decrease in the correlation between fish PCB concentrations in the Upper Hudson River and Lower Hudson River with distance downstream. [151] EPA interprets the data to show that the Lower Hudson River recovers more slowly than the Upper Hudson under MNA. [152] In fact, the data that EPA relies on in the Proposed Second FYR show that decay rates during the MNA period from Poughkeepsie/Kingston downstream are not statistically different from zero. [153]

TABLE 2

Decay Rates During the MNA Period According to EPA's Proposed Second FYR[154]

Monitoring Location	Wet-Weight	Lipid Normalized
Upper Hudson	16%	8%
Albany/Troy (RM152)	16%	10%
Catskill (RM113)	11%	3%
Poughkeepsie/Kingston (RM90)	8%	~0%
Newburgh (RM50)	1%	~0%

Rates of decay in the Lower Hudson River also vary by species. While EPA claims that several species are at or near the 0.4 mg/kg or 0.2 mg/kg targets in the Lower Hudson River, and that

[150] 2017 FYR App x 3 at 7-2.
[151] 2017 FYR at 6, 33, 57, 70; *id.* App x 3 at 7-1; *id.* App x 3 at 4-5.
[152] *Id.* App x 3 at 4-7.
[153] 2017 FYR App x 3 at 7-1.
[154] 2017 FYR App x 3 at 4-5, 4-6

yellow perch are at the 0.05 mg/kg target, other species are recovering at a slower rate. In fact, EPA admits that decay rate estimates are variable across species and locations, with the brown bullhead demonstrating the slowest recovery . . . . [155]

EPA attributes the differences between the Upper Hudson and Lower Hudson to a number of potential factors, including the fate and transport of PCBs in the Lower Hudson River.[156] It is likely true that fate and transport in the tidal Lower Hudson River differs from the Upper Hudson River. According to HRF, the complexity of sediment transport in the Lower Hudson contributes to the lack of response in fish to the upriver dredging.[157] As HRF explains, [t]he continuous interaction of the overlying water with sediments (through setting, resuspension, and pore water exchange) and the large capacity of the sediments to sorb PCBs work together to dampen the PCB responses downstream and to greatly extend PCB response times to changes in Upper Hudson PCB loads. [158] Significantly, HRF concludes that the Lower Hudson River appear[s] to be responding very slowly to changes in PCB inputs from the Upper Hudson. [159]

DEC has also expressed concerns about the relationship between localized sediments and fish tissue concentrations in the Lower Hudson. Specifically, DEC asserts that the degree to which local sediments influence fish PCB concentrations is greater than thought at the time of remedy selection. As a result, there will be little additional improvement in fish PCB concentrations in the lower Hudson, particularly south of Albany, as a result of the dredging. [160] Ultimately, the issues raised by HRF and DEC regarding fate and transport of PCBs in the Lower Hudson River and the degree to which localized sediments impact fish tissue concentrations support the need for a full investigation.

EPA also speculates that other sources of PCB contamination may be responsible for the slow recovery in the Lower Hudson River. Although other sources of PCBs do exist—namely ARCO in Hastings and BICC Cables in Yonkers—EPA has stated in public meetings that it is undisputed that GE is the primary contributor. As such, the mere presence of other sources of PCB contamination should not deter EPA from ordering GE to undertake a full remedial investigation and feasibility study in the Lower Hudson River.

Despite the slow response thus far, EPA still maintains that the PCB load reduction from the Upper Hudson River will benefit the Lower Hudson River.[161] However, it is not clear how quickly that will occur, if at all. HRF anticipates that it would take a decade or more to see appreciable change in PCB water column, sediment, and fish concentrations at many locations in the Lower Hudson. [162] DEC takes an even less optimistic view:

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[155] *Id.* at 4-11; *see also, id*. at 4-12 (stating that declines in PCB concentrations are occurring more rapidly in the Upper Hudson and less rapidly in the Lower Hudson with estimate rates not statistically different from zero for several species at RM113, RM90 and RM50).
[156] 2017 FYR at 57.
[157] *Id.* at i.
[158] *Id.* at 16.
[159] *Id.* at 17.
[160] DEC Report at 37.
[161] 2017 FYR at 57.
[162] HRF Report at iii.

> Insufficient data are available in the lower Hudson to answer the question as to the magnitude of the delay in reaching the Remediation Goal of 0.05 ppm PCB in fish, However, given the limited impact of the remedy to date on fish in the Lower Hudson below Albany it is not anticipated that there will be further improvements in fish PCB in this area as a result of the dredging. Currently available fish PCB concentrations indicate ongoing exposures present unacceptable human health and ecological risk.[163]

DEC concludes that the anticipated reductions in fish PCB concentrations in the lower Hudson, as a result of the remedial work in the upper Hudson, will likely not occur as anticipated in the ROD.[164]

Furthermore, the National Oceanic and Atmospheric Administration (NOAA) recently published a peer-reviewed study that used model emulation to predict fish tissue concentrations in the Lower Hudson River based on post-ROD data.[165] The data that NOAA relied on showed that surface sediment concentrations in the Upper Hudson River were higher than expected.[166] In addition, NOAA determined that a 3% sediment recovery rate was more in line with the data than the 8% recovery rate used in the modeling for the 2002 ROD.[167] By considering these different inputs, NOAA s analysis indicates that EPA may have greatly underestimated the timeframes for fish recovery in the Lower Hudson River, and that it could take decades longer than anticipated to meet the interim targets south of the Troy Dam.[168] To illustrate this point, NOAA includes specific projections for white perch at RM 152.[169] Using updated sediment concentrations and assuming a 3% decay rate, white perch would not meet the 0.4 mg/kg and 0.2 mg/kg targets for 44 and 67 years, respectively.[170]

Even with skepticism and disagreement from NOAA, DEC, and HRF, EPA maintains that the slow response in the Lower Hudson is consistent with the 2002 ROD expectations.[171] This is despite its finding that the model used for the 2002 ROD underpredicted fish tissue levels in the Lower Hudson.[172] Moreover, EPA makes several vague and seemingly inconsistent statements about the remedy. For example, EPA contends that the 2002 ROD did not predict significant impacts from dredging, but nevertheless predicted [s]ome improvements as a

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[163] DEC Report at 26.
[164] *Id.* at 28.
[165] *See generally* L. Jay Field, et al., *Re-visiting projections of PCBs in Lower Hudson River fish using model emulation* 489, 493 (July 1, 2016) [hereinafter NOAA Study] *available at* http://www.sciencedirect.com/science/article/pii/S0048969716302820; Nat l Oceanic and Atmospheric Admin., *Powerpoint: Re-visiting Model Projections of Lower Hudson River Fish PCBs* (Aug. 15, 2015) [hereinafter September 15, 2016 NOAA Powerpoint] *available at* https://casedocuments.darrp.noaa.gov/northeast/hudson/pdf/CSF2015_AUG20_LHR_Fish_final_dist.pdf. (Attachment P)
[166] NOAA Study at 493.
[167] *See id.* at 497.
[168] *Id.* at 499.
[169] *Id.* at 495-97.
[170] *Id.* at 497.
[171] *See* 2017 FYR App x 3 at 7-2
[172] *See* NOAA Study at 499.

result of the remedy.[173] Unsurprisingly, EPA claims that [b]oth predictions are consistent with observations. [174] However, EPA also admits that observations support a lack of significant response between Upper Hudson processes, e.g., dredging releases, and Lower Hudson impacts. [175] Regardless of EPA s current characterization of the 2002 ROD expectations, the lack of any significant response suggests that EPA was incorrect in assuming that the Lower Hudson River would meet the final remedial goal of 0.05 mg/kg in the same timeframe as River Section 3.[176]

In sum, the Lower Hudson River is responding very slowly to the cleanup, if at all. Evidence suggests that there is a disconnect between the remedial activities in the Upper Hudson River and the response in the Lower Hudson River. Therefore, EPA should require GE to conduct a full remedial investigation and feasibility study to address the ongoing PCB contamination in the Lower Hudson River.

B. Sediment Contamination

The 2002 ROD acknowledged that [o]nce introduced to the river, PCBs adhere to the sediments. Physical, chemical, and biological release mechanisms allow PCBs in the sediment to be available for redistribution and to be a source of PCB contamination to the water column. Sediments would continue to release contamination to the water column and to biota, through aquatic and benthic food chains, *unless they are managed or remediated*. [177] Consequently, to address the threat to human health and the environment posed by PCB contaminated sediments, the 2002 ROD included the following remediation objectives: [r]educe PCB levels in sediments in order to reduce PCB concentrations in river (surface) water that are above applicable or relevant and appropriate requirements, [178] and [r]educe the inventory (mass) of PCBs in sediments that are or may be bioavailable in order to ultimately reduce PCB levels in fish and the associated risks to human health and the environment.[179] So, the mass of PCBs that may become bioavailable are closely related to the concentration of PCBs in surface sediments. [180] Consequently, the remedy focused on removing PCBs from targeted (dredged) areas, with focus on surface sediment concentrations as the main mechanism through which PCB concentrations in fish would be influenced.

The 2002 ROD thus required [r]emoval of all PCB-contaminated sediments within areas targeted for remediation [namely, hot spots] with an anticipated residual of approximately 1 mg/kg Tri+ PCBs, as well as the [u]se of environmental dredging techniques to minimize and control resuspension of sediments during dredging. [181] To this end, the 2002 ROD estimated [that the] volume of sediments to be removed is 2.65 million cubic yards, which is estimated to

[173] 2017 FYR App x 3 at 7-2.
[174] *Id.*
[175] *Id*.
[176] *See* 2002 ROD at 103.
[177] *Id.* at 15 (emphasis added).
[178] *Id.* at 17.
[179] *Id.* at 18.
[180] 2017 FYR at 48.
[181] *Id.* at ii-iii (emphasis added).

contain 70,000 kg (about 150,000 lbs) of total PCBs. [182] This remediation target for the chosen alternative, REM 3/10/Select, was broken down by River Section in the 2002 ROD Table 13-1, with estimated removals of 36,000 kg total PCBs for River Section 1; 24,300 kg total PCBs for River Section 2; and 9,500 kg PCBs total for River Section 3.[183]

During Phase 1 of the implementation of the selected remedy, REM 3/10/Select, EPA discovered that it had underestimate[d] the depth of contamination during the original remedial design, [184] and consequently, it ordered additional sediment sampling (coring) to inform Phase 2 of dredging.[185] In addition to underestimating the depth and mass of PCB contamination, EPA also underestimated the concentration of PCBs in surface sediments.[186] Yet, despite acknowledging (1) that the 2002 ROD had underestimated the concentration, depth, and mass of PCB contamination in the sediment—*and* left more behind; (2) that operational adjustments meant dredging was not implemented in the manner anticipated in the 2002 ROD; and (3) the fact that dredging began later than anticipated in the ROD, [187] the Proposed Second FYR concludes that EPA s remedy for the sediments was implemented successfully and within expectations described in the ROD. [188]

1. Average Surface Sediment PCB Concentrations After Dredging Are Two to Three Times Higher Than Anticipated in the 2002 ROD, Undermining EPA s Will Be Protective Determination.

Surface sediment concentrations are the primary source of PCBs bioavailable to fish species, and are closely linked to fish tissue concentrations.[189] For this reason, reducing surface sediment concentrations of PCBs is essential to the RAO of reducing PCB concentrations in fish.[190] The 2002 ROD anticipated that the remedy (dredging followed by MNA) would reduce surface sediment Tri+ PCB concentrations from an average of 4.6 mg/kg for River Section 1; 2.26 mg/kg for River Section 2; and 0.53 mg/kg for River Section 3; to an average of 0.96 mg/kg in River Section 1; 0.08 mg/kg in River Section 2, and 0.51 mg/kg in River Section 3.[191]

In the First FYR, EPA used the surface sediment data collected during the Sediment Sampling and Analysis Program (SSAP) survey conducted from 2002-2005 as a pre-dredging baseline to re-estimate expected reductions in average Tri+ PCBs concentrations from implementing the dredging remedy.[192] The SSAP re-estimate with 2003 pre-dredging data found that, in actuality, two to three times *higher* Tri+ PCB concentrations existed in surface sediment.[193] Thus, as noted

[182] *Id.* at 60; *see also id.* at Table 10-1 (estimating total PCB mass removed to be 70,000 kg).
[183] *Id.* at Table 13-1.
[184] 2017 FYR at 4.
[185] *Id.* at 47.
[186] 2017 FYR App x 4 at Table A4-5.
[187] 2017 FYR at 30.
[188] *Id.* at 3.
[189] 2017 FYR at 48.
[190] *Id.*
[191] 2012 FYR App x A at Table 1 (see note 4).
[192] 2017 FYR at 49.
[193] 2012 FYR App x A at Table 1.

by NOAA, the higher than expected pre-dredging surface sediment PCB concentrations likely extend the time required to reach recovery thresholds in fish tissue.[194]

Utilizing incorrect inputs for pre-dredging surface sediment PCB concentrations also impacts EPA s model predictions for post-dredging concentrations. Similarly, the EPA s 2002 ROD prediction for post-dredging PCB surface sediment concentrations *underestimates* the PCB concentration in surface sediment after dredging.[195] After correcting this input assumption, NOAA found that post-dredging residual PCB surface sediment concentrations were three to five times higher than predicted in the 2002 ROD, with even greater differences for River Sections 2 and 3.[196] Even utilizing EPA s own projected 8% decay rate (which NOAA and others dispute), recovery would be delayed by 25 years.[197] Comparing the EPA s 2002 ROD predictions with observed post-dredging PCB concentrations in surface sediment, NOAA found this prediction borne out, as a three to five times higher PCB concentration was actually observed in surface sediment after dredging.[198]

EPA admitted in the First FYR that after re-estimating Tri+ PCB concentrations with SSAP data, higher concentrations of PCBs would remain in river surface sediment after dredging than anticipated by the 2002 ROD.[199] However, the agency does not take the logical next step in evaluating whether this will impact its current will be protective determination by undermining the assumptions held in the 2002 ROD. In the Proposed Second FYR Appendix 4, EPA describes average Tri+ PCB surface sediment concentrations remaining after dredging only in *percentages*.[200]

Rather than compare the actual observed reductions in PCB residual concentrations in surface sediment with the 2002 ROD expectations and targets, EPA compares this data with the less-stringent interim expectations described in the First FYR,[201] without any justification of why this is correct.

Table 3 shows EPA s comparison of the percentages of PCBs remaining by concentration in surface sediment after dredging between the First FYR re-estimate and the Proposed Second FYR observed data. Looking at the actual values of the residual PCB concentrations—rather than percentages—it becomes evident that the remedy as implemented does not conform with 2002 ROD expectations or meet remediation goals judged necessary to achieve protection of human health and the environment in River Sections 2 and 3. To elucidate this discrepancy, Table 3

[194] September 15, 2016 NOAA Powerpoint at 9.
[195] *Id.* at 21.
[196] *Id.*
[197] *Id.* at 22.
[198] September 15, 2016 NOAA Powerpoint at 9.
[199] 2012 FYR App x A at 54. *E.g. id.* (The notable difference between the ROD-anticipated reduction and that predicted from the remedial design occurs in RS2 [River Section 2]. The reduction anticipated by the ROD (64 percent) is about twice as much of an improvement for RS2 as predicted from the remedial design (36 percent). *This indicates that it will take RS2 longer to reach its ultimate remedial goals than the forecast in the ROD*. . . . Thus based on the discussion above, achievement of the various remedial goals for RS2 may lag those anticipated by the ROD by about 10 years.) (emphasis added).
[200] *See* 2017 FYR at 50; *id.* App x 4 at 5-2.
[201] 2017 FYR App x 4 at 5-2.

compares the 2002 ROD expectations with the 2012 re-estimation and the 2017 actual observed surface sediment data.

TABLE 3

River Section (area weighted average)	**2002 ROD expectation**			**2012 FYR re-estimate (using 2003 pre-dredge data from SSAP survey)**			**2016 actual observed (from 2017 FYR)**	
	pre-dredge mg/kg [202]	post-dredge mg/kg [203]	% reduction	pre-dredge mg/kg [204]	post-dredge mg/kg [205]	% reduction [206]	post-dredge mg/kg [207]	% reduction [208]
River Section 1	4.6	0.96	79%	14.2	1.9	87%	0.77	96%
River Section 2	2.26	0.80	66%	11	7.1	36%	1.34	88%
River Section 3	0.53	0.51	4%	3.3	3.1	5.1%	0.83	80%

Looking at actual concentrations of residual PCBs in surface sediment, the remedy as implemented has *not* achieved the residual surface sediment PCB concentration goals in River Sections 2 and 3. This is because the pre-dredging PCB concentrations were much higher than anticipated. Because the 2002 ROD model predicting the rate of decay of residual PCB contamination has not been updated using the higher surface sediment concentration levels (the 2012 re-estimate), the model probably over predicts the rate of decay. EPA s will be protective determination is thus premised on inaccurate input assumptions, and cannot support the protectiveness determination.

2. <u>The Proposed Second FYR Misleadingly Compares Percentages of PCBs Removed with the 2002 ROD Percentage Reduction Goals in Concluding that the Goals Are Being Met, Despite Acknowledging that Up to Nearly Two and A Half Times More PCBs Were Found in Surface Sediment Than Expected.</u>

The Proposed Second FYR puts the remedy in the best possible light by stating that 72% of the overall PCB mass from the Upper Hudson River was removed by the dredging, which exceeds the 65% reduction assumed in the ROD. [209] This statement ignores the fact that more than two

[202] Data points for this column taken from 2012 FYR App x A at Table 1.
[203] *Id.*
[204] Data points for this column taken from 2017 FYR at 50.
[205] *Id.*
[206] *Id.*
[207] Data points for this column taken from 2017 FYR App x 4 at Table A4-5
[208] Data points for this column taken from 2017 FYR at 50. *See also id.* App x 4 at Table A4-5.
[209] *Id.* at 32.

times more PCBs were found in the areas targeted for dredging than originally anticipated in the 2002 ROD.[210]

A less rosy picture is painted by examining the actual values of the total mass of PCBs removed by dredging—rather than percentages. Using this approach, it is evident that the remedy as implemented does not conform with 2002 ROD expectations, as shown in Table 4.[211]

TABLE 4	**ROD expectations (2002 ROD)**	**actual observed (2017 FYR)**
percentage of PCBs to be removed by dredging	65%	(72%)
mass of PCBs existing in dredging area	107,400 kg[212]	216,333 kg
mass of PCBs removed by dredging	69,800 kg[213]	155,760 kg[214]
residual PCBs after dredging	37,600 kg	60,573 kg

The 2002 ROD anticipated that 37,600 kg of total PCBs would remain after dredging had been completed. However, the Proposed Second FYR data indicates that 60,573 kg of total PCBs remained in the targeted river sections after dredging. This means that *much more PCBs remain* in the dredged areas than was assumed in the 2002 ROD.

Despite acknowledging that more PCBs remain than anticipated by the 2002 ROD—and, thus, that the remedy has not been implemented as described—EPA concluded that, after MNA, it will be protective of human health and the environment.[215] This conclusion ignores that the conditions the 2002 ROD predicted would exist after dredging, and upon which it prefaced its MNA determinations, were not achieved by the dredging, since at least two times more PCBs remain after dredging in the targeted river sections. With two times more PCBs remaining in the sediment in some areas, the 2002 ROD predictions about natural attenuation are significantly undermined, and cannot reasonably form the basis for EPA s will be protective determination.

The failure of the remedy to reduce the amount of residual PCBs after dredging to conform with the 2002 ROD expectations renders EPA s protectiveness finding arbitrary. EPA does not evaluate whether the fact that between two and nearly two and a half times more PCBs remain in the riverine environment will have an impact on the MNA process, potentially slowing natural recovery dramatically. The 2002 ROD itself rejected the alternatives employing MNA without

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[210] *Cf.* 2017 FYR App x 8 at 2-4 (the PCB mass removed by dredging . . . was 2.3 times the prospective ROD estimate); *see also* 2017 FYR at 31 (underestimates of the depth of contamination [were]primarily caused by wood debris that interfered with sediment sampling); *id.* at 4 (Total PCB and Tri+ PCB mass removed were greater than planned, due to underestimates of the depth of contamination during the original remedial design. PCB mass in non-dredged areas is also greater than estimated in the 2002 ROD, although to a lesser extent than within the dredged areas.).

[211] *See also* NOAA Study at 495, Figure 5 (comparing, for each River Section, the extent to which more PCBs were present than anticipated prior to dredging and the higher-than-expected concentration post-dredging).

[212] *See* 2002 ROD at 21 (listing total PCB mass in the sediments in River Sections 1, 2 and 3).

[213] *See* 2017 FYR App x 2 at Table A2-3.

[214] *Id.*

[215] 2017 FYR at 24.

dredging in favor of a remedy that included dredging and subsequent MNA precisely because the slow remediation timeline presented was inadequate to protect human health and the environment.[216] Such slowing here, by leaving behind at least two times more PCB mass than anticipated, and three to five times more PCBs in surface sediments, similarly fails to protect human health and the environment.

DEC echoed this criticism of the Proposed Second FYR, explaining that the fact that sediment concentrations higher than anticipated will remain after dredging[] indicates that the targeted fish PCB concentrations will not be reached in the time frames identified in the ROD. [217] Both the greater-than-expected PCBs remaining after dredging and the operational delays in implementing the dredging contradict[] the basis upon which EPA selected the remedy, namely, that a delay in abating the uncontrolled ecological and human health exposures was not acceptable. [218]

Thus, not only does the Proposed Second FYR s comparison of the targeted river sections 2017 status with the 2002 ROD goals via percentages mislead the public with regard to the effectiveness of the dredging, but it also raises serious questions as to the accuracy of the EPA s finding that the remedy as implemented will be protective of human health and the environment.[219] Furthermore, DEC calls for the site conceptual model [] to be updated to take into account the data gathered [] since the ROD was issued that showed that higher surface sediment PCB concentrations would be left behind than anticipated . . . . [220] Similarly, NOAA calls for the model to be updated both with the increased PCB contamination input as well as the corrected rate of decay.[221]

Accordingly, EPA must require additional dredging to remove the remaining PCBs in accordance with the 2002 ROD expectations, or reevaluate the anticipated MNA rate to account for the two times more PCBs remaining in the environment, taking into account the inadequacy of slow MNA-only timelines rejected in the 2002 ROD.

C. Water Column

To reduce environmental and human health risks, the 2002 ROD proposed two remedial action objectives related to PCB concentrations in the Upper Hudson River water column: (1) to [r]educe PCB levels in sediments in order to reduce PCB concentrations in river (surface) water that are above surface water ARARs [applicable or relevant and appropriate requirements] ; and (2) to [m]inimize the long-term downstream transport of PCBs in the river. [222] PCBs that are transported downstream in the water column are available to biota, contributing to the risk to

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[216] 2002 ROD at 73. *See also* DEC Report at 1 (EPA selected this remedy primarily based upon the time it would take to achieve targeted fish PCB concentrations after dredging. . . . Specifically, EPA stated in the ROD that a delay of ten years in dredging and 0.2 mg/kg within 16 years of the completion of dredging was unacceptable. This ten year delay was used as a basis for rejecting the Monitored Natural Attenuation (MNA) remedial alternative. . . . EPA conclude the dredging was needed to accelerate the time it would take to reach the remedial targets for fish flesh in order to quickly reduce human health and ecological risk compared other alternatives that were evaluated.).
[217] DEC Report at 2-3.
[218] *Id.* at 19.
[219] 2017 FYR at 24.
[220] DEC Report at 44.
[221] NOAA Study at 499.
[222] 2002 ROD at 51-52.

human health and the environment from the Site s PCB contamination. Downstream transport also moves PCBs from highly contaminated areas to lesser contaminated or clean areas, and from the Upper Hudson to the Lower Hudson.[223]

1. The 2002 ROD Predictions Were Optimistic for the Lower Hudson.

At the time the Proposed Second FYR was issued, EPA had compiled pre-dredging period water quality data for 17 years (1991-2008), dredging period data for six years (2009-2015), and post-dredging data for one year (2016). Despite having collected over 25 years of Hudson River PCB data, some of EPA s critical modeling failed to predict trends, concentrations, decay and volatilization rates of PCBs.[224] Furthermore, the model used to analyze Lower Hudson River data systematically under-predicted Tri+ PCB concentrations at Poughkeepsie.[225] Two of EPA s models disagreed on rates of decay and neither was accurate: decay was slower than the HUDTOX MNA model predicted at Stillwater and Waterford and faster than the 2002 ROD MNA model predicted at Thompson Island Dam and Schuylerville.[226]

EPA claimed that the 2002 ROD forecast rate of natural attenuation (9.6% - 10.6%) during the pre-dredging period (2004-2008) was comparable to the observed decay rates at the four Upper Hudson River stations (4.5% - 13.1%).[227] Although Tri+ PCB concentrations at the Albany station—the uppermost river segment of the two Lower Hudson stations—were in close agreement with the four Upper Hudson stations, the modeling used to predict Tri+ PCB concentrations at Poughkeepsie under-predicted concentrations for the pre-dredging period from 2004 to 2008.[228]

EPA has conceded that the effects of PCB load reduction from Upper Hudson to Lower Hudson are unknown. Additional years of monitoring data will be required to sufficiently evaluate MNA trends following completion of dredging activities. [229]

2. Load Reductions to the Lower Hudson Are Not as Large As Expected.

As HRF observes, in both the pre-dredging and post-dredging periods, Tri+ PCB concentrations decreased with increasing flow for river flows less than approximately 13,000 cfs (or 1.6 times the long-term mean river flow at Waterford) and [f]or river flows greater than 13,000 cfs, Tri+ PCB concentrations increased with increasing flows. [230] HRF indicates that the second result is expected, and is associated with increased flow-induced erosion of the streambed and the accompanying increase in suspended sediment loads (and particulate phase PCB transport) during the higher flows. [231] These findings also indicate that resuspension of

[223] *Id.*
[224] *See, e.g.*, 2017 FYR at 2-1 (The modeling analysis yielded much lower estimated concentrations of volatilized concentrations in the air compared to empirical data.).
[225] *Id.* App x 4 at 4-5, Fig. A1-4.
[226] *Id.* at 4-4.
[227] *Id.*
[228] *Id.* at 4-5.
[229] *Id.* at 2-6.
[230] HRF Report at ii, 7.
[231] *Id*. at 7.

localized sediments, rather than upstream inputs, is driving Tri+ PCB concentrations during high flow events.[232]

Modeling indicated a significant reduction in Tri+ PCB loadings during high flow events and minimal reduction during low flow events (*see* HRF Report Figure 9, reproduced below).[233] Even less reduction was predicted when based on the actual flow record from 2004 to 2008. Under this scenario, Tri+ PCB loads during the pre-dredging and post-dredging periods would be reduced by only 13% if pre- and post-dredging flows were comparable.[234]

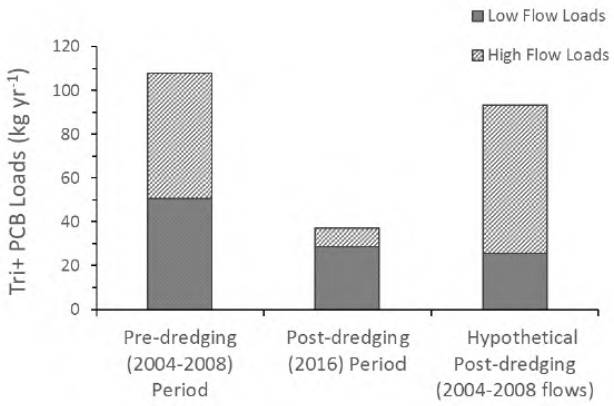

Figure 9. Estimated Tri+ PCB loads to the Lower Hudson for the 2004-2008 pre-dredging period, the 2016 post-dredging period and a hypothetical post-dredging period based on 2004-2008 flow record. Results are presented as stacked bars for low-flow (< 13,000 cfs) and high-flow (> 13,000 cfs) loads.

According to HRF,

> Tri+ PCB loads for low flow conditions were approximately 27 kg/yr for both the 2016 post-dredging period and hypothetical post-dredging scenario. This indicates that year-to-year variations in river flow will have a small effect on Tri+ PCB loads during low flows. However, Tri+ PCB loads during high flows showed large differences. This result indicates that Tri+ PCB loads during high flow conditions will likely show large year-to-year variations; e.g., from 8.3 kg/yr based on the 2016 flow record to potentially more than 100 kg/yr if the river experiences another year like 2011 with three major high flow events.[235]

[232] *Id.* at ii-iii.
[233] *Id.* at 34.
[234] *Id.* at 14.
[235] *Id.* at 14.

This analysis shows that EPA should never have expected dredging above the Federal Dam to have a major effect on the river downstream. This conclusion is further bolstered by existing post-dredging data, since 2016 was actually an abnormally low year in terms of PCB loading, but fish tissue levels still showed little to no recovery.

3. No Water Column Response Was Observed in the Lower Hudson and the Response Is Not as Anticipated in the Upper Hudson.

EPA s water column data to date shows that the impacts on water column PCB concentrations from dredging are much more immediate and localized than assumed in the 2002 ROD. Although Tri+ PCB concentrations in the Upper Hudson water column showed a relatively rapid response to the dredging, the Lower Hudson River has been slow to respond. This lag is due in part to the cyclic transfer between the surface sediment and water column during resuspension and deposition, and the fact that post-dredging Tri+ PCB concentrations averaged four times higher than predicted by the 2002 ROD models.[236] The result is that additional years of MNA will be required to reduce PCB concentrations in the water column—as well as in fish and sediment—to acceptable levels.

In short, dredging produced results in water column concentrations upriver, but not downriver. EPA s modeling is inadequate to predict if and when Lower Hudson water column PCB concentrations will reach the target concentration of 5 ng/L. Tri+ PCB concentrations at Lower Hudson River monitoring stations 1995-2016 show *no* decline,[237] and, contrary to the 2002 ROD expectations, the Poughkeepsie water column data showed no dredging impacts, suggesting that water column PCB concentrations are regulated by local conditions. [238] HRF attributed the faulty modeling at Poughkeepsie to the complexity of sediment transport and dynamic response in the Lower Hudson.[239] Ultimately, it is unlikely that activities or conditions in the Upper Hudson River will have any significant impact on water column concentrations below the Federal Dam, further supporting the need for a full remedial investigation and feasibility study of the Lower Hudson River.

VII. EPA Must Make a Not Protective Finding for the Lower Hudson.

In the Proposed Second FYR, EPA makes no protectiveness determination with regard to the Lower Hudson River—over 150 miles of the Superfund Site from the Federal Dam in Troy to the Battery in New York City. In contrast, in the First FYR, EPA found that the remedy will be protective for the entire 197-mile Superfund Site.[240] EPA is now walking back that conclusion, finding that the will be protective determination only applies to the Upper Hudson River—the 40 miles of the Superfund Site north of the Federal Dam.[241]

[236] NOAA Study at 499.
[237] 2017 FYR App x 1 at Fig. A1-2.
[238] *Id.* at 4-8.
[239] HRF Report at iii.
[240] 2012 FYR at vi, 40.
[241] 2017 FYR at 8, 24.

EPA s decision not to include a sitewide protectiveness determination is a major departure from the First FYR (although EPA fails to discuss it openly or clearly), and further evidences the need for a full remedial investigation and feasibility study for the Lower Hudson River. EPA s decision not to evaluate whether the remedy is protective for the lower 150 miles of the site renders the Proposed Second FYR deficient. The Lower Hudson River, which constitutes nearly 80% of the Hudson River Superfund Site, is lined with cities, towns, and villages that depend on the river for recreation, economic opportunities, and drinking water. Despite the significance of this portion of the Site, EPA has declined to make any protectiveness determination, essentially choosing to ignore the reality that the benefits of the sediment cleanup that were supposed to materialize downstream have not done so in any meaningful way.

If EPA had undertaken the required analysis for the Lower Hudson, it should have led the agency to conclude that a full remedial investigation and feasibility study for the Lower Hudson is necessary.[242] But, even without that analysis, EPA has enough information to reach the same conclusion. The Proposed Second FYR candidly states that data collected so far show that the active remediation—the dredging— in the Upper Hudson is not having any measurable impact on PCB contamination levels in the Lower Hudson.[243] In addition, the model that EPA relied upon to devise the 2002 ROD goals underestimated fish tissue concentrations in the Lower Hudson.[244] Furthermore, the Proposed Second FYR calls for more monitoring in the Lower Hudson,[245] although it fails to establish any mechanism or timeframe for this to take place.

In short, existing data shows that the remedy is not currently protective of human health and the environment in the Lower Hudson River. This is particularly troubling considering that many people in the Lower Hudson, including many New York City residents from low-income communities and communities of color, either rely on subsistence fishing from the Hudson River as an important source of food or would like to do so. The simple fact that subsistence fishing occurs in the 150-mile stretch of the Hudson River below the Federal Dam, particularly in and around New York City, reinforces the need for EPA to ensure that the cleanup is protective of the entire site. Omitting a protectiveness determination for this portion of the Site does nothing but create further concern and confusion among the millions of people who live, work, and play along the Lower Hudson from Troy to Manhattan.

VIII. Changes in Implementation of Dredging Project Do Not Explain Lack of Fish Recovery Within Expected Timeframes.

The selected remedy was premised on achieving a relatively rapid decline in fish tissue PCB concentrations, reaching a species-weighted average concentration of 0.4 mg/kg within five

[242] At the recent Informational Meeting in Poughkeepsie, NY, EPA officials stated that they weren t there yet when it comes to whether a RI/FS is warranted for the Lower Hudson River. *See* Oceans 8 Films, *Hudson River Action - Tell EPA: Protect people and wildlife, not GE* at minute 2:45 (June 28, 2017) *available at* https://vimeo.com/225670244. EPA itself in the 2017 FYR has called for more data collection in the Lower River, 2017 FYR at 57, but has not explained *how* such information collection will be conducted. Similarly, the 2017 FYR does not evaluate on what timeline such research will be undertaken, nor what event or evidence would trigger a full-blown RI/FS for the Lower Hudson River, rather than the mere additional information described in the 2017 FYR.

[243] 2017 FYR at 6, 33.

[244] NOAA Study at 499.

[245] 2017 FYR at 57.

years of the completion of dredging, or by 2020.[246] The most recent data from 2016 indicates that average fish tissue concentrations measured at 1.3 mg/kg,[247] or more than three times the 0.4 mg/kg target level. As discussed in more detail above, based on independent scientific analysis as well as analyses by DEC and NOAA, EPA s estimated 8% decay rate is not supported, and a significantly lower decay rate of 3-5% is likely more accurate.[248] However, even assuming *arguendo* that EPA s 8% rate is accurate, it is incredibly improbable that fish tissue levels will approach 0.4 mg/kg by 2020. In fact, at an 8% decay rate, it will take another 11 years to reach the five-year target; at a 5% decay rate, it will take another 18 years. In order to reduce 1.3 mg/kg to 0.4 mg/kg by 2020, the decay rate would have to be over 25%—a practical impossibility.

In an attempt to explain away this reality and justify its will be protective determination, EPA spent an entire appendix to the Proposed Second FYR discussing why fish tissue levels remain so far above the 2002 ROD expectations. EPA s unsubstantiated hypothesis is that changes in the implementation of the dredging project from what was anticipated in the 2002 ROD led to increased PCB levels in water that have delayed fish tissue recovery.[249]

The 2002 ROD anticipated that dredging would occur from upstream to downstream, and that two sediment processing facilities would be used, at least one of which would be located downstream of most dredging operations.[250] However, for various operational reasons, EPA determined that the project would follow a general upstream-to-downstream progression, but at times dredging would occur in multiple river sections at the same time, especially during the last two to three seasons of dredging.[251] EPA also decided to use a single, upstream facility, which resulted in more vessel traffic over the project area in the later years of dredging.[252] EPA claims that these operational changes resulted in increased levels of suspended PCBs over the entire project area and, therefore, fish tissue concentrations may still be within the ROD-anticipated period of equilibration. [253] Based on this rationale, EPA rejects attempts to compare observed data to ROD forecasts.[254]

However, EPA fails to mention that the 2002 ROD timeframes for the interim targets of 0.4 mg/kg and 0.2 mg/kg—within five years and 16 years after dredging, respectively—already took into account up to two years for equilibration. Thus, the time to reach equilibration should not be a justification to extend the interim targets even farther into the future.

A closer look at the Remedial Action Monitoring Program (RAMP) data also belies EPA s hypothesis. The expected fish tissue recovery trend can be described as a significant rapid decline in concentrations very soon after dredging (a step-function), followed by a reasonably

[246] 2002 ROD at 50; *see also id.* at 73 (Table indicating .4 mg/kg average fish tissue concentration will be reached in 2012, or three years after the then-expected end of dredging in 2009).
[247] 2017 FYR at 33.
[248] *See generally* SSPA, DEC Report, NOAA Study.
[249] *See* 2017 FYR at 37.
[250] *Id*.
[251] *Id*.
[252] *Id.*
[253] 2017 FYR App x 8 at 2-17.
[254] *Id*. at 3-3.

stable decay rate. This trend is illustrated by the fish recovery trends for the Cumberland Bay-Wilcox site, highlighted by EPA in Figures A8-5.1 and 5.2 in the Proposed Second FYR. Upon a review of the project data, it is apparent that the step-function decline seen at Cumberland Bay in two to four years post-dredging have already occurred for the vast majority of species at the vast majority of stations along the Hudson River Site. This indicates that fish tissue levels have likely already reached equilibrium. Fish tissue levels elevated beyond what was expected at this point post-dredging are probably *not* short-term impacts due to differences in project implementation, but an indication of a significant delay in long-term recovery with negative implications for the protectiveness of the remedy.

This step-function trend is visible in Figures 5A-5R of the SSPA Report and in Figures A8-4.1-4.12 of the Proposed Second FYR. For nearly every species at every station, one discerns a trend where in the pre-dredging period concentrations remain more or less stable, and then when dredging occurs near a particular station, one observes an increase in concentrations for one or two data points. Following the increase, there is a clear, sharp decline for the one to two years. That sharp decline subsequently stabilizes into a slow, gradual decline.

EPA itself admits that [i]n general, fish tissue PCB levels were observed to recover to pre-dredging levels within one to three years after completion of dredging upstream of a monitoring station. [255] In River Section 1, the RAMP data indicates that fish tissue levels peaked within one to two years after dredging, then rapidly declined.[256] For River Section 2 and River Section 3 the same general patterns prevail, with very few exceptions (e.g., black bass at fish monitoring stations SW1 and ND2).[257] Out of 20 measured trends in River Section 1 (four species each at five stations), 16 trends have already declined to at or below the Baseline Monitoring Program (BMP) mean, and 15 have decreased below the Lower Confidence Level (LCL) of the BMP as of 2016 – indicating they have reached or surpassed equilibrated levels.[258] Out of 16 trends in River Section 2 (four species each at four stations), 13 have declined to or below the BMP mean and 11 have declined to or below the BMP LCL.[259] In River Section 3, all 20 trends (four fish each at five stations) were at or below the BMP mean in 2016, although only 11 have fallen below the BMP LCL.[260] EPA also stated in a presentation to the Community Advisory Group for the Site that fish tissue concentrations decrease rapidly following spikes related to environmental dredging at other sites, and that the agency likewise expects a rapid return to baseline in the Hudson River.[261]

In short, post-dredging equilibration has already occurred in the Upper Hudson River. There has already been a large step-down in fish tissue concentrations as a result of the dredging, and from this point forward, EPA should only anticipate a stable decay rate in the absence of further removal or sequestration of PCBs. Moreover, Figures A8-1 and A8-2 demonstrate the extent to

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[255] 2017 FYR App x 8 at 2-14
[256] *Id.* at Figs. A8-4.1 to 4.3.
[257] *Id.* at Figs. A8-4.4 to 4.12.
[258] 2017 FYR at Table A8-7.
[259] *Id*.
[260] *Id*.
[261] U.S. Envtl. Prot. Agency, *Powerpoint: PCBs in Fish Tissues at the Hudson River PCBs Superfund Site: Update on Results of Baseline and Remedial Action Monitoring (2004-2013)* at 18 (Oct. 30, 2014) *available at* http://www.hudsoncag.ene.com/files/FishDataSummaryOct2014.pdf. (Attachment R)

which the 2002 ROD envisioned dredging approach was consistent with actual implementation. As EPA states, there are some deviations from the way in which the 2002 ROD contemplated the dredging project. However, the 2002 ROD approach and actual project approach are remarkably similar, with few relatively minor deviations (i.e., dredging an upstream Certification Unit (CU) one dredging season prior to the CU immediately downstream). A notable exception is the dredging upstream of the TD1 monitoring station in River Section 1 in 2015, but this is an outlier to the general pattern. Overall, it is apparent that the dredging program progressed more or less as planned. EPA simply does not have adequate justification to identify these changes in implementation as the main reason fish tissue levels remain elevated.

In sum, the data does not support the idea that fish tissue concentrations are still being significantly impacted by the dredging activity. The expected step-function drop in fish tissue levels has already occurred; in most species at most stations, the fish have already equilibrated. Thus, EPA is left with fish tissue concentrations that are more elevated than expected at the time of the 2002 ROD and it is very unlikely that these concentrations will decline at the rate EPA predicted. In light of these conditions, the agency needs to take a hard look at what went wrong and what must be done to ensure the RAOs are met within the approximate timeframes set forth in the 2002 ROD.

IX. EPA Failed to Act On the Follow-Up Recommendations and Key Concerns from The 2010 Peer Review Panel, the Natural Resource Trustees, New York State, and the Hudson River Foundation To Adaptively Manage the Remedy.

Since at least 2010, scientists from federal, state, and independent institutions have repeatedly shared with the EPA and the public substantive and credible analyses that clearly indicate the Hudson River Superfund Site remedy and cleanup to date is *not* protective of human health and the environment.

A. 2010 Peer Review Panel Findings.

In the Spring of 2010, a panel of seven independent scientists selected by both the EPA and GE was tasked with evaluating all aspects of Phase 1 dredging operations from the first year of active remediation and reporting back recommendations for changes to remedial designs for Phase 2 operations. The panel s report was released in September 2010.[262] While the Panel acknowledged there were serious challenges in Phase 1, it recommended that with appropriate adjustments based on good data and sound science, Phase 2 remedial action should proceed. One of the most critical observations (and resulting recommendation) made by the panel was that neither EPA nor GE has sufficient data or a credible tool to project recovery.[263] The panel stated that the HUDTOX/FISHRAND models (originally used during development of the 2002 ROD) are outdated and inadequate to accurately project MNR and post- dredge fish recovery rates.[264]

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[262] *See generally Hudson River PCBs Site Peer Review of Phase 1 Dredging* (Sept. 10, 2010) [hereinafter Phase 1 Review] *available at* https://www3.epa.gov/hudson/pdf/hudsonriverphase1dredgingreport_final.pdf. (Attachment S)

[263] *Id.* at 13.

[264] *Id*.

The panel told EPA and GE that in order to create more effective and comprehensive dredge-design paradigms for successive remediation protocols, they should collaborate on the creation of a new Fate, Transport and Risk Model utilizing the real-time data collected during the first phase of dredging and data from year one of Phase 2. The updated fate, transport, and risk model would enable EPA and GE to better understand the implications of operational changes on long-term recovery rates, and would support EPA and GE in making appropriate and meaningful risk management decisions about dredging productivity, BMPs, and the long-term fate and transport of PCB residuals and resuspension and release.[265]

The panel further advised the five-year timeline for project duration should be extended to provide necessary flexibility to meet the actual remediation need of the river while protecting long-range remedy goals. Finally, the panel stated that there should not be a limit on the PCB mass to be removed during remediation as the total amount of PCB inventory in the river is unknown.

B. Federal Trustees Study Supports A "Not Protective" Determination.

In direct contradiction to the conclusions made by EPA Region 2 staff in the First FYR and in the Proposed Second FYR, scientists from federal, state and independent institutions have shared with EPA and with the public substantive and credible analysis that clearly indicate the Hudson remedy and the cleanup action to date is not protective of human health and the environment as implemented.

In an inter-agency communication to EPA Region 2 and in a peer-reviewed study,[266] NOAA informed EPA that recovery of the Upper and Lower Hudson will not be reached due to elevated PCBs remaining in surface sediment equivalent to a series of Superfund Sites being left behind, [267] and that post-remedial PCB concentrations in the Upper Hudson River sediments will exceed previous EPA model predictions by a factor of 3-to-5 times. [268] The Trustees also warned EPA that achieving the Remediation Goals for PCB fish tissue concentrations in the Lower Hudson River would take several decades longer than expected,[269] and that additional removal of PCB-contaminated sediment in the [Upper Hudson] [is] needed to achieve reductions in [Lower Hudson] fish PCBs anticipated in the ROD. [270]

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[265] 2012 FYR at 36.

[266] *See* NOAA Study; *see also* Nat l Oceanic and Atmospheric Admin., *Powerpoint: Re-visiting Model Projections of Lower Hudson River Fish PCBs* (May 19, 2015) [hereinafter May 19, 2015 NOAA Powerpoint] *available at* https://www.fws.gov/northeast/ecologicalservices/HudsonRiver/docs/Lower%20Hudson%20River%20Fish%20HRF%20Field%2005192015.pdf. (Attachment P)

[267] Letter from Dr. Robert Haddad, Nat l Oceanic and Atmospheric Admin., to Robert Sussman, U.S. Envtl. Prot. Agency, entitled Phase 2 Remediation, Hudson River PCB Superfund Site (Dec. 2, 2010) [hereinafter Haddad Letter], available at http://www3.epa.gov/hudson/pdf/CorrespondenceReceived_FiveYearReview_HudsonRiverPCBs.pdf. (Attachment T)

[268] May 19, 2015 NOAA Powerpoint at 15.

[269] NOAA Study at 499.

[270] May 19, 2015 NOAA Powerpoint at 36.

C. New York State Analysis and Review Support A "Not Protective" Determination.

Acting on behalf of the interests of New York State and its citizens, the Office of the Attorney General (OAG) notified EPA in September 2016 that it is now clear that the remedy has not met the remedial action objective of reducing PCB concentrations in fish to 0.4 mg/kg by 2016, and may not reach the ROD s more dramatic reductions to 0.05 mg/kg. [271]

The OAG advised the EPA that it must determine with reasonable certainty the time-frame by which there will be a reduction of PCB concentrations in fish so that fish consumption advisories for PCBs may be lifted in all contaminated River reaches of the Hudson River for all species and that EPA s determination of remedy s protectiveness must be supported by a comprehensive Fish Consumption Survey to quantify current and potential future human exposure. [272] Furthermore, the OAG insisted that EPA must clearly define the time-frame for achieving the remedial action objectives set forth in the ROD and cautioned that in evaluating that time-frame, EPA must take into account the change in fish tissue sampling that occurred during GE s implementation of the baseline and remedial fish monitoring.[273] The OAG letter reflects the State s deep concerns regarding localized effects of human exposure in certain more contaminated areas of the River and urged EPA to evaluate those effects as part of EPA s Five Year Review and protectiveness determination. [274]

In December 2016, the DEC—a Hudson River Superfund Site Trustee and primary natural resource manager for the State s natural resources— issued a preliminary review of the effectiveness of the cleanup to date.[275] DEC concluded that the Remedy is not protective of human health and the environment based on uncontrolled risks, and EPA should undertake all necessary actions to ensure that the remedy becomes fully protective to the benefit of the people of New York State. [276]

DEC s review identified a serious failure that EPA continues to dismiss: that there are known exposures to both human and ecological receptors which have not been controlled and which remain in excess of EPA s acceptable risk range. [277] Moreover, sediment concentrations higher than anticipated will remain after dredging, [which] indicates that the 3 targeted fish PCB concentrations will not be reached in the time frames identified in the ROD. [278] DEC recommended that EPA optimize the remedy through further remedial work as necessary to achieve the targeted fish PCB reductions identified in the ROD. [279]

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[271] New York State Attorney General Letter (Sept. 16, 2016) *available at* http://www.scenichudson.org/sites/default/files/9.16.16_Letter-NYOAG-to-EPA-re-cleanup-failure.pdf. (Attachment U)
[272] *Id.* at 4-5.
[273] *Id.*
[274] *Id.*.
[275] *See generally* DEC Report.
[276] DEC Report at 3.
[277] *Id.* at 2.
[278] *Id.* at 2, 3.
[279] *Id.*

DEC s review also underscores the failure of the remedial action to achieve benefits in the Lower Hudson River. As such, DEC informed EPA that it must expand the investigation of the site to include performance of a Remedial Investigation and Feasibility Study for the portion of the site between the Federal Dam at Troy and the Battery in New York City . . . to address the currently uncontrolled unacceptable risks to human health and the environment.[280]

D. Hudson River Foundation Report Does Not Support "Will Be Protective" Determination.

In fall of 2016, HRF directed a team of independent scientists and engineers with a wealth of expertise related to PCBs and the Hudson River to review project data related to the Upper Hudson Superfund dredging program. [281] On June 1, 2017, HRF released its report, finding that based on 2016 post-dredging monitoring, TPCB concentrations in fish throughout the Upper and Lower Hudson remain above interim target levels and remediation goal specified in the ROD. [282]

Similar to concerns expressed by NOAA and DEC, HRF observed that EPA has not planned an adequate data collection program to find out if monitored natural attenuation will work as expected. HRF advised that modifications to the post-dredging monitoring program and continued evaluation of the next few years of monitoring data are therefore recommended to assess if natural attenuation will be sufficient in reducing PCB concentrations in fish in a reasonable time frame or if additional remedial actions will be required. [283]

The panel corroborated the findings of DEC and the Federal Trustees that a major assumption in the ROD—that the Lower Hudson would receive similar benefits from the dredging action in the Upper Hudson—did not, and is not, likely to occur. In fact, water column, sediment and fish in the Lower Hudson below Albany are showing slow responses to the Upper Hudson dredging program. [284] This may be due to the complexities of sediment transport in the Lower Hudson [285] as noted by HRF, but the indisputable fact is the lower portion of the Superfund Site is showing little or no benefit from the dredging in the Upper Hudson. Over the past 10 years, 5.2 million pounds of PCB-contaminated sediment have landed in the Lower Hudson, presenting an uncontrolled risk that EPA is failing to address.

While HRF did not seek to answer the question of whether the cleanup of the Hudson River Superfund Site is protective, it did analyze all available project data, concluding that there is not enough post-dredging information to make a definitive conclusion regarding the success of the remedy.[286] HRF suggests that a fuller and more comprehensive analysis of the effects of the

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[280] *Id.* at 2.
[281] Hudson River Found., *About HRF* (last visited Aug. 31, 2017) http://www.hudsonriver.org/?x=about (The purpose of the Hudson River Foundation is to make science integral to decision-making with regard to the Hudson River and its watershed and to support competent stewardship of this extraordinary resource.).
[282] See generally HRF Report at 17.
[283] *Id.* at i.
[284] *Id.*
[285] *Id.* at iii.
[286] *Id.* at 9-10 (Only a year s worth of post-dredging data was available to the panel. . . . It could therefore be argued that one year of post-dredging monitoring data is not sufficient to evaluate the full benefits of the dredging program.).

dredging will be possible as new data are collected and other evaluation tools, such as numerical models, are utilized in understanding the longer-term impacts and trajectories. [287]

X. Answering the Three Five Year Review Questions Result in the Conclusion That Remedy is Not Protective of Human Health and the Environment.

Question A: Is the remedy functioning as intended? NO.

As discussed in detail in herein, current data demonstrates that the remedy is not functioning as intended. While removal of PCB-laden sediment has resulted in some reduction in fish tissue, surface sediment, and water PCB concentrations, the fact remains that all three media were far more contaminated than EPA believed at the time it issued the ROD. EPA failed to reevaluate its chosen remedy in light of this information, despite disagreement from its sister federal agencies, New York State, and independent scientists, as well as environmental organizations and the public.

As a result, fish tissue levels remain 300% greater than the first interim goal—0.4 mg/kg—which, according to the ROD, should be reached in less than three years, by 2020. Even EPA acknowledges that it is extremely unlikely this target will be met. In addition, EPA admits that the Lower Hudson is not responding as predicted to the dredging upriver and that it appears that local sediments, rather than upstream load, are the main driver of fish body burdens of PCBs.

Question B: Are the exposure assumptions, toxicity data, and Remedial Action Objectives used at the time of the remedy still valid? NO.

EPA has failed to acknowledge in the Proposed Second FYR any new information related to exposure assumptions or toxicity data that could impact the human health risk assessment. First, recent science indicates that PCBs are more toxic than previously thought. While EPA is still classifying PCBs as probable human carcinogens (EPA has not officially changed the Integrated Risk Information System listing, toxicity values or carcinogenicity of PCBs in the last 17 years[288]) with a cancer weight-of-evidence classification B2,[289] the International Agency for Research on Cancer (IARC), of the World Health Organization, has now listed PCBs as a known human carcinogen.[290] In addition, dioxin-like PCBs can now be evaluated via EPA s listing of non-cancer endpoints for dioxin[291] via the reference dose (RfD)[292] in EPA s

[287] HRF Report at 2.
[288] *See generally* U.S. Envtl. Prot. Agency, *Integrated Risk Systems Information Chemical Assessment Summary: Polychlorinated biphenyls* (May 1, 1989) [hereinafter EPA IRIS for PCBs] *available at* https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0294_summary.pdf.
[289] *See generally* ATDSR PCBs Case Study, *supra* n.7.
[290] IARC, a branch of the World Health Organization, coordinates and conducts research on the causes of human cancer and develops scientific strategies for cancer control. In February 2013, 26 experts from 12 countries met at IARC, Lyon, France, to reassess the carcinogenicity of polychlorinated biphenyls (PCBs). On the basis of sufficient evidence of carcinogenicity in humans and experimental animals, the IARC classified PCBs as carcinogenic to humans (Group 1). *See generally* IARC PCBs Carcinogen Evaluation, *supra* n.9.
[291] U.S. Envtl. Prot. Agency, *Risk Assessment for Dioxin at Superfund Sites* (Feb. 17, 2012) *available at* https://www.epa.gov/superfund/risk-assessment-dioxin-superfund-sites_toxicity.
[292] An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects

Integrated Risk Information System (IRIS)[293] as well as several additional toxicological endpoints which have been updated in terms of health effects.

Second, recent science indicates that exposure to PCBs through inhalation is a more significant risk than previously believed. The risk characterization of the ROD and the intention of the RAOs are primarily intended to control unacceptable PCB exposures through consumption of contaminated food (i.e. fish). [294] However, since 2002, the scientific community has documented that exposures to PCBs can occur through contaminated water, direct skin contact, or breathing contaminated air.[295] In a 2015 Review of Scientific Literature, David O. Carpenter, M.D., presents information that indicates the inhalation of vapor-phase PCBs may be as or even more important than ingestion via fish consumption and other animal fats.[296] The research highlights the severity of the potential risks from volatilized or airborne PCBs, which have been associated with certain chronic illnesses—such as cancer, cardiovascular disease, hypertension, and diabetes—even at relatively low levels.[297]

All of this information adds to the growing body of research which demonstrates that PCBs are more toxic to humans than previously believed when the human health risk assessment was being developed for the ROD. As a result, the Proposed Second FYR needs to address the greater toxicity as a change in assumptions and new information that was not available at the time the ROD was developed.

Finally, significant changes in demographics and fish consumption patterns on the Hudson River, particularly in the Lower Hudson, mean that more people are relying on Hudson River fish for subsistence than at the time the ROD was issued. Due to the failures of longstanding fish consumption advisories to protect human health, an uncontrolled exposure through consumption of fish. Recent angler surveys have shown consumption of fish from the Hudson River remains a major health concern despite the existence of longstanding NYSDOH fish consumption advisories. In 2012, the Cornell Cooperative Extension performed a survey of over 300 anglers, finding that approximately 11% of those surveyed ate Hudson River fish.[298] In 2013, NYSDOH

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during a lifetime. *See* U.S. Envtl. Prot. Agency, *Reference Dose (RfD): Description and Use in Health Risk Assessments* (Mar. 15, 1993) [hereinafter EPA RfD Fact Sheet] *available at* https://www.epa.gov/iris/reference-dose-rfd-description-and-use-health-risk-assessments.

[293] The IRIS Program is located within EPA s National Center for Environmental Assessment (NCEA) in the Office of Research and Development (ORD). *See* U.S. Envtl. Prot. Agency, *Basic Information about the Integrated Risk Information System* (last visited Aug. 31, 2017) *available at* https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system. (Attachment V)

[294] EPA s program and regional offices identify human exposure pathways and estimate the amount of human exposure under different exposure scenarios (Exposure Assessment). EPA RfD Fact Sheet at 1.3.3.Then they combine their exposure assessment with the hazard information and toxicity values from IRIS to characterize potential public health risks (Risk Characterization). *Id.* at 1.3.4.

[295] *See* EPA IRIS for PCBs.

[296] D. Carpenter, *Exposure to and Health Effects of Volatile PCBs,* Rev. Envtl. Health 1 (Feb. 2015) (Attachment W)

[297] *See* M. Kouznetsova et al., *Increased Rate of Hospitalization for Diabetes and Residential Proximity of Hazardous Waste Sites*, 115(1) Envtl. Health Perspectives 75 (Jan. 2007); Alexander Sergeev & David Carpenter, *Hospitalization Rates for Coronary Heart Disease in Relation to Residence Near Areas Contaminated with Persistent Organic Pollutants and Other Pollutants*, 113(6) Envtl. Health Perspectives 756 (Jun. 2005). (Attachment X)

[298] *See* New York State Dep t of Health, *Hudson River Fish Advisory Outreach Project Update*, 5 (Sep. 19, 2013), *available at*

presented preliminary results of its own angler survey showing even higher consumption percentages (near 50%), also noting that awareness of fish consumption advisories in the more populated and linguistically diverse Lower Hudson was about half of what it was in the Mid- and Upper Hudson regions.[299]

Since 2000, additional populations that rely on subsistence fishing have moved into Mid- and Lower Hudson River communities, and surveys indicate these anglers feed fish to their families.[300] The Proposed Second FYR also fails to consider these changes in subsistence fish consumption patterns, which increase exposure and human health risks. Subpopulations of subsistence anglers are currently consuming small forage fish in ways that not been included in the human health risk assessment, such as utilizing the entire fish in preparing spiced whole fish mash or paste for flavoring traditional dishes. Previous risk assessments were limited to the risks of consuming larger, traditional trophy or game fish, such as bass and perch. It is important that the exposure assumptions take into account all of the consumption patterns order to accurately capture the risks that the Hudson River Superfund Site poses to human health.

Question C: Has any other information come to light that could call into question the protectiveness of the remedy? YES.

The decision-making process that led to the ROD relied on a complex suite of human health risk assessment tools and guidelines, [301] as well as multiple sediment and water sampling programs. Those were in turn used by EPA and GE as the baseline informational database used in multipart mechanistic mathematical models to forecast future concentrations of PCBs in the Hudson River.[302] While the extensive body of scientific information for the site was appropriately employed in the remedy selection, EPA has failed to apply that same diligence to the evaluation of the newest scientific analysis and actual project data in the Proposed Second FYR.

Post-ROD data collected after 2002 show higher levels of surface sediment contamination than anticipated in portions of River Sections 2 and 3 that were not targeted for dredging. In fact, analyses of post-ROD data indicate that post-remediation PCB concentrations will be five times higher than assumed by the ROD. These residuals raise significant scientific uncertainty as to whether all RAOs, including target PCB levels in fish, will be fully achieved.

Furthermore, sediment and bioaccumulation models (HUDTOX and FISHRAND) used in the ROD are no longer considered scientifically valid. The models require re-examination, in light of

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http://www.hudsoncag.ene.com/files/Hudson%20Fish%20Health%20Advice%20Outreach%20091913.pdf. (Attachment Y)

[299] *See id.* at 6, 20; Hudson River PCBs Community Advisory Group, *Hudson CAG Meeting Summary*, 5-6 (Sep. 19, 2013) available at http://www.hudsoncag.ene.com/files/Final%20Meeting%20Summary_Sept192013.pdf. (Attachment Z)

[300] Garcia, Michael, *Hudson River Angler Survey*, Scenic Hudson and Sierra Club (Dec. 2016), *available at* http://www.scenichudson.org/sites/default/files/HR_Angling_Study.pdf (Attachment AA)

[301] Phase 1 Review (summary of existing conditions), 1991 Database Report, 1995 Data Evaluation and Interpretation Report, 1997 Low Resolution Sediment Coring Report, 1998 Human Health Risk Assessment, 1999 Revised Baseline Ecological Risk Assessment, 2000 Revised Human Health Risk Assessment, 2000 Revised Baseline Monitoring Report, 2000 Feasibility Study Report, *all available at* https://www3.epa.gov/hudson/plans.html.

[302] *See* DEC Report.

the above-referenced data, to determine the likelihood that RAOs will be fully achieved. Post-Phase 1 modeling by GE validated the ROD s conclusions that dredging of contaminated sediment does not impede recovery of the river through resuspension of PCBs, but rather achieves significant progress towards RAOs by removing PCBs from the system.[303] However, neither this model nor any other updated sediment transport or bioaccumulation model has been used to date to evaluate how much higher-than-expected surface sediment PCB concentrations outside of the area targeted for dredging will impact the ability of the remedy to be protective of human health and the environment in the future.

XI. EPA Must Take the Following Actions Necessary to Ensure Protectiveness.

A. Clearly Define Goalposts for Success and Failure of the Cleanup and Order Additional Remediation.

EPA s Proposed Second Five Year Review of the Hudson River Superfund Site lacks clear metrics to evaluate the success or failure of the cleanup. Without clear metrics, the public is left in the dark as to how EPA compared current conditions with the 2002 ROD expectations to reach its conclusion that the remedy will be protective. Therefore, we urge EPA to identify and list the criteria that it used to evaluate the performance of the remedy in the Final Second FYR, as well as the criteria that the agency will use for subsequent reviews. This should lead to a fair consideration of all relevant targets, not a selective view of only the targets that are being met.

The interim and final fish tissue concentration targets should be among the most important benchmarks that EPA uses to evaluate the success or failure of the remedy. Despite EPA s reliance on the accelerated timelines to meet fish tissue targets in selecting the remedy, the agency fails to measure current conditions against them in a straightforward way. This is not acceptable. Clear benchmarks, measured in years after dredging, would ensure that all interested stakeholders—GE, Federal Trustees, DEC, community and environmental advocates, and the public—understand whether the cleanup is making the necessary progress toward protection of human health and the environment. Moreover, benchmarks would ensure that EPA, and in turn, GE, can be held accountable for cleaning up the River *within in the timeframes anticipated in the 2002 ROD*.

We urge EPA to expressly include at least the following benchmarks as a way to measure the success or failure of the remedy to protect human health and the environment both in subsequent five-year reviews and as more data becomes available each year:

1. Species-weighted fish fillet Upper Hudson average PCB concentrations must be at or below 0.4 mg/kg within five years of the completion of dredging (by 2020).
2. Species-weighted fish fillet Upper Hudson average PCB concentrations must be at or below 0.2 mg/kg within sixteen years of the completion of dredging (by 2031).
3. Largemouth bass, whole body PCB concentrations must be within the recalculated range of 0.2 mg/kg to 0.07 mg/kg within 23 years of the completion of dredging (by 2038).
4. Species-weighted fish fillet River Section 3 average PCB concentrations must be at or below 0.05 mg/kg within 43 years of the completion of dredging (by 2058).

[303] *See* Phase 1 Report.

Because the selected remedy is not currently protective, and people are still face unacceptable human health risks *daily* due to PCB contamination, EPA should further clarify that failure to meet these benchmarks means that the remedy is not functioning as intended. EPA should also develop a plan for adaptive management so that it is prepared to address potential problems with the remedy as they become apparent.

Moreover, the failure to meet the benchmarks should indicate to the agency that further active remediation is necessary. As discussed *supra*, the time to reach the human health targets was an important factor in EPA s selection of an active remedy. EPA s own rationale makes it clear that delays of ten or more years in reaching the interim and final 2002 ROD targets are unacceptable. Therefore, failure to meet the benchmarks within the timeframes anticipated in the ROD— including the current failure to meet the five-year target— should prompt EPA to order GE to perform additional remedial action. Finally, EPA should consider adding species-specific or more geographically limited targets, as well as ecological targets, to the criteria that evaluates to determine the success or failure of the remedy.

B. Immediately Order GE to Initiate a Remedial Investigation and Feasibility Study for the Lower Hudson.

EPA implicitly admits that the cleanup is not protective of human health and the environment in the Lower Hudson River by omitting a protectiveness determination for the 150-mile stretch below the Federal Dam.[304] As discussed above, in the First FYR, EPA issued a sitewide protectiveness determination for the entire 197-mile Superfund site.[305] However, the Proposed Second FYR contains no such determination.[306] While EPA claims that the cleanup will be protective in the Upper Hudson River (despite evidence to the contrary discussed herein), EPA makes no official protectiveness determination about the cleanup for the 150-mile stretch of the Hudson River below the Federal Dam.[307]

It is abundantly clear that EPA should order a full remedial investigation and feasibility study for the Lower Hudson River. EPA admits that fish tissue concentrations in the Lower Hudson River are not responding as anticipated; EPA concedes that the Lower Hudson is responding more slowly under MNA; and EPA recognizes that there is little to no change in fish tissue concentrations from Poughkeepsie downstream. Furthermore, the remedy only produced results in water column concentrations upriver, not downriver, indicating that it is unlikely that additional activities in the Upper Hudson River will have any significant impact on the Lower Hudson River.

While EPA says it will continue to investigate the Lower Hudson, it provides no specific plan of action to do so and no criteria to indicate under what conditions it would order a remedial investigation and feasibility study. That is not acceptable. Evidence right now suggests that there is a disconnect between the remedial activities in the Upper Hudson River and the response in

[304] *Id.* at 8.
[305] 2012 FYR at iv, 40.
[306] *Compare* 2017 FYR at 8, 24, 70 *with* 2012 FYR at iv, 40.
[307] *See* 2017 FYR at 8, 24, 70.

the Lower Hudson River. As such, EPA should immediately require GE to conduct a full remedial investigation and feasibility study to address the ongoing PCB contamination in the Lower Hudson River.

C. Collect Additional Data as Expeditiously as Possible.

Among the few points of consensus in EPA s Proposed Second Five Year Review is the need for more data to predict future trends. EPA must ensure that future data collection takes into account expert advice, including that of DEC and HRF.

DEC maintains that a more robust fish and sediment sampling program is necessary for the Hudson River Superfund Site.[308] An expanded sampling program would allow EPA to determine if the current surface sediment PCB concentrations are capable of meeting the intent of the ROD. [309] DEC also recommends that EPA utilize a pool by pool scale in designing the sampling program to better understand the progress made as a result of the remedy.[310]

While EPA claims that it needs eight to ten more years of data, HRF s report suggests that a more reliable sampling program would allow EPA to begin to evaluate future trends much sooner—as early as a couple of years from now. Specifically, HRF recommends that:

(i) EPA Method 1668 (a high resolution, congener-based method) should be used to improve the accuracy and reproducibility of PCB water column, sediment, and fish measurements,
(ii) the USGS suspended sediment monitoring at Waterford should be re-instated to support evaluations of PCB loads to the Lower Hudson,
(iii) additional high flow samples should be collected at Waterford to support evaluations of PCB loads to the Lower Hudson for high flow conditions, and
(iv) PCB concentrations should be monitored in surface sediments and sediment cores from selected locations in the Lower Hudson to improve our understanding of time responses in the tidal freshwater and estuarine portion of the river. [311]

EPA cannot take a wait-and-see approach to data collection, kicking the can down the road to the *next* five-year review or the one after that. While EPA is collecting data, people and wildlife continue to be exposed to dangerous levels of PCBs on a daily basis. EPA must devise fish, sediment and water sampling plans that gather data in an aggressive manner to discern the effectiveness of the remedy as quickly as possible.

[308] DEC Report at 2 (recommending that EPA perform the sampling work necessary to complete a detailed evaluation of the performance of the remedy, including increasing the sampling of sediment and fish tissue to the scale and frequency necessary to optimize the remedy through further remedial work as necessary to achieve the targeted fish PCB reductions identified in the ROD).
[309] *Id.* at 28 (also stating that the current EPA approved sampling plan is not designed to answer that question with the appropriate degree of statistical certainty.).
[310] DEC Report at 40.
[311] HRF Report at iii; *see also id.* at 19-20.

D. Update the HUDTOX and FISHRAND Models.

Using model emulation, NOAA has found that the higher than anticipated residual PCBs could lead to lengthy delays in fish recovery times.[312] In addition, as discussed *supra*, projections of fish recovery indicate considerable delay in the short-term targets. Moreover, estimates of the amounts of PCBs in the sediment changed dramatically after the ROD was issued. Under these circumstances, it is unreasonable to continue to rely upon simulations from a model that is now wholly outdated. Instead, EPA should develop a new transient model that takes account of all the observed data collected during the dredging phase and can provide useful short-term simulations of fish recovery.

E. Evaluate Effectiveness of NYSDOH Fish Consumption Advisories.

EPA did not evaluate the effectiveness of the institutional controls, such as NYSDOH s fish consumption advisories, in addressing the human health risks associated with PCB contamination. Understanding that institutional controls are an imperfect means of managing risk, the 2002 ROD only included them because of the limited time period for which the most restrictive fish consumption advisories would remain in place (i.e., until the interim goals were met). Currently, however, the remedy is failing to meet the ROD goals in the Upper Hudson River, and the remedy is having little to no impact on the Lower Hudson River. The known ineffectiveness of the institutional controls, particularly in light of the remedy s failure to meet the interim goals, means an impermissible level of risk to human health currently exists at the Hudson River Superfund Site.

Furthermore, EPA has not conducted sufficient outreach to subsistence anglers regarding the risks of consuming Hudson River fish. EPA s repeated reliance (particularly in the agency s recent Public Information Meetings) on NYSDOH s fish consumption advisories is insufficient. Despite acknowledging that the fish consumption advisories are *not* successful in preventing people from consuming PCB contaminated fish in unsafe amounts, EPA continues to insist that the implementation of NYSDOH s institutional controls are not within its jurisdiction. However, EPA holds the ultimate statutory responsibility for reducing risk to human health and the environment. If the NYSDOH fish advisories are inadequate to protect the public from PCB contamination risks (as DEC contends, in contrast to EPA s statements in the Proposed Second FYR[313]), *EPA* must either find ways to make those controls protective or implement additional controls. Therefore, it is imperative that EPA improves outreach to communities that are most likely to engage in subsistence fishing.

F. Update the Community Involvement Plan for the Hudson River Superfund Site.

EPA is not performing adequate outreach to communities along the length of the Site. While EPA has a Community Involvement Plan (CIP), it has not been updated in approximately eight

[312] NOAA Study at 495.

[313] *Compare* 2017 FYR at 24 (In the interim, human exposure pathways that could result in unacceptable risks are being controlled.) *with* DEC Report at 27 (Available information indicates that people continue to eat fish despite the institutional controls, and that these exposures represent human health risk beyond the EPA acceptable risk range.).

years.[314] The most recent update to the CIP, in 2009, was only intended to guide activities through the completion of dredging. [315] Now that dredging is complete, EPA should revise the CIP to better address the ongoing risks associated with PCB contamination that will continue for decades along the entire Hudson River Superfund Site.[316]

Although the Proposed Second FYR discusses additional measures mentioned in the First FYR,[317] the agency failed to organize any outreach to environmental justice communities during this comment period. In updating the CIP, EPA should ensure that its outreach extends to the diverse communities present along the Lower Hudson River. The CIP indicates that EPA s community involvement efforts have largely focused on upriver communities.[318] However, communities along the Lower Hudson River, including low-income communities, communities of color, and subsistence fishing communities, will also be exposed to PCB contamination for the foreseeable future.

EPA s community involvement goals include providing understandable information to the public, ensuring that the public has a meaningful opportunity to engage with EPA, and helping the public understanding the Superfund decision-making process.[319] However, it is difficult to understand how many of EPA s community involvement activities could actually meet these goals as they relate to downriver communities. For example, it is not reasonable to expect people who live near the Lower Hudson to benefit from EPA s enhanced physical presence in the Upper Hudson through field offices, public meetings, community events, and media appearances. Additionally, EPA recognizes that the far more populated and diverse Lower Hudson is home to a greater number of non-English speaking residents.[320] However, there is no indication that EPA has made specific efforts to ensure that its outreach materials, like fact sheets, technical documents, and e-mails, are widely available to various audiences.

The CIP s goal with regard to environmental justice is to increase awareness and information about the project, especially in communities that may not know how to access information or that may not have many opportunities or methods to do so. [321] We urge EPA to take a hard look at whether the agency is meeting this goal. EPA originally only scheduled two public information meetings on the Proposed Second FYR, neither of which were located in or near New York City. Moreover, it was clear from the first public information meeting in Poughkeepsie that EPA has failed to undertake sufficient outreach to subsistence fishing communities. When asked who among the crowd of over 300 people was a subsistence fisher, not a single person raised their

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[314] *See generally* U.S. Envtl. Prot. Agency, *Hudson River PCBs Superfund Site Community Involvement Plan* (June 2009) [hereinafter CIP] *available at* https://www3.epa.gov/hudson/cip.htm.
[315] CIP at 1.
[316] *See id*. at 1-4 (stating that [b]ecause EPA does not have the information necessary to identify the precise timing of all activities and points for community involvement, this CIP will remain a living document that will continue to evolve as the project progresses).
[317] 2017 FYR at 25.
[318] *See CIP*. at 1-2 (stating that one of the major elements of EPA s CIP is a notable EPA presence in the upriver community via the Hudson River Field Office.); *id*. at 3-2 (noting that [t]he Upper Hudson River is the focal point for project activities.).
[319] *Id*. at 4-1.
[320] *Id*. 3-2 to 3-5.
[321] *Id.* at 4-12.

hand. EPA should follow its own directive from the CIP, and seek assistance from agencies who work with immigrant, low-income, and non-English speaking communities to inform people about the extent of the contamination in the river and the existing fish consumption advisories.[322] EPA should also consider developing specific strategies for reaching out to underrepresented communities, as it has done in other locations.

XII. Conclusion

For the aforementioned reasons, EPA must find that the remedy for the Hudson River Superfund Site is not protective of human health and the environment in its Final Second FYR. EPA s preliminary determination that the Hudson River remedy will be protective of human health and the environment is arbitrary and capricious and not supported by data and analyses by independent scientists, the Natural Resource Trustees for the Site, and New York State. The Final Second FYR must outline next steps toward additional remediation of the Upper Hudson to meet the remedial objectives within the timeframes set forth in the 2002 ROD. Moreover, the Final Second FYR must include a commitment to a full remedial investigation and feasibility study of the Lower Hudson River. A finding by EPA that the remedy is not protective will put the entire Hudson River on the path to quicker recovery, and will realize the Superfund program s goal of protecting the health of the people and wildlife living in and around the Hudson River.

[322] *See id*.

Attachment A

Comprehensive Five Year Review Guidance

United States Environmental Protection Agency
Office of Emergency and Remedial Response (5204G)
EPA 540-R-01-007
OSWER No. 9355.7-03B-P
June 2001

Superfund

Comprehensive Five-Year Review Guidance

Office of Emergency and Remedial Response
U.S. Environmental Protection Agency
Washington, D.C. 20460

URL: http://www.epa.gov/superfund/pubs.htm
Superfund Information 1-800-424-9346

Preface

The U.S. Environmental Protection Agency (EPA) is issuing this "Comprehensive Five-Year Review Guidance" to assist EPA Headquarters (HQ), Regional staff, and support agencies responsible for conducting five-year reviews under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). This guidance generally is intended to promote consistent implementation of the five-year review process.

Section 121 of CERCLA, as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA), requires that remedial actions which result in any hazardous substances, pollutants, or contaminants remaining at the site be subject to a five-year review. The National Oil and Hazardous Substances Pollution Contingency Plan (NCP) further provides that remedial actions which result in any hazardous substances, pollutants, or contaminants remaining at the site above levels that allow for unlimited use and unrestricted exposure be reviewed every five years to ensure protection of human health and the environment.

The Five-Year Review requirement applies to all remedial actions selected under CERCLA §121. Therefore, sites with CERCLA remedial actions may be subject to a five-year review. Consistent with Executive Order (EO) 12580, other Federal agencies are responsible for ensuring that five-year reviews are conducted at sites where five-year reviews are required or appropriate.

This guidance is designed and intended to:

- Provide an approach for conducting five-year reviews;
- Facilitate consistency across the ten EPA Regions;
- Clarify current policy; and
- Discuss roles and responsibilities of various entities in conducting or supporting five-year reviews.

This guidance supersedes the following directives on five-year reviews:

- Office of Solid Waste and Emergency Response (OSWER) Directive 9355.7-02 (May 23, 1991), *Structure and Components of Five-Year Reviews*;
- OSWER Directive 9355.7-02FS1 (August 1991), Factsheet: *Structure and Components of Five-Year Reviews;*

- OSWER Directive 9355.7-02A (July 26, 1994), *Supplemental Five-Year Review Guidance;* and

- OSWER Directive 9355.7-03A (December 21, 1995), *Second Supplemental Five-Year Review Guidance.*

In addition, this guidance updates and supersedes the text regarding five-year reviews in:

- OSWER 9200.1-23P (July 1999), *A Guide to Preparing Superfund Proposed Plans, Records of Decision, and Other Remedy Selection Decision Documents.*

Questions or comments concerning this guidance should be directed to the appropriate EPA Headquarters Regional Center.

Table of Contents

Exhibits

Appendices

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List of Acronyms

AOC	Administrative Order on Consent
ARARs	Applicable or Relevant and Appropriate Requirements
CA	Cooperative Agreement
CAG	Community Advisory Group
CD	Consent Decree
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CERCLIS	Comprehensive Environmental Response, Compensation, and Liability Information System
CFR	Code of Federal Regulations
CIC	Community Involvement Coordinator
CIP	Community Involvement Plan
DOD	Department of Defense
DOE	Department of Energy
EO	Executive Order
EPA	United States Environmental Protection Agency
ESD	Explanation of Significant Differences
FCOR	Final Close Out Report
FFA	Federal Facility Agreement
FFRRO	Federal Facilities Restoration and Reuse Office
FR	Federal Register
HASP	Health and Safety Plan
IAG	Interagency Agreement
IC	Institutional Control
IRIS	Integrated Risk Information System
LOAEL	Lowest Observed Adverse Effect Level
MCLs	Maximum Contaminant Levels
MNA	Monitored Natural Attenuation
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NOAEL	No Observed Adverse Effect Level
NPL	National Priorities List
O&M	Operation and Maintenance
OECA	Office of Enforcement and Compliance Assurance
OERR	Office of Emergency and Remedial Response
OSHA	Occupational Safety and Health Administration
OSWER	Office of Solid Waste and Emergency Response
OU	Operable Unit
PCOR	Preliminary Close Out Report
PRP	Potentially Responsible Party
RA	Remedial Action
RAGS	Risk Assessment Guidance for Superfund

RAO	Remedial Action Objective
RCRA	Resource Conservation and Recovery Act
RD/RA	Remedial Design/Remedial Action
RI/FS	Remedial Investigation/Feasibility Study
ROD	Record of Decision
RPM	Remedial Project Manager
SARA	Superfund Amendments and Reauthorization Act of 1986
SMOA	Superfund Memorandum of Agreement
SPIM	Superfund Program Implementation Manual
SSC	Superfund State Contract
TAG	Technical Assistance Grant
TBCs	To Be Considereds
USACE	United States Army Corps of Engineers
USCG	United States Coast Guard
UU/UE	Unlimited Use/Unrestricted Exposure
WasteLan	The Regional database related to CERCLIS

1.0 OVERVIEW

This chapter covers the purpose of five-year reviews, when are reviews required or appropriate, discontinuation of five-year reviews, and triggering actions for five-year reviews. This chapter also discusses the application of the Five-Year Review policy to sites with multiple operable units (OUs), division of large complex sites, pre- and post-Superfund Amendments and Reauthorization Act of 1986 (SARA) sites, Records of Decision (RODs), and deleted or partially deleted sites. You will also find information on Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) remedial actions (RAs), CERCLA remedial actions at sites with Resource Conservation and Recovery Act (RCRA) response, and interim/early remedial actions. Finally, the chapter discusses how no action or no further action RODs, monitored natural attenuation (MNA), and institutional controls (ICs) impact five-year reviews.

1.1 What is the purpose of a five-year review?

The purpose of a five-year review is to evaluate the implementation and performance of a remedy in order to determine if the remedy is or will be protective of human health and the environment. Protectiveness is generally defined in the National Contingency Plan (NCP) by the risk range and the hazard index (HI). Evaluation of the remedy and the determination of protectiveness should be based on and sufficiently supported by data and observations.

1.2 When are five-year reviews required or appropriate?

Five-year reviews should be conducted either to meet the statutory mandate under CERCLA §121(c) or as a matter of EPA policy. Consequently, five-year reviews are classified in this guidance as either "statutory" or "policy." The Five-Year Review requirement applies to all remedial actions selected under CERCLA §121. Regions may also conduct other five-year reviews at their discretion.

You should consider a number of factors when determining whether to conduct a five-year review, as discussed in the following two sections (see Sections 1.2.1 and 1.2.2). In general, five-year reviews are required whenever a remedial action results in hazardous substances, pollutants, or contaminants remaining on site. Under the Agency's interpretation contained in the NCP, the requirement in CERCLA §121(c) is triggered when remaining on-site hazardous substances, pollutants, or contaminants are above levels that allow for "unlimited use and unrestricted exposure." See 40 CFR §300.430(f)(4)(ii).

CERCLA §121(c) states the following:

> *If the President selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site, the President shall review such remedial action no less often than each five years after the initiation*

of such remedial action to assure that human health and the environment are being protected by the remedial action being implemented. In addition, if upon such review it is the judgment of the President that action is appropriate at such site in accordance with section [104] or [106], the President shall take or require such action. The President shall report to the Congress a list of facilities for which such review is required, the results of all such reviews, and any actions taken as a result of such reviews.

The Agency interpreted this requirement further in the National Contingency Plan (NCP) (40 CFR §300.430(f)(4)(ii)) which states:

If a remedial action is selected that results in hazardous substances, pollutants, or contaminants remaining at the site above levels that allow for unlimited use and unrestricted exposure, the lead agency shall review such action no less often than every five years after the initiation of the selected remedial action.

"Unlimited use and unrestricted exposure" (UU/UE) means that the selected remedy will place no restrictions on the potential use of land or other natural resources. In general, if the selected remedy relies on restrictions of land and/or groundwater use by humans and/or ecological populations to be protective, then the use has been limited and a five-year review should be conducted. For example, if a site is cleaned up to an industrial-use level, and/or other types of uses are restricted (*e.g.*, residential use), then, generally, UU/UE is not met. Exhibit 1-1, "Types of Actions Subject to Five-Year Reviews," provides examples of the types of remedial actions subject to statutory and policy reviews.

1.2.1 When is a statutory review required?

CERCLA requires five-year reviews if both of the following conditions are true:

- Upon completion of the remedial action, hazardous substances, pollutants, or contaminants will remain on site[1]; and
- The ROD for the site was signed on or after October 17, 1986 (the effective date of SARA[2]) and the remedial action was selected under CERCLA §121.

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[1] The general response authority of CERCLA §104(c)(4) applies to both removal and remedial actions. 104(c)(4). Also see 40 CFR §300.430(f)(4)(ii).

[2] Generally, SARA became effective the date it was passed (October 17, 1986). See Pub. L. 99-499, Oct. 17, 1986, 100 Stat. 1672.

Exhibit 1-1: Types of Actions Subject to Five-Year Reviews

If the action/site is . . .	then a review is . . .	and examples of actions or components of actions include . . .
a ***post-SARA*** remedial action that, upon completion, will leave hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure	required by ***statute***	– waste stabilization, fixation, or encapsulation on site – landfill cap or covers and slurry walls – institutional controls – sediment capping
a ***pre- or post-SARA*** remedial action that, upon completion, will not leave hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure, **but requires five or more years to complete**	conducted as a matter of EPA ***policy***, until cleanup levels are achieved, allowing unlimited use and unrestricted exposure	– long-term monitored natural attenuation – long-term groundwater pump and treatment – long-term bioremediation of groundwater or soil – other long-term remedies, such as soil washing and land farming – monitored natural recovery (sediments)
a ***pre-SARA*** remedial action that will leave hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure	conducted as a matter of EPA ***policy***	– waste stabilization, fixation, or encapsulation on site – landfill cap or covers and slurry walls – institutional controls
a ***removal*** **action that takes place at a site on *the NPL*** that leaves hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure and **where no remedial action has or will take place**	conducted as a matter of EPA ***policy***	– excavation and treatment where hazardous substances, pollutants, or contaminants remain on site above levels that allow for unlimited use and unrestricted exposure

1.2.2 When is a policy review appropriate?

Five-year reviews generally should be conducted as a matter of policy for the following types of actions:

- A pre- or post-SARA remedial action that, upon completion, will not leave hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure, but requires five years or more to complete;
- A pre-SARA remedial action that leaves hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure; or

- A removal-only site on the NPL where a removal action leaves hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure and where no remedial action has or will take place.

1.2.3 When should five-year reviews be completed?

The first five-year review generally should be completed and signed by the EPA Region within five years of the initial trigger date (see Sections 1.3.1 and 1.3.2). As a matter of policy, you should complete subsequent statutory or policy five-year reviews no later than five years following the signature date of the previous Five-Year Review report. Five-year reviews may be conducted earlier or more frequently than every five years, if needed, to ensure the protection of human health and the environment.

1.2.4 When can five-year reviews be discontinued?

Five-year reviews may no longer be needed when no hazardous substances, pollutants, or contaminants remain on site above levels that allow for unlimited use and unrestricted exposure. The basis for this finding should be documented in your final Five-Year Review report. When you make this determination prior to the first five-year review, you should record it in a document subject to public comment, such as a Proposed Plan or a Notice of Intent to Delete. When notice of five-year review discontinuation is given in a document other than a Five-Year Review report, the Region should submit a memorandum, signed by the Regional Administrator or his/her designee, to Headquarters. The memorandum should provide the reason for not conducting five-year reviews and cite the document in which this decision was made and supported.

1.3 When does the five-year review period begin?

The initiation or trigger date that starts the five-year review period depends upon whether the review is categorized as statutory or policy. However, the review should be completed within 5 years of its trigger date regardless of its category. Lead agencies may choose to conduct a five-year review earlier, or more frequently, than every five years to ensure the protection of human health and the environment. A discussion of the first and subsequent triggers for both statutory and policy review is provided below.

1.3.1 What actions first trigger a statutory review?

In accordance with CERCLA §121 and the NCP, a statutory review is triggered by the initiation of the first remedial action that leaves hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure. In cases where there are multiple remedial actions, the earliest remedial action that leaves hazardous

substances, pollutants, or contaminants on site should trigger the initial review, even if it is an interim remedial action.

For the purpose of a five-year review, a remedial action typically is initiated on the date of "actual RA on-site construction" or the "actual RA start" date for Federal facilities. The date of actual RA on-site construction generally corresponds to the date the contractor begins work at a site for the remedial action, typically the date of on-site mobilization. The definition of the "actual RA start" varies as outlined in the Superfund/Oil Program Implementation Manual (SPIM). For remedies where on-site mobilization may not occur, as a matter of policy, the date of the first monitoring event following ROD signature or the ROD signature itself should be used to trigger the five-year review period.

1.3.2 What actions first trigger a policy review?

A policy review initially should be triggered by the date that construction is completed at a site. The date of construction completion is generally the date of the Preliminary Close Out Report (PCOR) or the date of the Final Close Out Report (FCOR) for sites that do not have a PCOR. The PCOR or FCOR date also triggers the initial five-year review at NPL removal-only sites.

1.3.3 What are triggers for subsequent statutory and policy reviews?

After completion of the first statutory or policy five-year review, the trigger for subsequent reviews is the signature date of the previous Five-Year Review report. For reviews led by other Federal agencies, States, or Tribes, and where EPA has a concurrence role, the trigger for subsequent reviews corresponds to EPA's concurrence signature date of the preceding Five-Year Review report (see Sections 3.7.2 and 3.7.3).

1.4 How do five-year reviews apply to a site with multiple operable units?

Five-year reviews for sites with multiple OUs, as a matter of policy, should address all OUs and remedial actions that have been initiated at the time of the review, except for situations as described in Section 1.4.2. At the Regions' discretion, the five-year review may also include and consider areas of a site where no remedial action has been selected or initiated.

1.4.1 How is a multiple operable unit site categorized?

Five-year reviews for multiple OU sites can be categorized as either statutory or policy. As a matter of policy, a site is subject to a statutory review if any one of its initiated remedial actions is subject to a statutory review. A site is subject to a policy review if no initiated actions are subject to a statutory review and at least one action is subject to a policy review.

1.4.2 When is it appropriate to conduct a separate five-year review for different areas of a large and complex site?

At some large and complex sites, individual OUs, or groups of OUs, may have been treated as separate sites throughout the remedial process. Under these circumstances, Regions may continue to treat these areas separately and conduct individual five-year reviews for each area. Each five-year review should include the status and protectiveness determination of the five-year reviews conducted for the other areas of the entire site. Regions may choose to combine the separate reviews of different areas into a single five-year review prior to, or following, construction completion for the entire site. However, no area should be reviewed later than five years after its trigger date or previous review.

Actions within each area may trigger its respective statutory or policy review. However, in cases where site-wide construction completion will not be achieved for an extended period of time, the initial trigger date for a policy review should correspond to the date that physical construction is complete at the area under consideration. The Region should establish this date on a site-specific basis which should be based on the signature date of the Interim or Final RA Report.

1.4.3 How is a site with pre- and post-SARA RODs categorized?

At sites where there are both pre- and post-SARA RODs, the pre-SARA remedial actions are subject under this policy to post-SARA Five-Year Review procedures. For example, suppose a pre-SARA remedial action initially is subject to a policy review because hazardous substances, pollutants, or contaminants are permanently left on site above levels that allow for unrestricted use and unlimited exposure. If a post-SARA ROD is signed for that same site, a five-year review should be conducted, unless the post-SARA ROD selects a remedy that removes all on site hazardous substances, pollutants, or contaminants including the hazardous substances, pollutants, or contaminants left on site by the pre-SARA action. In such cases, the original five-year review schedule should be maintained as a matter of policy. If no schedule has been established, the post-SARA trigger should be utilized.

1.5 What are some other considerations for five-year reviews?

This section discusses other considerations (*i.e.*, deletions, RCRA responses, interim and early remedial actions, no action or no further action RODs, monitored natural attenuation, and institutional controls) that may affect the need for and conduct of five-year reviews.

1.5.1 Are five-year reviews required for a site that has been deleted or partially deleted from the NPL?

It is EPA's policy that the Five-Year Review requirement is independent of and unaffected by the deletion process.[3] Consistent with the NCP, a site can be deleted or partially deleted from the NPL once the deletion criteria have been satisfied. If a site has been deleted or is in the process of being deleted, your Five-Year Review report should address the status of any deletion action. Five-year reviews continue as needed after deletion.

1.5.2 Are five-year reviews required for a site with a RCRA response?

In 1996, EPA established a policy to defer some CERCLA cleanup activities to the RCRA program. The policy is outlined in the memorandum "Coordination Between RCRA Corrective Action and Closure and CERCLA Site Activities."[4] This policy allows site managers to defer cleanup activities for all or part of a site from CERCLA to RCRA (or vice versa). If a site is deferred to RCRA prior to being placed on the NPL, or is deleted from the NPL prior to the selection of the remedy and deferred to RCRA for corrective action, you do not need to conduct a five-year review.

In cases where full deferral is not appropriate, it is possible that both RCRA and CERCLA authorities will be used to address a site. When a RCRA action is included as a part of a CERCLA action, the RCRA action should be included in the five-year review as a matter of policy, if a five-year review is required or appropriate.

1.5.3 How is a site that has an interim or early remedial action handled?

Regions should conduct five-year reviews for interim or early actions selected under CERCLA §121 consistent with Section 1.2 of this guidance.[5] For instance, Regions should conduct a review if an alternate water supply is installed and hazardous substances, pollutants, or contaminants remain on site above levels that allow for unlimited use and unrestricted exposure. If a subsequent action reduces the hazardous substances, pollutants, or contaminants on site to

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[3] In 1991, EPA clarified its policy on whether a site deleted from the NPL is subject to a five-year review. See "Notice of Policy Change," 56 FR 66601 (December 24, 1991). In appropriate circumstances, a site does not need to be kept on the NPL solely for the purposes of conducting five-year reviews (See 55 Fed Reg at p. 8699).

[4] The memorandum "Coordination Between RCRA Corrective Action and Closure and CERCLA Site Activities" was issued by Steven A. Herman, Assistant Administrator, Office of Enforcement and Compliance Assurance, and Elliott P. Laws, Assistant Administrator, OSWER (September 24, 1996).

[5] Interim and Early actions are defined in Chapter 8 in *A Guide to Preparing Superfund Proposed Plans, Records of Decision, and other Remedy Selection Decision Documents*. EPA 540-R-98-031, OSWER 9200.1-23P (July 1999)

levels that allow unlimited use and unrestricted exposure, then reviews may be discontinued (see Section 1.2.4).

1.5.4 How is a site that has a no action or a no further action ROD handled?

Consistent with Section 1.2, Regions should conduct a five-year review for a remedy where a no action or no further action ROD leaves hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure. For example, as a matter of policy Regions should conduct a review for an NPL site with a no action ROD where a removal-only action leaves hazardous substances, pollutants, or contaminants on site above levels that allow for unlimited use and unrestricted exposure, or where groundwater monitoring or other types of monitoring of contamination above action levels is the only remedial action selected. However, no five-year review may be needed when monitoring is used only to verify absence of contamination.

1.5.5 How is a ROD that includes monitored natural attenuation handled?

CERCLA §121 remedies relying on monitored natural attenuation or natural attenuation may be subject to five-year reviews consistent with Section 1.2. If monitored natural attenuation is included in a no action or a no further action ROD, then that ROD is not considered to be no action or no further action and therefore, Regions may need to conduct a five-year review, consistent with this guidance.

1.5.6 How is a ROD that includes institutional controls handled?

Institutional controls may be part of remedies selected under CERCLA §121 and consistent with Section 1.2 of this guidance may be subject to five-year reviews.[6] If institutional controls are included in a no action or a no further action ROD, and protectiveness relies on the institutional control, then that ROD is not considered to be no action or no further action and therefore, Regions may need to conduct a five-year review.

[6] Regions should refer to OSWER 9355.0-74FS-P, dated September 2000, entitled *Institutional Controls: A Site Manager's Guide to identifying, evaluating and selecting Institutional Controls at Superfund and RCRA Corrective Action Cleanups* for further information on institutional controls and remedy selection.

2.0 ROLES AND RESPONSIBILITIES FOR EPA, STATES, TRIBES, AND OTHER FEDERAL AGENCIES

This chapter discusses the roles and responsibilities of U.S. Environmental Protection Agency (EPA), other Federal agencies, State agencies, and Tribes, in conducting five-year reviews. As a general matter, for remedies selected under Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) §121, except at non-NPL Federal facility sites, EPA has the ultimate authority for determining whether a remedy subject to the Five-Year Review requirements in CERCLA §121(c) is protective. The National Oil and Hazardous Substances Pollution Contingency Plan (NCP) addresses, in general, the involvement of State agencies and Tribes in CERCLA actions in 40 CFR §300.515 and §300.520. Finally, CERCLA §120 and Executive Order (EO) 12580[7] address the responsibilities of Federal agencies in carrying out CERCLA cleanups.[8]

2.1 What are the roles of the lead and support agencies?

Under the NCP, the lead agency provides for the remedial project manager (RPM) "to plan and implement [the] response action;"[9] a response action would include conducting a five-year review. A support agency "furnish[es] necessary data to the lead agency, reviews response data and documents, and provides other assistance."[10] The NCP also encourages appropriate State and Tribal involvement for Fund-financed and Enforcement-lead remedial actions (see 40 CFR §300.515 and §300.520). Where the State or Tribe is the lead agency, the NCP provides that EPA concurrence is needed on remedy selection decisions (see 40 CFR §300.515(e) and §300.520).

The relative roles and responsibilities for lead and support agencies can vary significantly depending on ability, resources, and legal authorities. There are a number of documents that can be used to specify roles and responsibilities of lead and support agencies. Some of these are general in scope, while others are more narrow in scope and apply solely to a specific site. General instruments include Superfund Memoranda of Agreement (SMOAs), Cooperative Agreements (CAs), and Superfund State Contracts (SSCs). Normally, SMOAs are general, non-site-specific agreements that EPA uses to define roles and interactions in conducting a response action. EPA uses CAs to transfer Superfund monies to States or Tribes for response activities. SSCs are used to identify EPA and State or Tribal roles and responsibilities required under

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[7]Executive Order No. 12580 of January 23, 1987, as amended on August 28, 1996.

[8]As discussed in section 2.4, State enforcement-lead cleanups are not subject to this guidance.

[9]See 40 CFR §300.5.

[10]Id.

CERCLA §104. Site-specific agreements include Consent Decrees, Administrative Orders on Consent, and Federal Facility Agreements (FFAs). If no SMOA, SSC, or CA is available, a letter of agreement should be written to define roles and responsibilities for the five-year review, consistent with the NCP (see 40 CFR §300.515). Wherever possible, the specific roles and responsibilities regarding the conduct of a five-year review should be detailed in a single document to avoid confusion and disputes at a later date.

2.1.1 What are the roles of the lead agency?

The lead agency conducts the five-year review, prepares the Five-Year Review report, and submits the report to the support agency for review and comment. The lead agency is also responsible for conducting community involvement activities and for ensuring that recommendations and follow-up actions identified during five-year reviews are completed. Generally, funding for five-year reviews is provided by EPA for Fund-financed sites, Potentially Responsible Parties (PRPs) for Enforcement-lead sites (through appropriate mechanisms), and by other Federal agencies or departments for Federal facility sites.

Where EPA is the lead agency pursuant to 40 CFR §300.515, the Region should submit a copy of its final Five-Year Review report to EPA Headquarters (HQ) within 10 days of signature, and provide copies to the support agency and site information repositories. Where the State or Tribe is the lead agency, pursuant to 40 CFR §300.515, the State should submit a copy of the final Five-Year Review report to the Region; once the Region has concurred, the Region should provide a copy to EPA HQ within 10 days of signature, to any other support agencies, and to site information repositories. Where another Federal agency or department is the lead agency, pursuant to CERCLA §120 and EO 12580, the Federal agency or department should submit a copy of the final Five-Year Review report to the Region; once the Region has concurred, the Region should provide a copy to HQ within 10 days of signature, to any other support agencies, and to site information repositories.

2.1.2 What are the roles of the support agency?

The role of the support agency is to participate in the review process, if requested, and review and comment on the Five-Year Review report. Where the State or Tribe is the lead agency for a response action (such as conducting a five-year review), the NCP provides that it must obtain EPA's concurrence (see 40 CFR §300.515(e)).

The lead agency should give the support agency an adequate opportunity to participate in the five-year review process and to review and comment on the draft Five-Year Review report before it is finalized. When there is more than one support agency involved, time allowances for review and comment should be the same for all support agencies who choose to participate in the review process. The amount of time that a support agency will have to review the Five-Year Review report should be documented in the SMOA, SSC, CA, or other agreement documents,

but should not be less than review times for other remedy decision documents (see 40 CFR §300.515(h)(3)). The goal should be to resolve any concerns of support agencies before drafting the final report. In any case, the support agency or agencies may provide written comments on the Five-Year Review report. Lead and support agencies should work together throughout the five-year review process to ensure that concerns are resolved in a timely manner, and to the extent practicable, prior to finalizing the Five-Year Review report.

2.2 Who conducts the review at a Fund-financed site?

At Fund-financed sites, the ultimate responsibility for the protectiveness determination rests with EPA. As described in Section 2.1, EPA may be the lead or support agency.

Regions may acquire the services of a contractor or establish agreements with other agencies (*e.g.*, the U.S. Army Corps of Engineers) to perform studies, conduct investigations, and/or develop draft Five-Year Review reports. In all cases, Regions should ensure the quality and completeness of review activities and the content of the final Five-Year Review report.

2.3 What if a site is an Enforcement-lead site?

At CERCLA Enforcement-lead sites, the ultimate responsibility for the quality and completeness of review activities and the content and protectiveness determinations of the Five-Year Review report rests with EPA. As described in Section 2.1, EPA may be the lead or support agency.

At sites in which EPA is the lead agency Regions may acquire the services of a contractor or establish agreements with other agencies (*e.g.*, the U.S. Army Corps of Engineers) to perform studies, conduct investigations, and/or develop draft Five-Year Review reports.

PRPs or PRP-hired contractors may perform certain support activities (*e.g.*, data collection, studies or analysis) according to provisions of an enforceable agreement.

2.4 What if site activities are led by a State or Tribe?

As described in Section 2.1, States and Tribes can be the lead agency in carrying out a five-year review. In those cases, States or Tribes should ensure the quality and completeness of review activities and the content of the final Five-Year Review report, prior to submitting the report to the Region for EPA's concurrence. When a State or Tribe provides EPA with a Five-Year Review report, EPA can choose to concur with the report and protectiveness statements or make its own protectiveness determinations.

Where a State or Tribe conducts a cleanup using its own legal authorities (*e.g.*, State enforcement action under a CERCLA-equivalent State law), the remedy is not selected pursuant to CERCLA §121 and is not subject to the Five-Year Review requirement.

Exhibit 2-1 provides an overview of the typical roles of different parties for each type of response action.

Exhibit 2-1: Typical Roles in the Five-Year Review Process*

If the response action is...	at...	under...	then conducting the review is the responsibility of...	with funding by...	and with the EPA Region...
Fund-financed	a site	CERCLA §121,and CERCLA §104	the lead agency; when the lead agency is a State or Tribe, EPA concurs;	Superfund	making or concurring with the protectiveness determination.
Enforcement -lead	a site	CERCLA §104 and §121, along with a Consent Decree or other enforcement document	the lead agency; when the lead agency is a State or Tribe, EPA concurs. (PRPs may be allowed to provide certain support for five-year reviews);	PRPs	making or concurring with the protectiveness determination.
Other Federal agency or department (*e.g.*, led by Department of Defense, Department of Energy or Department of the Interior)	a Federal facility NPL site	CERCLA §104, §120 and §121, Executive Order 12580, and a Federal Facility Agreement	the respective Federal agency or department	the respective Federal agency or department	making or concurring with the protectiveness determination.
Other Federal agency	a Federal facility non-NPL site	CERCLA §104 and §121, and Executive Order 12580	the respective Federal agency or department	the respective Federal agency or department	commenting on the protectiveness determination (if requested).

Note: * The scenarios presented in the exhibit are not all inclusive. Regions should determine the respective roles in the five-year review process when other circumstances exist. EPA does not have a role in five-year reviews at non-NPL Federal facility sites; however, EPA Regions may comment or be asked to comment on a site-specific basis.

2.5 What if site activities are led by another Federal agency or department?

CERCLA §104, §120, and §121 identify functions and responsibilities vested in the President for undertaking response efforts and coordinating all other efforts at the scene of a

release on or from Federally-owned property (or vessels). The President, in EO 12580, delegates some of these responsibilities to the respective Federal agencies and departments for Federally-owned or Federally-operated facilities over which these lead agencies have jurisdiction, custody, or control.

Therefore, at sites where activities are led by another Federal agency or department, the Federal agency or department has responsibilities for selecting remedies and implementing the remedial actions, and for conducting all required five-year reviews. The Federal agency or department is responsible for planning, coordinating, funding, and conducting five-year reviews and for making protectiveness determinations upon conclusion of each five-year review. Federal agencies or departments are encouraged to have EPA, States, and Tribes participate and comment throughout the five-year review process, as appropriate. Federal agencies or departments are also responsible for initiating resolutions to issues and following up on all recommendations that result from these five-year reviews. Federal agencies or departments may not adopt or utilize guidelines that are inconsistent with EPA's Five-Year Review guidance or certain other EPA guidance, as specified in CERCLA §120(a)(2).

- ***Federal facility sites that are listed on the NPL*** – EO 12580 paragraphs 2(d) and (g) delegate remedial responsibilities to the Department of Defense (DOD) and the Department of Energy (DOE), and to EPA, respectively. In addition, at all Federal facility NPL sites, CERCLA §120 requires Federal agencies or departments to perform remedial investigation and feasability studies (RI/FS) (see CERCLA §120(e)(1)), to enter into Inter-Agency Agreements (IAGs) (frequently called Federal Facility Agreements), and to initiate remedial actions, subject to EPA concurrence. Therefore, five-year reviews are conducted by the Federal agency or department that has jurisdiction, custody, or control, but EPA retains final authority over whether the five-year reviews adequately address the protectiveness of remedies. EPA will either concur with the final Federal agency or department protectiveness determination, or EPA may provide independent findings. Disputes which arise related to protectiveness determinations or independent findings by EPA may be resolved on a site-specific basis through formal dispute resolution procedures, typically established in FFAs. Exhibits 2-2 and 2-3 and Sections 2.5.1 and 2.5.2 discuss Federal facility NPL sites and FFAs in more detail.

- ***Non-NPL Federal facilities*** – EO 12580, paragraphs 2(d) and (e), give remedial responsibilities, and therefore five-year review responsibilities, to the Federal agency or department having jurisdiction, custody, or control. EPA may also be asked to comment, to the extent practical, on five-year reviews or protectiveness determinations at non-NPL Federal facilities. Section 2.5.3 discusses non-NPL Federal facilities in more detail.

Exhibit 2-2 below provides an overview of relevant EO 12580 sections and their applicability.

Exhibit 2-2: Federal Responsibilities Under Executive Order 12580

In EO 12580 section(s)...	the President delegates to...	certain remedial functions and responsibilities in CERCLA section(s)...	and those remedial functions and responsibilities at Federal facilities generally pertaining to...
2(b)	***EPA*** (in consultation with the National Response Team)	121(f)(1)	promulgation of regulations assuring substantial and meaningful State involvement (in initiation, development, and selection of remedial actions to be undertaken in the State).
2(d)	***DOD, DOE,*** (subject to the requirements described in CERCLA §120)	104(a), 104(b), 104(c)(4), and 121	selecting and taking NPL and non-NPL[1] [2] remedial actions, which includes both conducting five-year reviews and making protectiveness determinations (with EPA concurrence at NPL sites).
2(e)	***Federal Departments/ Agencies*** (for non-NPL Federal facility sites.)	104(a), 104(b), 104(c)(4), and 121	selecting and taking non-NPL remedial actions, which includes both conducting five-year reviews and making protectiveness determinations.
2(g)	***EPA*** (subject to the above delegations)	104(a), 104(b), 104(c)(4), and 121	selecting and taking NPL remedial actions, which includes conducting five-year reviews and making protectiveness determinations at Fund-lead and Enforcement-lead NPL sites.

Note: [1] EPA does not have a role in five-year reviews at non-NPL Federal facility sites; however, EPA Regions may be asked to comment on a site-specific basis.

[2] In addition to the EO 12580 delegation of remedy selection and remedial action responsibilities to all Federal agencies and departments for non-NPL Federal facility sites, CERCLA §120(e) establishes remedy selection and remedial action responsibilities for Federal agencies and departments for all Federal facility NPL sites, as well. For example, CERCLA §120(e)(2) requires Federal agencies and departments to enter into NPL IAGs (frequently called FFAs) with EPA (States may participate.) CERCLA §120(e)(4) requires FFAs to address selection of remedies and completion of remedial actions at Federal facility NPL sites. FFAs, where applicable, should specify the procedures to be followed with respect to conducting five-year reviews at Federal facility NPL sites.

The following subsections detail responsibilities for conducting five-year reviews at sites led by other Federal departments and agencies.

2.5.1 What is the purpose of FFAs at other Federal agency NPL sites?

CERCLA §120(e)(2) requires that EPA sign an IAG (frequently called an FFA) with responsible Federal agencies or departments to detail respective roles and responsibilities for remedial actions at NPL sites. CERCLA §120(e)(1) requires Federal agencies or departments to conduct remedial investigations in consultation with EPA and appropriate State authorities at Federal facility NPL sites. Most Federal facility NPL sites will have site-specific roles and responsibilities specified in the FFA. CERCLA §120(e)(4) requires FFAs to include selection of remedies, completion of remedial actions, and arrangements for long-term operation and maintenance of the facility. Therefore, the procedures for conducting five-year reviews and making protectiveness determinations fall within the scope of FFAs. FFAs should specify in detail the procedures governing five-year reviews at Federal facility NPL sites.

OSWER Directive 9320.0-75 (November 29, 1996), "Federal Facilities Streamlined Oversight Directive" reiterates EPA's responsibility for oversight of remedial activities at Federal facility NPL sites. States and Tribes, as regulators, may also have an oversight role, defined in the FFA, at a facility. Exhibit 2-3 describes the topics to be addressed in an FFA.

Exhibit 2-3: Federal Facility Agreements and Five-Year Reviews

CERCLA § 120(e)(2) requires that the relevant Federal agency or department must enter into an FFA (IAG in the statute) with EPA within six months after EPA's review of the Remedial Investigation/Feasibility Study (RI/FS) is completed. States may be signatories to the FFA and under CERCLA §120 (f) must be included in the decision-making process at Federal facility NPL sites. Whenever a Federal facility is located on Tribal lands, the appropriate Tribal government should be involved.

CERCLA §120(e)(4), in the case of schedules, requires that the EPA/DOD and EPA/DOE Model FFA contain procedures for the submission and review of documents, schedules of cleanup activities, and provisions for dispute resolution. Regions should examine FFAs with respect to the performance of five-year reviews to clarify:

- Roles, responsibilities, and milestones;
- Arrangements for long-term operation and maintenance of the facility; and
- Opportunities for public involvement.

For Federal facilities only, EPA considers Five-Year Review reports to be stand-alone primary documents or part of another related primary document that should have an enforceable schedule within the framework of the FFA. Where EPA enters into an FFA, the agreement should include all site-specific Five-Year Review requirements, such as provisions for reviews, public participation, and addressing or resolving issues.

Where the roles and responsibilities for conducting five-year reviews and making protectiveness determinations are not specified in an FFA (for example, the FFA may not have been signed, or it may be silent or unclear with respect to five-year reviews), then the parties should rely on this guidance for fulfilling EPA's obligations under CERCLA §120 and §121, including making protectiveness determinations. Five-year review requirements should be

identified early in the FFA process, so that the parties to the Agreement have clearly defined roles and responsibilities for implementing CERCLA §121(c) with respect to five-year reviews. However, consistent with CERCLA §120(g), FFAs cannot re-delegate EPA's final authority over whether the five-year reviews adequately address the protectiveness of remedies.

2.5.2 What is EPA's role at NPL sites under the jurisdiction of another Federal agency or department?

CERCLA §120 and EO 12580 provide the basis for EPA's oversight role at other Federal agency NPL sites. This role includes the following:

- Assisting in the determination of cleanup remedies or potentially selecting the remedies, in consultation with the lead agency and appropriate State authorities, beginning at the commencement of remedial investigations and feasibility studies;
- Ensuring that Federal agencies or departments appropriately consider all relevant guidance and policies that EPA determines are appropriate;
- Ensuring compliance with signed FFAs; and
- Determining that decisions protect human health and the environment and that such decisions are adequately supported in the Five-Year Review report (whether as a stand-alone primary document or part of a related primary document).

EPA is not responsible for conducting five-year reviews at Federal facility NPL sites. However, EPA's final remedy selection authority at Federal facility NPL sites requires that EPA retain final authority to make protectiveness determinations. Accordingly, EPA will either concur with any protectiveness determinations to ensure protection of human health and the environment, consistent with EPA's statutory and regulatory authorities or EPA may provide independent findings. EPA Regions should review Federal facility NPL Five-Year Review reports (whether as a stand-alone primary document or part of a related primary document) and protectiveness determinations for consistency with this guidance and adequacy of the supporting basis, and should participate or comment throughout the five-year review process, as appropriate.

2.5.3 What is EPA's role at a non-NPL site under the jurisdiction of another Federal agency or department?

EO 12580 paragraphs 2(d) and (e)(1) delegates the authority in CERCLA §104 and §121 to the Federal agencies or departments for selecting and conducting remedial actions addressing releases or threatened releases at sites that are not on the NPL. Consistent with CERCLA §121 and this guidance, Federal agencies or departments should conduct five-year reviews for all CERCLA non-NPL remedial actions that require a review (discussed in Section 1.2.1 of this

guidance). It is EPA's expectation that Federal agencies or departments will also conduct five-year reviews as a matter of policy at sites that would be subject to policy reviews if they were on the NPL (see Section 1.2.2). EPA does not have a statutorily defined role in five-year reviews at non-NPL Federal facility sites. However, where EPA has had active and substantial involvement at a non-NPL Federal facility, or where agencies, States, Tribes, or citizens seek EPA comment on five-year reviews conducted at a non-NPL Federal facility, EPA may, to the extent practicable on a site-specific basis, comment on five-year reviews and protectiveness determinations made by other Federal agencies or departments at non-NPL Federal facilities, and/or provide independent findings, where applicable.

2.5.4 What are States' roles at non-NPL sites under the jurisdiction of a Federal agency or department?

Consistent with CERCLA §120(a)(4), at non-NPL Federal facilities sites, States generally have remedial oversight responsibilities and should be provided with adequate opportunity to participate in the five-year review process and to review the draft Five-Year Review document before it is finalized.

2.5.5 What happens when Federal agencies or departments transfer real property?

In instances of Federal-to-Federal transfer of jurisdiction, custody, or control of real property, the Federal agency or department having initiated CERCLA remedial actions generally should conduct any required or appropriate five-year reviews. Alternatively, the lead agency may assure that reviews are conducted by entering into reliable site-specific agreements with the Federal agency or department gaining control of the property, where those arrangements remain consistent with CERCLA and EO 12580. In instances of deed transfer of Federal property to third parties, the Federal agency or department having initiated CERCLA remedial actions generally should conduct any required or appropriate five-year reviews, unless other reliable site-specific procedures are arranged with the transferee (or others), and those arrangements remain consistent with CERCLA and EO 12580. Generally, however, the ultimate responsibility for conducting five-year reviews should remain with the Federal agency or department that initiated the CERCLA remedial actions.

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3.0 COMPONENTS OF THE FIVE-YEAR REVIEW PROCESS

This chapter discusses components of the five-year review process, including notifying potentially interested parties, developing a review schedule, establishing a review team, involving the community, and signing and submitting the Five-Year Review report. Data and other site-specific information that form the foundation for the technical assessment of the remedy at the time of the five-year review are discussed in this chapter, including data and document review, site interview, site inspection, and components of a Five-Year Review report.

3.1 Who is notified when planning the five-year review?

In the initial planning stages of the five-year review, all potentially interested parties should be notified that the five-year review will be conducted. This notification may include States and/or Tribes, appropriate representatives of the community, local officials, Federal and/or State Trustees for Natural Resources (Trustees)[11], appropriate EPA offices, and the Community Involvement Coordinator (CIC) for the site. Potentially responsible parties should be notified for Enforcement-lead sites.

3.2 How should I develop a review schedule?

You should develop a review schedule to meet the appropriate five-year review date of completion. The review schedule should allow sufficient time for each component of the five-year review process, including document review, site inspection, interviews, the assessment of the protectiveness of the remedy (see Chapter 4), and report development and final submission. You should incorporate into the five-year review schedule appropriate time for internal and inter-agency review and comment periods, community involvement activities, if needed, and finalizing the report with all required signatures.

3.3 How should I establish a review team?

You should determine the appropriate level of assistance and team structure. For some reviews, the project manager may be the only member of the team, consulting with technical experts as necessary. For other reviews, a multi-disciplinary team may be needed to adequately review the protectiveness of the remedy. Once team members are identified their roles should be clearly defined. Communication among team members, agencies, and organizations is critical to ensure that all parties remain informed throughout the entire five-year review process.

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[11] OSWER Directive 9200.4-22A *CERCLA Coordination with Natural Resource Trustees*, dated July 31, 1997.

Exhibit 3-1 below provides examples of potential team members for a five-year review.

Exhibit 3-1: Potential Members of the Five-Year Review Team

- Project Manager (EPA, State, Tribal, DOD, DOI)
- Regional Biological Technical Assistance Groups (BTAGs)
- Federal and State Natural Resource Trustees
- Community Involvement Coordinator (CIC)
- State and/or local regulatory agency representatives
- Tribal representatives
- TAG representatives and/or community representatives
- Other Federal agency representatives (*e.g.*, U.S. Army Corps of Engineers, U.S. Fish and Wildlife Service, Agency for Toxic Substances and Disease Registry, U.S. Geological Survey, National Oceanic and Atmospheric Administration)
- Technical Experts
  - Construction representative
  - Engineers (*e.g.*, civil, geo-technical, structural, chemical, process)
  - Hydrogeologist
  - Chemist
  - Risk assessor
  - Biologist
  - Ecologist/ecological risk assessor
  - Attorney/legal advisor
  - Environmental regulatory specialist

3.4 How should I involve the community?

You should begin working with the site's CIC during the initial planning stages of your five-year review to determine the appropriate level of community involvement. At a minimum, your community involvement activities during the five-year review should include notifying the community that the five-year review will be conducted, notifying the community that the five-year review has been completed, and providing the results of the review to the local site repository (see Exhibit 3-2).

Together with the CIC, you should consider conducting additional community involvement activities at high profile sites, those with significant public interest, and any other sites for which the Region determines a need for additional community involvement activities. This may include notifying local public officials, including the primary local health agency, and the leadership of any relevant neighborhood and civic groups. (For ideas on notifying the public see *Publishing Effective Public Notices*, which is part of the CIC Toolkit (Web address: http://www.epa.gov/superfund/action/community/index.htm).)

In addition to this notification, you may also wish to interview several community members, at least some of whom live or work near the site, to get their views about current site conditions, problems, or related concerns. If there was or is a Community Advisory Group or a

Technical Assistance Grant related to the site, representatives of these groups should be briefed at the outset of the five-year review process, and, if requested, at other appropriate points. You may also want to consider appropriate ways, such as public meetings or an opportunity for submitting written comments, to get broader public involvement. For further information on community involvement during the five-year review process, see Appendix A, "Community Involvement."

Exhibit 3-2: Notification Requirements for Five-Year Reviews

At the beginning: Your notice to the community that a five-year review will be conducted should identify:

- The site name, its location and web address (if available);
- The lead agency conducting the review;
- A brief description of the selected remedy;
- A summary of contamination addressed by the selected remedy;
- How the community can contribute during the review process;
- A contact name and telephone number for further information; and
- The scheduled completion date of the five-year review.

At the end: Your notice to the community that a five-year review has been completed should include:

- The site name, its location and web address (if available);
- The lead agency conducting the review;
- A brief description of the selected remedy;
- A summary of contamination addressed by the selected remedy as provided in the initial notice;
- A brief summary of the results of the five-year review;
- The protectiveness statement(s);
- A brief summary of data and information that provided the basis for determining protectiveness, issues, recommendations, and follow-up actions directly related to the protectiveness of the remedy;
- Location(s) where a copy of the five-year review can be obtained or viewed (including site repositories);
- A contact name and telephone number where community members can obtain more information or ask questions about the results; and
- The date of the next five-year review or a statement and supporting rationale that five-year reviews will no longer be required.

3.5 What data do I need to evaluate the remedy?

Data and other pertinent site specific information that you should review include sampling and monitoring plans and results from monitoring activities, operation and maintenance (O&M) reports or other documentation of remedy performance, including previous Five-Year Review reports. These are the primary bases of the technical analyses and subsequent protectiveness determination(s). The type and quality of data are essential to your five-year review and its findings and conclusions. You may collect these types of data through a variety of means, including document review, interviews, and a site inspection. You also may need to conduct supplemental sampling or collect other data.

3.5.1 How are documents reviewed?

A review of documents is one of the first steps in the five-year review process. You are responsible for gathering all relevant documents, data, and other information in support of the five-year review. Generally, for an initial five-year review, this may require you to evaluate record keeping and the location of pertinent data and information. In cases where records are difficult to obtain, you should establish appropriate record keeping procedures to minimize future efforts needed to gather all necessary documents for subsequent five-year reviews.

Documents should be reviewed to obtain relevant information and data concerning a response action from which to base an assessment of its performance. The scope of the review is dependent on the complexity of the remedy(s) and the stage of remedy construction. You may need to review various documents to obtain the necessary information, including those for remedy decisions (*e.g.*, Records of Decision (RODs), Explanation of Significant Differences (ESDs)), enforcement (*e.g.*, Consent Decrees (CDs), Administrative Orders on Consent (AOCs)), site investigations (*e.g.*, remedial investigation/feasibility study (RI/FS)), design (*e.g.*, remedial design (RD)) and construction (*e.g.*, Preliminary Closeout Reports (PCOR), remedial action (RA) reports), and remedy performance and post-closure. (See Appendix B, "Document Review," for a more complete discussion of document review for the five-year review).

Your review team should be familiar with appropriate site-specific data and information including the items listed below:

- Remedial action objectives and cleanup levels, as specified in the ROD and other decision documents;
- Remedial action design and remedial action construction;
- O&M status;
- Implementation of institutional controls;
- Changes that affect the validity of cleanup levels (*e.g.*, standards identified as Applicable or Relevant and Appropriate Requirements (ARARs), "to be considereds" (TBCs), assumptions about contaminant characteristics and potential exposure); and
- Data supporting the effectiveness of the remedy in meeting cleanup levels and remedial action objectives.

3.5.2 How should I conduct interviews?

Interviews should be conducted, if necessary, to provide additional information about a site's status. The scope of interviews should be tailored to the remedy evaluation on a site-specific basis. Those interviewed may include the site manager; site personnel; Federal, State, and Tribal regulatory authorities; local officials; community action groups or associations;

residents and businesses located near the site; and other pertinent organizations or individuals. At an Enforcement-lead site, the lead agency should conduct the interviews. A Potentially Responsible Party (PRP) generally should not conduct interviews because there is a potential for a conflict of interest (see Appendix C, "Five-Year Review Interviews," for additional information). For Federal facility sites, a State and/or EPA representative may wish to be present at and/or participate in conducting interviews.

3.5.3 How should I conduct site inspections?

Your five-year review should include a recent site inspection. For purposes of conducting site inspections for five-year reviews, "recent" generally means no more than nine months from the expected signature date of the review. The review should be performed by objective parties without bias or preconceived views or conclusions about the remedy and conditions at the site. Site inspections are conducted to provide information about a site's status and to visually confirm and document the conditions of the remedy, the site, and the surrounding area.

At an Enforcement-lead site, the lead agency should conduct the site inspection. A PRP generally should not conduct the site inspection because of the potential for a conflict of interest. At Federal facility sites, a State and/or EPA representative may wish to be present and/or participate in conducting site inspections.

Appendix D, "Five-Year Review Site Inspection Checklist," may serve as your guide for planning and documenting a site inspection for containment, groundwater, and surface water remedies. Using this checklist should aid you in the planning and documentation of the site inspection. Therefore, you may adapt this checklist for other types of remedies or use other site inspection tools and checklists that have been developed by others for this purpose. You can find other checklists by accessing the web site: http://www.frtr.gov/optimization/general/ and clicking on " USACE Remediation System Evaluation Checklists."

3.6 What should I include in Five-Year Review reports?

In your Five-Year Review report, you should present the findings and conclusions of the review, including recommendations, follow-up actions to issues, and protectiveness determination(s). The report should also contain the data and information necessary to support all findings and conclusions.

Where your review only addresses a portion of a site, the report should provide a summary of the status of other operable units (OUs) and/or the remainder of the site. Similarly, for sites where you conduct a separate five-year review for different areas of a large or complex site (see Section 1.4.2), you should provide a summary of the status of the other areas of the site in your Five-Year Review report. Additionally, if you receive written comments on the Five-Year Review report from support agencies and/or the community (*e.g.*, States, Tribes, other

Federal agencies or departments, local governments, citizens, PRPs, other interested parties), you should attach a copy of these comments to the report.

A suggested "Five-Year Review Report Template" and "A Sample Five-Year Review Report" are provided in Appendices E and F, respectively. Exhibit 3-3 summarizes the recommended contents of a Five-Year Review report.

Exhibit 3-3: Contents of a Five-Year Review Report

The following report sections...	should include these topics when appropriate:
I. Introduction	– the purpose of the review – who conducted the review – when the review was initiated and completed – whether it is the first review or a subsequent review at the site – status of other five-year reviews, OUs, and/or areas of the entire site
II. Site Chronology	– dates of major events (such as the initial discovery of contamination, NPL listing, decision and enforcement documents, start and completion of remedial and removal actions, construction completion, and prior five-year reviews)
III. Background	– physical characteristics – land and resource use – history of contamination – initial response – summary of basis for taking action
IV. Remedial Actions	– remedy selection – remedy implementation – system operations/O&M
V. Progress Since Last Review (as applicable)	– protectiveness statements from last review – status of recommendations and follow-up actions from last review – results of implemented actions, including whether they achieved the intended purpose – status of any other prior issues
VI. Five-Year Review Process	– notification of potentially interested parties of start of review – identification of five-year review team members – components and schedule of your five-year review – document review – data review and evaluation – community notification – other community involvement activities – site inspection – site interviews

Exhibit 3-3: Contents of a Five-Year Review Report

The following report sections...	should include these topics when appropriate:
VII. Technical Assessment	*Question A: Is the remedy functioning as intended by the decision documents?* – remedial action performance and monitoring results – system operations/O&M – costs of system operations/O&M – opportunities for optimization – early indicators of potential remedy problems – implementation of institutional controls and other measures *Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy still valid?* – changes in exposure pathways – changes in land use – new contaminants and/or contaminant sources – remedy byproducts – changes in standards, newly promulgated standards, and TBCs – changes in toxicity and other contaminant characteristics – expected progress towards meeting RAOs – risk recalculation/assessment (as applicable) *Question C: Has any other information come to light that could call into question the protectiveness of the remedy?* – ecological risks – natural disaster impacts – any other information that could call into question the protectiveness of the remedy Summary of Technical Assessment – summary of findings and conclusions related to Questions A, B, and C
VIII. Issues	– issues that were identified during the technical assessment and other five-year review activities (*e.g.*, site inspection) – a determination of whether issues affect current or future protectiveness – a discussion of unresolved concerns or items raised by support agencies and the community (States, Tribes, other Federal agencies or departments, local governments, citizens, PRPs, other interested parties)
IX. Recommendations and Follow-up Actions	– list of any recommendations, including follow-up actions to ensure protectiveness – parties responsible for implementation – agencies with oversight authority – schedule for completion
X. Protectiveness Statement(s)	– protectiveness statement(s) developed at the OU level – protectiveness statement developed for the site as a whole at construction complete sites
XI. Next Review	– statement of when the next review is to be completed, or explanation of why no further five-year reviews are needed

3.7 How should I submit a Five-Year Review report?

The procedures for submitting reports to EPA Regions and Headquarters are described below. This process takes place after all reviews of draft reports, and other interagency reviews are completed, appropriate concurrences and signatures are obtained, and, to the extent practicable, issues are resolved.

3.7.1 How is an EPA-lead report submitted?

A report prepared by EPA is complete when it is signed by the EPA Regional Administrator or his/her designee. The Region should submit one copy of the signed Five-Year Review report to EPA Headquarters within ten days of the signature date. The Region should also place a copy of the report in each site information repository.

3.7.2 How is a Federal facility-lead report submitted?

When a Federal agency or department other than EPA conducts a five-year review, the report should be submitted to the Region for review pursuant to the terms of the Federal Facility Agreement or other authorized agreement. The Region should review the report for accuracy, protectiveness determination/statement, and the basis/support for such determination and consistency with this guidance. The EPA Regional Administrator or his/her designee should issue a memorandum that documents any unresolved items or concerns and either concurs with the report findings or provides EPA's own independent findings and protectiveness determination. Within ten days of the signature date of the memorandum, the Region should forward a copy of the report, with the memorandum attached, to EPA Headquarters, and a copy should be placed in each site information repository.

In some cases, EPA may have minimal involvement at the site or in the development of the Five-Year Review report or protectiveness statements. In such cases, Regions should determine whether to rely solely on the information presented by the other Federal agency or department without independent verification. When the Region relies solely on the representations of another Federal agency or department, the Regional Administrator or his/her designee should note this in the memorandum. It is important to consider who signed the Five-Year Review report at the other Federal agency or department. EPA expects that a Five-Year Review report generally will be signed by the other Federal agency or department at the senior management level.

3.7.3 How is a State or Tribal-lead report submitted?

When a State or Tribe conducts a five-year review, the report should be submitted to the respective Region for review of accuracy, protectiveness determination/statement and the basis/support for such determination and consistency with this guidance. The EPA Regional Administrator or his/her designee should issue a memorandum that documents any unresolved

items or concerns and either concurs with the report findings and protectiveness statement(s) or provides EPA's own independent findings and protectiveness determination. Within ten days after the memorandum is signed, the Region should forward a copy of the report, with the memorandum attached, to EPA Headquarters and a copy should be placed in each site information repository.

3.8 What are the annual reporting requirements to EPA Headquarters?

Each EPA Region should report annually to EPA Headquarters on the progress of the five-year reviews for each of their sites. At a minimum, at the end of each fiscal year each Region should provide to EPA Headquarters the following:

- A list of sites that had five-year reviews due for that fiscal year;
- If a five-year review due date changes for any site, or a site no longer needs a five-year review, identify the sites and the basis for the change or discontinuation;
- A list of those sites where five-year reviews were completed;
- For each completed five-year review, a summary of the protectiveness determination(s), issues that impact protectiveness, follow-up actions, and the schedule and entity responsible for implementing such actions;
- Status of protectiveness when Five-Year Review reports from previous fiscal years made a "not protective" determination or "needed further information" before making a protectiveness determination, or deferred protectiveness; and
- Status of follow-up actions identified in Five-Year Review reports from previous fiscal years.

The exact date for submitting the annual report should be provided at the work planning sessions at the beginning of each fiscal year or through your Headquarters Regional Center contact.

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4.0 ASSESSING THE PROTECTIVENESS OF THE REMEDY

A five-year review should determine whether the remedy at a site is or upon completion will be protective of human health and the environment. The level of effort necessary to conduct a five-year review is site-specific and should be tailored appropriately for the remedial action and its stage of implementation. In general, five-year reviews of remedial actions under construction are narrower in scope than five-year reviews of remedies that have been constructed.

Your technical assessment of a remedy should examine the following three questions, which provide a framework for organizing and evaluating data and information and ensure that all relevant issues are considered when determining the protectiveness of the remedy:

- ***Question A*** – Is the remedy functioning as intended by the decision documents?
- ***Question B*** – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?
- ***Question C*** – Has any other information come to light that could call into question the protectiveness of the remedy?

The following sections present Questions A, B, and C in more detail. Exhibit 4-1 summarizes a number of items that you should consider in answering questions A, B, and C in your evaluation of a remedial action.

Exhibit 4-1: Three Questions Used to Determine Whether a Remedy is Protective

When you ask...	you should consider whether...
Question A: Is the remedy functioning as intended by the decision documents?	• performance standards (*e.g.*, cleanup levels, plume containment, pumping rates) are or will likely be met; • there are problems with the remedy that could ultimately lead to the remedy not being protective or suggest protectiveness is at risk (*e.g.*, shrubs or bushes growing on a landfill cap that was designed to have a grass vegetative cover, extent of plume not fully delineated); • access (*e.g.*, fencing, security guards) and institutional controls needed at the particular stage of the remediation are in place and prevent exposure; • other actions (*e.g.*, removals) necessary to ensure that there are no exposure pathways that could result in unacceptable risks have been implemented; and • maintenance activities (*e.g.*, pumping and treating, monitoring slurry walls, mowing cap), as implemented, will maintain the effectiveness of response actions.

Exhibit 4-1: Three Questions Used to Determine Whether a Remedy is Protective

When you ask...	you should consider whether...
Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives used at the time of the remedy selection still valid?	• there are changes in standards identified as Applicable or Relevant and Appropriate Requirements (ARARs) in the ROD, newly promulgated standards, and/or changes in TBCs identified in the ROD, that could call into question the protectiveness of the remedy; • there are changes in land use or the anticipated land use on or near the site; • new human health or ecological exposure pathways or receptors have been identified; • new contaminants or contaminant sources have been identified; • there are unanticipated toxic byproducts of the remedy not previously addressed by the decision documents; • there are changes in the physical site conditions; and • there are changes in the toxicity factors for contaminants of concern.
Question C: Has any other information come to light that could call into question the protectiveness of the remedy?	• ecological risks have been adequately addressed at the site, and/or there is a plan to address them through a future action; and • the site is/was subject to natural disasters, such as a 100-year flood.

4.1 Question A: Is the remedy functioning as intended by the decision documents?

In general, to determine if the remedy is functioning as described in the decision documents, you should first consider its implementation status, (*e.g.*, whether the remedy is under construction, operating, or completed). You should also look for available information about the remedy and compare it to the requirements in the decision documents and remedial design/construction specifications. For purposes of this guidance, definitions of remedial actions under construction, operating remedial actions, and completed remedial actions are as follows:

- ***Remedial actions under construction*** are those actions where physical construction has been initiated, but is not yet complete.

- ***Operating remedial actions*** are those actions that are ongoing, but where cleanup levels have not yet been achieved. Such actions typically have remedial components requiring several years to reach cleanup levels (*e.g.*, groundwater and surface water restoration, monitored natural attenuation, soil vapor extraction, and bioremediation).

- ***Completed remedial actions*** are those actions where construction is complete and cleanup levels have been achieved.

4.1.1 How do I answer Question A for a remedial action that is under construction?

In the case where a remedy is under construction, the focus of your review should be to determine if the remedy is being constructed in accordance with the requirements of the decision documents and design specifications, and if the remedy is expected to be protective when it is completed. In addition, you should confirm that access controls (*e.g.*, fencing, security guards) necessary at this stage of the remediation are in place and successfully prevent exposure. If the remedial action includes institutional controls (ICs), then your five-year review should also consider the implementation status of those controls. For example, answer the following questions: Have specific ICs been identified? Are there ICs needed at this stage of remediation to prevent exposure? Who is responsible for implementing ICs? What is the plan, schedule, and current status for IC implementation?

4.1.2 How do I answer Question A for a remedial action that is operating or completed?

Your review of an operating or completed remedial action generally will address more aspects of the remedy implementation than a review of a remedial action under construction. In general, you should assess the following:

- ***Remedial action performance*** – Determine whether the remedial action continues to operate and function as designed (*e.g.*, extent of groundwater plume is well defined and updated plume maps confirm containment), and has achieved, or is expected to achieve, cleanup levels.

- ***System operations/operation and maintenance (O&M)*** – Determine whether maintenance procedures, as implemented, will maintain the effectiveness of response actions. This evaluation might include, but is not limited to, visual inspection of the system and the review and evaluation of monitoring reports (*e.g.*, groundwater data from extraction and monitoring wells, biological monitoring data, discharge requirements, wetland monitoring data, leachate monitoring for containment remedies).

- ***Costs of system operations/O&M*** – Review and consider system operations/O&M costs if they are available. Compare actual/current annual O&M costs to the original cost estimate; large variances from the original cost estimate might indicate potential remedy problems. (Note: This information may not be readily available at Enforcement-lead sites, but should be requested.)

- ***Implementation of institutional controls and other measures*** – Determine whether access controls (*e.g.*, fencing, security guards) and ICs that are needed at this stage of the remediation are in place and successfully prevent exposure. If

ICs are not in place, determine why not, and obtain the schedule for implementation; determine whether other actions (*e.g.*, removals) necessary to ensure that exposure pathways that could result in unacceptable risks have been implemented.

- ***Monitoring activities*** – Determine whether monitoring activities required to ensure the effectiveness of the remedy (*e.g.*, performance and environmental data collected and results evaluated) are being conducted and whether they are adequate to determine the protectiveness and effectiveness of the remedy.

- ***Opportunities for optimization*** – If readily apparent during the course of conducting five-year review activities, identify any opportunities to improve the performance and/or reduce the costs of sampling and monitoring activities and operating treatment systems. If changes in these activities are recommended in the Five-Year Review report, you should also provide the rationale/basis for such changes. If appropriate, your report can also recommend that an optimization study be conducted.

- ***Early indicators of potential remedy problems*** – Investigate and identify problems that could lead to the remedy being not protective or suggest protectiveness is at risk unless changes are made. Problems could include frequent equipment breakdowns or replacement, or large variances in operating costs (if cost data are available). Some examples of indicators of potential remedy problems could include erosion and/or subsidence of a cap, trend analysis of sampling data showing no decrease in contaminant levels, monitoring data showing evidence of leachate migration, or that the extent of the groundwater contamination plume exceeds the outer reaches of the monitoring network.

4.2 Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives used at the time of remedy selection still valid?

In conducting your five-year review, you should evaluate the effects of significant changes in standards and assumptions that were used at the time of remedy selection. Changes in the promulgated standards or "to be considereds" (TBCs) may impact the protectiveness of the remedy. Similarly, you should investigate the effect of significant changes in the risk parameters that were used to support the remedy selection, such as reference doses, cancer potency factors[12], and exposure pathways of concern. Finally, you should evaluate whether the original assumptions regarding current and future land/groundwater uses and contaminants of concern are

[12] Note that risk parameters in EPA publications such as the Integrated Risk Information System (IRIS) (see http://www.epa.gov/IRIS) are guidance only, and should be applied only as appropriate for the remedy being reviewed.

still valid, and whether any physical features (or understanding of physical sites conditions) have changed (*e.g.*, changes in anticipated direction or rate of groundwater or identification of a new groundwater divide). All of these factors may have a bearing on the validity of the remedial action objectives and may affect the protectiveness of the remedy.

Exhibit 4-2 presents a series of example questions that you should consider in determining whether the exposure assumptions and toxicity data used at the time of remedy selection are still valid and, if you determine that they are no longer valid, whether they call into question the protectiveness of the remedy. Exhibit 4-2 also groups the questions according to the type of assumption.

Exhibit 4-2: Example Questions to Determine if Assumptions Upon Which the Remedy was Based Have Changed

For an assumption based on ...	**an example question may be...**
standards and TBCs	Are there changes in the standards identified as ARARs in the ROD that bear on the protectiveness of the remedy? Are there newly promulgated standards that might apply or be relevant and appropriate to the site and that bear on the protectiveness of the remedy? Are there changes in TBCs identified in the ROD that bear on the protectiveness of the remedy?
cleanup levels	What is the basis for each cleanup level identified in the ROD (*e.g.*, risk-based or promulgated standards as ARARs)? Have there been changes to the basis of the cleanup levels? (See sample questions for "standards or TBCs" above, and for "toxicity and other contaminants characteristics" below.)
exposure pathways	Has land use or expected land use on or near the site changed (*e.g.*, industrial to residential, commercial to residential)?
exposure pathways	Have any human health or ecological routes of exposure or receptors changed or been newly identified (*e.g.*, dermal contact where none previously existed, new populations or species identified on site or near the site)?
exposure pathways	Are there newly identified contaminants or contaminant sources?
exposure pathways	Are there unanticipated toxic byproducts of the remedy not previously addressed by the decision documents (*e.g.*, byproducts not evaluated at the time of remedy selection)?
exposure pathways	Have physical site conditions changed such that protectiveness may be affected (*e.g.*, changes in anticipated direction or rate of groundwater flow)? Has understanding of physical site conditions changed (*e.g.*, identification of a new groundwater divide)?
toxicity and other contaminant characteristics	Have toxicity factors for contaminants of concern at the site changed (*e.g.*, Integrated Risk Information System (IRIS) evaluations? (See http://www.epa.gov/IRIS) Have other contaminant characteristics changed? Have ecological toxicity reference values and/or ecological "no observed adverse effect levels/lowest observed adverse effect" (NOAELs/LOAELs) levels changed.

4.2.1 How should I check the impact of changes in standards and TBCs?

Cleanup levels or actions may be based on ARARs identified in the Record of Decision (ROD) (as opposed to calculated site-specific risk, as discussed in Section 4.2.3). For example, the cleanup levels for a groundwater remedy may be based on the Safe Drinking Water Act maximum contaminant levels (MCLs) if these were identified as ARARs in the ROD.

In the preamble to the final National Contingency Plan (NCP), EPA states its policy that it will not reopen remedy selection decisions contained in RODs (*i.e.*, ARARs are normally frozen at the time of ROD signature) unless a "new or modified requirement calls into question the protectiveness of the selected remedy." 55 FR 8757 (March 8, 1990). The preamble goes on to state that "a policy of freezing ARARs at the time of ROD signing will not sacrifice protection of human health and the environment because the remedy will be reviewed for protectiveness every five years, considering new or modified requirements at that point, or more frequently, if there is reason to believe that the remedy is no longer protective of health and environment." 55 FR 8758 (March 8, 1990). The preamble also states that a remedy would not necessarily need to "be modified solely to attain a newly promulgated or modified requirement," but that "newly promulgated or modified requirements contribute to [the] evaluation of protectiveness." 55 FR 8758 (March 8, 1990).

Therefore, although ARARs generally are "frozen" at the time of ROD signature, in conducting a five-year review, you should determine the effect of a newly promulgated or modified standard on the protectiveness of the remedy originally selected in the ROD. You should evaluate the newly promulgated or modified requirement to determine if the cleanup level established in the ROD remains protective. TBCs may also have been used to select cleanup levels. Therefore, you should also review any new or modified TBCs to ensure that any changes will not impact the protectiveness of the remedy.

Generally, you should only consider changes in standards that were identified as ARARs in the ROD, newly promulgated standards for chemicals of potential concern, and TBCs identified in the ROD that bear on the protectiveness of the remedy. As such, you should review any newly promulgated standards, including revised chemical-specific requirements (such as MCLs, ambient water quality criteria), revised action and location-specific requirements, and State standards if they were considered ARARs in the ROD.

In evaluating a change in a standard that was identified as an ARAR in the ROD, or a newly promulgated standard or TBC, you should establish whether the new requirement indicates that the remedy is no longer protective. You should recommend a follow-up action when the remedy is not protective. For example, based on revised risk information for a specific chemical, a new standard (*e.g.*, more stringent MCL for a chemical) may result in a situation where the cleanup level to be achieved by the original remedy would pose a 10^{-3} cancer risk. In that circumstance, the five-year review could recommend that a new cleanup level based on the new

standard be adopted and, if necessary, that the remedy be modified. However, a change in a standard may not necessarily result in a change in the resulting risk and therefore may not always impact protectiveness. An illustration of a method and an example for evaluating changes in standards is provided in Appendix G, "Methods and Examples for Evaluating Changes in Standards and Toxicity," Exhibit G-1, "Evaluating Changes in Standards," Exhibit G-2, "Hypothetical Scenario for a Change in a Standard," and Exhibit G-3, "Decision Process for a Hypothetical Change in Standard."

4.2.2 How should I check the impact of changes in exposure pathways?

You should consider changes in site conditions that could result in increased exposure. These changes could include changed or new land uses, including zoning changes, changed or new routes of exposure or receptors, changed physical site conditions that may affect the protectiveness of the remedy, new contaminants, or a new understanding of geological conditions. In evaluating this information, you should work closely with a risk assessor to establish the impact that such changes may have on the estimated risk associated with your site. Depending on the significance of the changes, it may be necessary for you to recalculate human health risk and re-examine ecological risks. Generally, your human health determination should be based on whether the cancer risk could now be greater than 10^{-4} and/or the hazard index could be greater than 1 for non-carcinogenic effects.

In some cases, it may be necessary to revise or expand the previous risk assessment as part of your five-year review. For example, you may need to revise the risk assessment when there is a new exposure pathway, a new potential contaminant of concern, or an unanticipated toxic byproduct of the remedy. In all cases, you should evaluate whether the remedy can mitigate any unacceptable risk or whether additional actions may need to be taken. Your five-year review can also recommend further investigation to determine whether an additional response action is needed.

4.2.3 How should I check the impact of changes in toxicity and other contaminant characteristics?

Cleanup levels at a site may be based on the calculated risk for chemicals and/or media where there are no promulgated standards (*e.g.*, site-specific soil and sediment action levels) or existing standards are not sufficiently protective for site-specific conditions. If the remedy is intended to meet a site-specific, risk-based cleanup level, you should check to see whether toxicity or other contaminant characteristics used to determine the original cleanup level have changed. In addition to toxicity, you should examine other contaminant characteristics that determine the nature and extent of contaminant migration and effects on receptors (*e.g.* sorption characteristics, ability to bioaccumulate, bioavailability). If there have been changes in the understanding or in our knowledge of these physical/chemical characteristics, you may need to recalculate risk using the original cleanup level or using the current concentration if it has not been identified as a contaminant of concern. An increase in the cancer slope factor, for example,

may suggest that the risk from a chemical concentration is above the generally acceptable cancer risk range (10^{-4} to 10^{-6}). You should also consider changes in toxicity and other contaminant characteristics relating to ecological receptors.

You may work with your Region's risk assessor to determine whether there have been changes in toxicity or other contaminant characteristics and whether further investigation is needed. The risk assessor is also familiar with efficient use of the Superfund Technical Support Center and its hotline. One preferred resource for checking changes in toxicity information is EPA's Integrated Risk Information System (IRIS) (http://www.epa.gov/IRIS). However, many contaminants found at Superfund sites are not found in IRIS. You may find it useful to refer to the Superfund Risk Assessment Tools of the Trade page for databases and additional links and pointers (http://www.epa.gov/superfund/programs/risk/tooltrad.htm#gp). Beginning in the summer of 2001, this page should link risk-based concentration tables which provide screening levels for specific exposure scenarios, a risk calculation tool, and should identify recent toxicity data and their sources.

The flowchart presented in Appendix G, Exhibit G-4, "Evaluating Changes in Toxicity and Other Contaminant Characteristics," shows the process you should use to evaluate the significance of changes in toxicity values and other contaminant characteristics when conducting a five-year review. You should first identify any site-specific, risk-based, cleanup levels and investigate relevant changes in contaminant characteristics. If the estimated risk for a contaminant has not changed, your analysis on this point should be complete.

If the estimated risk has increased, then you should determine whether the new estimated risk is acceptable. In most cases, you should base this determination on whether the risk is within or below the generally acceptable risk range of 10^{-4} to 10^{-6} for carcinogenic risk and the hazard index is below 1 for non-carcinogenic effects. If the estimated risk is not protective, you should determine what actions need to be taken to achieve an acceptable level of risk. Appendix G, Exhibit G-5, "Hypothetical Scenario for a Change in Toxicity," and Exhibit G-6, "Decision Process for a Hypothetical Change in Toxicity," provide an example of the evaluation process when there are changes in toxicity and other characteristics. Note: Future guidance will address the appropriateness of using various statistical methods in making the determination about when remedial action objectives (RAOs) have been attained.

4.2.4 How should I review RAOs and evaluate their impact?

As part of the five-year review, you should conduct an evaluation of the RAOs stated in the ROD to determine whether the remedy is meeting or will meet RAOs. Depending on the outcome of the evaluation, you may find it necessary to modify the RAOs, modify the remedy, or conduct further response actions. For example, an RAO phrased in terms of "achieving the drinking water standard in ten years" may be significantly affected by a new MCL that establishes a more stringent standard. Conversely, an RAO may be general enough to accommodate a new or modified requirement.

If your evaluation of data indicates that the remedy is not meeting and will not be able to meet the RAO stated in the ROD, then you may need to determine if the remedy is protective and, if not protective, what additional actions are needed. For example, if the risk associated with the cleanup levels currently being achieved by the remedy are within EPA's acceptable risk range, the remedy generally should be considered protective. However, if the remedy will not be able to meet the RAOs, further actions may be needed, depending on the specificity of the original RAOs in the ROD. Your Five-Year Review report should identify such further actions as recommendations and/or follow-up actions.

New site conditions, such as discovery of new contaminants, can also impact the RAOs and remedy protectiveness. During your five-year review, you should evaluate whether the RAOs in the ROD are sufficiently comprehensive to cover any new or changed conditions at a site. If a new condition at the site is not covered by the RAOs, you should recommend further investigation in the Five-Year Review report to determine whether additional response actions are needed.

Further response actions may not necessarily involve additional physical construction activities but could include sampling, studies, and/or investigations. For example, modifying RAOs will require a ROD Amendment, but does not require a physical site activity.

4.3 Question C: Has any other information come to light that could call into question the protectiveness of the remedy?

You should consider any other information that comes to light that could call into question the protectiveness of the remedy. It is expected that most considerations related to the protectiveness of the remedy will be covered by Questions A and B. However, in some instances, there may be other factors about the remedy or the site that you should consider during the review.

Situations to watch for include the following:

- Ecological risks have not been adequately addressed at a site, and there is not a plan to address them through a future action;
- The site, although located entirely above the 500-year flood boundary, was partially inundated by a 100-year flood (which now may require a flood plain redesignation of the region); and
- Land use changes that are being considered by local officials.

If ecological risks have not been adequately addressed at a site, and there is not a plan to address them through a future action, then you may need to address them by conducting a screening ecological risk assessment as part of the Five-Year Review using *Final Guidance: Ecological Risk Assessment and Risk Management Principles for Superfund Sites*, OSWER

Directive 9285.7-28P (October 7, 1999). The ecological risk assessor on your team can help streamline the process appropriately.

4.4 How should I develop the conclusions of my five-year review?

The conclusions of your five-year review should include: 1) an identification of issues; 2) recommendations and follow-up actions; and 3) a determination of whether the remedy is, or is expected to be, protective of human health and the environment. You should arrive at these conclusions through a technical assessment of the information collected during the document review, data collection, interviews, site inspection, and other activities. Your evaluation should focus on the information collected through answering the three questions shown in Exhibit 4-1. (See Sections 4.1, 4.2, and 4.3, above, for a detailed discussion of how to assess the remedy by answering these three questions.) These conclusions should be documented in the Five-Year Review report as a technical assessment summary.

4.4.1 How should I identify issues?

You should identify all issues that currently prevent the response action from being protective, or may do so in the future. You should document all such issues and follow-up actions needed to ensure the proper management of the remedy in your Five-Year Review report. You should also identify early indicators of potential remedy problems. Early indicators of remedy problems may include operating costs that are greater than originally anticipated. For instance, excessive replacement of pumps or other equipment may indicate the need to reconsider system design or re-evaluate aquifer conditions.

Examples of issues that may be identified in a Five-Year Review report include the following:

- Inadequate access controls (*e.g.*, fencing has been breached, or fencing is not adequate to restrict access);
- Incomplete response action, including ICs (*e.g.*, environmental easements or well restrictions are not in place);
- Inadequate ICs (*e.g.*, well restrictions are in place but are not preventing exposure);
- Response action is not expected to achieve cleanup levels; plume containment has not been confirmed or achieved;
- Cleanup levels are not protective due to changes in chemical characteristics;
- Discharge requirements are exceeded;
- Inadequate operation and maintenance of physical remedial structures (*e.g.*, vegetative cover of cap mowed infrequently);

- Differences found in actual or proposed land use other than those assumed in the selection of the response action;
- RAOs will not be achieved;
- Monitoring is not being completed in a timely manner; and
- Inadequate monitoring activities to determine the protectiveness of the remedy (*e.g.*, the number and location of monitoring wells are not appropriate for monitoring remediation progress of the groundwater contamination plume).

You should describe each issue in sufficient detail so that EPA can appropriately track the progress to resolution. For each issue, you should determine if it currently affects the protectiveness of the remedy or may do so in the future.

Exhibit 4-3 provides an example of a tabular format that you can use to list issues in your Five-Year Review report.

Exhibit 4-3: Example Table for Listing Issues

Issues	Affects Protectiveness (Y/N)	
	Current	Future

4.4.2 When and how should I develop recommendations?

For each issue identified, the Region should document and ensure implementation of recommendations to resolve those issues. These recommendations should be identified along with follow-up actions in your Five-Year Review report. Follow-up actions should be completed to ensure long-term protectiveness of the remedy, or to bring about protectiveness of a remedy that is currently not protective. You may also have follow-up actions where a protectiveness determination cannot be made at the time of the five-year review. In addition, you may wish to make additional recommendations that do not directly relate to achieving or maintaining the protectiveness of the remedy, such as activities related to O&M of the remedy and coordination with other public and government authorities.

The following are types of recommendations that generally are considered appropriate as part of a five-year review:

- ***Provide additional response actions*** – For example, additional response actions may be necessary to ensure protectiveness if new risk information indicates that a remedy is not protective (*e.g.*, a treatment process will not be able to achieve soil cleanup levels). EPA may implement such further response any time pursuant to CERCLA §104 or §106 authority. In your Five-Year Review report, you can recommend further investigation and the implementation of further response actions.

- ***Improve O&M activities*** – For example, when a cap's vegetative cover is not mowed on a regular basis and/or vegetation other than that specified in the remedial construction contract specifications is present, you may recommend that actions be taken to improve compliance with the O&M Manual/Plan. The lack of O&M activities can lead to more serious remedy problems if not addressed. Your Five-Year Review report should recommend that O&M activities be conducted if they currently are not being performed or inadequately conducted and, if needed, expanded, reduced, or terminated. The report should also provide the rationale/basis for any of these recommendations.

- ***Optimize remedy*** – For example, when the limits of a groundwater plume have contracted due to pumping, and some monitoring wells no longer register contamination levels above cleanup levels, it may be appropriate to revise the sampling plan to eliminate these wells from the sampling routine or reduce the frequency of their sampling. It may also be possible to remove specific groundwater extraction wells from service and increase or reduce the pumping rate on others to optimize groundwater remediation. Similarly, it may be possible to remove treatment units that no longer contribute to the achievement of remedial goals.

- ***Enforce access controls and ICs*** – For example, when repeated site trespassing has been observed, you could recommend repair of the fence and an evaluation of the need for additional security measures. When you have evidence that groundwater wells continue to be installed despite well restrictions that are currently in place, you can recommend an evaluation of the need for further enforcement of institutional controls (*e.g.*, prohibit well drilling).

- ***Conduct additional studies or investigations*** – For example, after reviewing and evaluating all available data and information it is apparent that contaminant levels have not decreased as expected in the estimated time frame. Additional information will be needed to determine if the remedy, as is, will be able to achieve remediation goals within the estimated time frame. Other studies may include, but are not limited to, site characterization, ecological assessment,

focused feasibility studies, groundwater modeling, treatability studies, and/or sampling.

For each recommendation, you should identify the party responsible for implementation, the agency with oversight authority, a recommended schedule for implementation and completion, and the impact, if any, on current or future protectiveness. Exhibit 4-4 provides an example of a table that you can use in your Five-Year Review report for documenting both recommendations and follow-up actions.

Exhibit 4-4: Example Table for Listing Recommendations and Follow-up Actions

Recommendations/ Follow-up Actions	Party Responsible	Oversight Agency	Milestone Date	Follow-up Actions: Affects Protectiveness (Y/N)	
				Current	Future

Regions should track the progress and completion of recommendations and/or followup actions with documentation in the site file, and upon completion update the administrative record in the site information repository. See Section 3.8 for annual reporting responsibilities to EPA Headquarters.

4.5 How do I determine protectiveness?

After addressing Questions A, B, and C, you should be ready to determine the protectiveness of the remedy or remedies at a site and to document the rationale for your determination(s). You should make a protectiveness statement for each OU and an additional, comprehensive site-wide protectiveness statement for those sites that have reached construction completion.

Your determination of whether the remedy remains protective of human health and the environment generally should be based on the answers to Questions A, B, and C and the information obtained in the process of answering them. Although protectiveness generally is defined by the risk range and hazard index (HI), your answers to Questions A, B, and C may identify other factors and issues that may impact the protectiveness of a remedy.

At the end of your technical analysis and evaluation, if the answers to Questions A, B, and C are *yes, yes, and no*, respectively, then your remedy normally should be considered

protective. However, if the answers to the three questions are other than *yes, yes, no,* depending on the elements that affect each question, your remedy may be one of the following:

- Protective;
- Will be protective once the remedy is completed;
- Protective in the short-term; however, in order for the remedy to be protective in the long-term, follow-up actions need to be taken;
- Not protective, unless the following action(s) are taken in order to ensure protectiveness; or
- Protectiveness cannot be determined until further information is obtained. (A time frame should be provided when a protectiveness determination will be made. This should be done through an addendum. If this is the case, your next five-year review should be due five years from the date this report is signed, not the signature date of the addendum).

Even if there is a need to conduct further actions, it does not mean that the remedy is not protective. Normally, the remedy should be considered as not protective when the following occur:

- An immediate threat is present (*e.g.* exposure pathways that could result in unacceptable risks are not being controlled);
- Migration of contaminants is uncontrolled and poses an unacceptable risk to human health or the environment;
- Potential or actual exposure is clearly present or there is evidence of exposure (*e.g.*, institutional controls are not in place or not enforced and exposure is occurring); or
- The remedy cannot meet a new cleanup level and the previous cleanup level is outside of the risk range.

Exhibit 4-5 presents examples of protectiveness determinations. These examples cover only some of the possible situations you may observe at your site but should serve to guide your decision-making.

Exhibit 4-5: Examples of Protectiveness Determinations

1. Remedies Under Construction			
If the remedy involves...	**and you observe in your five-year review that...**	**then your answers to Questions A, B and C should be...**	**and...**
any remedial action under construction	• no changes to site conditions or any other parameters would impact protectiveness	A - Yes B - Yes C - No	the remedy will be protective.
a groundwater pump-and-treat system expected to operate for 30 years with institutional controls to restrict well drilling of groundwater wells	• an MCL for one of the contaminants of concern (COCs) has become more stringent since the ROD was signed; and the risk associated with the previous MCL is now outside of the risk range; • the remedy cannot meet the new standard (even with design modifications); and • ICs are in place,	A - Yes B - No C - No	the remedy is not protective because the remedy is not able to meet the new standard (ARAR) and the previous MCL is outside of the risk range. However, since ICs are in place there are no current exposures. Recommend that follow-up actions be taken to address the new MCL (ARAR) issue.
rerouting of contaminated surface runoff from tailings	• remedy in the ROD did not address ecological risks; • sediment sampling data from adjacent wetlands indicate high levels of heavy metals; • there were dead fish, and land animals with physical abnormalities; or • an ecological risk assessment was not previously conducted,	A - Yes B - Yes C - Yes	defer protectiveness because more information is needed to make a protectiveness determination. Recommend that follow-up actions be taken to address inadequate ecological risk data.

Question A – Is the remedy functioning as intended by the decision documents?
Question B – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?
Question C – Has any other information come to light that could call into question the protectiveness of the remedy?

Exhibit 4-5: Examples of Protectiveness Determinations

2. Operating Remedies			
If the remedy involves...	**and you observe in your five-year review that...**	**then your answers to questions A, B and C should be...**	**and...**
any operating remedy	• no changes to site conditions or any parameters under Questions A, B, and C occurred,	A - Yes B - Yes C - No	the remedy is protective.
groundwater pump-and-treat system expected to operate for 15 years with ICs to restrict well drilling	• no well drilling restriction in place as required by ROD; • there is no known current exposure to groundwater, based on site visits, interviews with local officials and residents,	A - No B - Yes C - No	the remedy is considered protective in the short-term, because there is no evidence that there is current exposure. However, in order for the remedy to remain protective in the long-term, ICs restricting well drilling must be put in place.
groundwater pump-and-treat for 20 years; ICs restricting well drilling; RAO: restore groundwater to drinking water standards	• based on data and current groundwater modeling, the RAOs will not be met; • ICs are in place; • the system has been operating for ten years; • there are no changes in standards or contaminant characteristics for COCs; • there are no new standards; • contaminant levels of COCs have leveled off in the last five years; • optimization efforts have not been effective in further decreasing COC levels; • current levels of contamination are within EPA's risk range, however, RAOs have not yet been achieved,	A - No B - No C - No	the remedy is considered protective in the short-term because ICs are in place, and therefore, there is no current or potential exposure. Follow-up actions are necessary to address long-term protectiveness because RAOs are not expected to be met. Recommend that the remedial action objectives may need to be reevaluated and other potential actions be further evaluated.

Question A – Is the remedy functioning as intended by the decision documents?
Question B – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?
Question C – Has any other information come to light that could call into question the protectiveness of the remedy?

Exhibit 4-5: Examples of Protectiveness Determinations

If the remedy involves...	**and you observe in your five-year review that...**	**then your answers to questions A, B and C should be...**	**and...**
groundwater pump-and-treat for 10 years; ICs on well drilling; RAO: groundwater restoration to beneficial use	• ICs are in place; • there is a new State MCL for one of the COCs; • the standard (ARAR) in the original ROD is still protective because it is within the same order of magnitude as the new State MCL and remains within EPA's risk range; • there is no current exposure - residents with private wells in the area are on alternate water supply; • the State considers all groundwater to be a potential source of drinking water (However, there is no Comprehensive State Groundwater Protection Plan [CSGWPP]); and • the existing remedy (system) can achieve the new MCL,	A - Yes B - No C - No	the remedy is considered protective because the cleanup levels are still within EPA's risk range and there is no current or potential exposure.

Question A – Is the remedy functioning as intended by the decision documents?
Question B – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?
Question C – Has any other information come to light that could call into question the protectiveness of the remedy?

Exhibit 4-5: Examples of Protectiveness Determinations

If the remedy involves...	and you observe in your five-year review that...	then your answers to questions A, B and C should be...	and...
groundwater pump-and-treat for 20 years; ICs restricting well drilling; RAO: groundwater restoration to drinking water standards	• ICs are in place; • new Federal standard for one of the COCs; • the standard (ARAR) in the original ROD is still protective, within EPA's risk range; • no current or potential exposure to groundwater; and • existing remedy can remediate groundwater to the new standard,	A - Yes B - No C - No	the remedy is considered protective because cleanup levels are still within the risk range and there is no current or potential exposure. However, if the new MCL is not met, the groundwater will not meet the RAO of restoration to drinking water standards. Recommend consideration of follow-up actions to address the new standard and the issue of not achieving the RAO. However, in this case, the remedy can meet the new standard, and therefore, another option is to recommend that the new standard be adopted as the new cleanup level, which would then allow you to achieve the original RAOs. Adopting a new cleanup level would have to be done through the remedy decision process with a ROD Amendment or Explanation of Significant Differences (ESD).

Question A – Is the remedy functioning as intended by the decision documents?
Question B – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?
Question C – Has any other information come to light that could call into question the protectiveness of the remedy?

Exhibit 4-5: Examples of Protectiveness Determinations

3. Completed Remedies			
If the remedy involves...	**and you observe in your five-year review that...**	**then your answers to questions A, B and C should be...**	**and...**
any remedy that is complete with a five-year review requirement	• there were no changes to site conditions or parameters under questions A, B, and C,	A - Yes B - Yes C - No	the remedy is protective.
capping of 30-acre landfill with ICs to prevent disturbance of cap	• ICs were never put in place; • mowing and cap maintenance activities are ongoing and adequate; • there is no cracking, sliding, settlement of cap or other indicators of cap breaches; and • there is no evidence of an exposure (human or ecological),	A - No B - Yes C - No	the remedy is considered protective in the short-term because there is no evidence of a cap breach and thus no current exposure. However, in order for the remedy to remain protective in the long-term, ICs must be put in place.
groundwater pump-and-treat for 10 years; ICs restricting well drilling; RAO: restore groundwater to drinking water standards; cleanup goals were achieved and RAOs were met (third five-year review is being conducted as a matter of policy in order to facilitate the deletion process)	• there is a new standard for one of the COCs; • Standard in original ROD (ARAR) is now outside of the risk range (due to a change in toxicity); and • ICs are no longer in place because RAOs were met last year,	A - Yes B - No C - No	the remedy is not protective because the standard in the ROD is no longer within the risk range and therefore no longer protective. In addition, the RAO is no longer being met. Recommend follow-up actions necessary to make remedy protective and deletion should not occur until this issue is resolved.

Question A – Is the remedy functioning as intended by the decision documents?
Question B – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?
Question C – Has any other information come to light that could call into question the protectiveness of the remedy?

Exhibit 4-5: Examples of Protectiveness Determinations

If the remedy involves...	and you observe in your five-year review that...	then your answers to questions A, B and C should be...	and...
excavation and disposal of top two feet of contaminated soil; ICs prohibiting residential and recreational use of the property; RAO: cleanup site to allow for industrial use; site was deleted three years ago	• ICs are still in place; • the remedy is intact, no physical disturbances, top two feet of clean soil remain undisturbed; and • the local government is considering changing the zoning of the property to allow for recreational use,	A - Yes B - Yes C - No	the remedy is considered to be currently protective. However, should the zoning of the property change to recreational use, the remedy may no longer be protective. Recommend follow-up actions with local officials to ensure that in the event that zoning changes the remedy will remain protective.

Question A – Is the remedy functioning as intended by the decision documents?
Question B – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?
Question C – Has any other information come to light that could call into question the protectiveness of the remedy?

4.5.1 How do I formulate protectiveness statements?

You should develop a protectiveness statement for each OU at which a remedial action has been initiated. For sites that have reached construction completion and have more than one OU, you should develop an additional comprehensive site-wide protectiveness statement covering all of the remedies at the site. You should not include this additional protectiveness statement until construction completion because, until then, all remedies at the site may not necessarily have been selected and constructed.

In order to promote consistency, you are strongly encouraged to model your protectiveness statements on the sample protectiveness statements provided in Exhibits 4-6 and 4-7. Your Five-Year Review report should present the protectiveness statements at the beginning of a discussion that should explain and provide the supporting rationale of the protectiveness determination.

Exhibit 4-6: Protectiveness Statements

If the remedial action at the OU is:	then use this statement ...
under construction and...	
protective or will be protective	"The remedy at OU X is expected to be protective of human health and the environment upon completion, and in the interim, exposure pathways that could result in unacceptable risks are being controlled."
not protective	"The remedy at OU X is not protective because of the following issue(s) (describe each issue). The following actions need to be taken (describe the actions needed) to ensure protectiveness."
protectiveness deferred	" A protectiveness determination of the remedy at OU X cannot be made at this time until further information is obtained. Further information will be obtained by taking the following actions (describe the actions). It is expected that these actions will take approximately (insert time frame) to complete, at which time a protectiveness determination will be made."

Exhibit 4-6: Protectiveness Statements

If the remedial action at the OU is:	**then use this statement ...**
operating or completed and...	
protective	"The remedy at OU X is expected to be protective upon completion or is protective of human health and the environment, and in the interim, exposure pathways that could result in unacceptable risks are being controlled."
protective in the short-term	"The remedy at OU X currently protects human health and the environment because (describe the elements of the remedy that protect human health and the environment in the short term). However, in order for the remedy to be protective in the long-term, the following actions need to be taken (describe the actions needed) to ensure long-term protectiveness."
not protective	"The remedy at OU X is not protective because of the following issue(s) (describe each issue). The following actions need to be taken (describe the actions needed) to ensure protectiveness.
protectiveness deferred	" A protectiveness determination of the remedy at OU X cannot be made at this time until further information is obtained. Further information will be obtained by taking the following actions (describe the actions). It is expected that these actions will take approximately (insert time frame) to complete, at which time a protectiveness determination will be made."

Exhibit 4-7: Comprehensive Protectiveness Statements for Sites That Have Reached Construction Completion

If the remedy(ies) is/are ...	**then use this statement:**
protective	"Because the remedial actions at all OUs are protective, the site is protective of human health and the environment."
not protective	"The remedial actions at OUs X and Y are protective. However, because the remedial action at OU Z is not protective, the site is not protective of human health and the environment at this time. The remedial action at OU Z is not protective because of the following issue(s) (describe each issue). The following actions need to be taken (describe the actions needed) to ensure protectiveness."

Appendix A
Community Involvement

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Community Involvement

This appendix provides a brief discussion about community involvement during the five-year review with a focus on the role of the 40 CFR §300 Community Involvement Coordinator (CIC), community involvement activities, notifying the community, additional recommended activities at high visibility sites, elements of a communications strategy, interviewing members of the community, an example timeline of communication activities, and sources for additional information on community involvement.

What is the role of the Community Involvement Coordinator (CIC)?

The Community Involvement Coordinator (CIC) serves as a public participation and communications advisor. It is his/her job to ensure effective communications with the community. You should consult with the CIC about the most appropriate methods for notifying and involving the community in the five-year review process. The CIC may advise, develop and implement activities designed to notify the community and to involve the community. Part of the community involvement process should involve reviewing the existing Community Involvement Plan (CIP) for the site. The CIP typically describes the history of the site, including any community involvement activities conducted in the past or special needs of the community. Many changes may have taken place in the community since the CIP was last revised or since the last five-year review. For example, the demographics of the community may have changed and new businesses and residents may live in the area. Some residents may speak a language other than English. The CIC can arrange for an interpreter and written materials can be translated into the appropriate language.

When should I begin community involvement activities?

You should begin working with the site's Regional CIC during the initial planning stages of the five-year review to determine the appropriate level of community involvement for the five-year review.

What points should be covered in notifying the community?

At a minimum, community involvement activities during the five-year review should include notifying the community that the five-year review will be conducted and notifying the community when the five-year review is completed. The CIC can recommend appropriate communication vehicles for notifying the public (*e.g.*, publishing a public notice in the newspaper, radio announcement, etc).

The site team should determine the best means for notifying the community that the five-year review process is underway. In some communities, holding an open house or public meeting where community members may stop by and ask questions or pick up fact sheets, brochures, etc., may work effectively. Other activities may include broadcasting a public service

announcement on radio or television and mailing, posting, or handing out a fact sheet. Depending on the nature of the site and the interest in the community, another option for involving the public is to provide a public comment period on the findings of the five-year review.

Notice to the community that a five-year review will be conducted should at a minimum provide:

- The site name, its location and web address (if available);
- The lead agency conducting the review;
- A brief description of the selected remedy;
- A summary of contamination addressed by the selected remedy;
- How the community can contribute during the review process;
- A contact point and phone number for further information; and
- The scheduled date of completion of the five-year review.

Notice to the community that a five-year review has been completed should include some of the information given in the initial notice plus additional information. At a minimum, the notice that a five-year review has been completed should include:

- The site name, its location, and web address (if available);
- The lead agency conducting the review;
- A brief description of the selected remedy;
- A summary of contamination addressed by the selected remedy as provided in the initial notice;
- A brief summary of the results of the five-year review;
- The protectiveness statement(s);
- A brief summary of data and information that provided the basis for determining protectiveness, issues, recommendations, and follow-up actions directly related to the protectiveness of the remedy;
- Location(s) where a copy of the five-year review can be obtained or viewed (including site repositories);
- A contact name and telephone number where community members can obtain more information or ask questions about the results; and
- The date of the next five-year review or a statement and supporting rationale that five-year reviews will no longer be required.

Are there any additional recommended activities that I should consider at high visibility sites?

At high profile sites or those with significant public interest, you should carefully consider methods for informing the community about the review. You should determine if additional or enhanced community involvement activities are appropriate. During the five-year review, active community members may be interested in some or all of the following topics:

- The five-year review process;
- How community members or groups can contribute information about site activities;
- Where to find written documentation about the review;
- What the protectiveness statements mean; and
- What happens after the review is complete, especially if the remedy is found to be not protective.

The CIC and other review team members that have knowledge of the community's needs and interests should be involved in decisions about the level of community involvement and appropriate activities.

What elements should I include when developing a communication strategy?

It is always a good idea to develop a communication strategy for high profile sites. This strategy should:

- Describe the public's concerns and communication needs;
- Identify specific communication activities that you plan to conduct;
- Outline a proposed schedule for these activities, and assign responsibilities for carrying them out; and
- Present expected results.

Consult Section V of the *Superfund Community Involvement Handbook (OSWER Directive 9230.0-94) and Toolkit (OSWER Directive 9230.0-95)* for an example of a communication strategy. This strategy does not need to be added to the official record, and can be as informal or detailed as community needs demand.

How should I approach interviewing members of the community?

In addition to notifying the community about the five-year review, you and the CIC, in conjunction with the site team, should consider interviewing community members (especially those living near the site) to get their views about site conditions and related concerns. If there is a Community Advisory Group or a group with a Technical Assistance Grant related to the site, they should be briefed at the outset of the five-year review process in addition to other interviews you may conduct.

You, the CIC, and other team members should review the community profile in the CIP to obtain useful information about the community, such as business owners or residents living near the site, and the past level of interest from individuals and groups in the community. The CIP can also be a source for identifying other stakeholders who have been active in site activities in the past and who could provide additional information about site conditions.

Other important sources of information are local officials. In many cases, the CIC may be the best person to consult local officials, because they may have met or spoken with them previously and established rapport.

See Appendix C, "Five-Year Review Interviews," for additional information about conducting interviews as part of a five-year review.

What is the timeline for communication activities during a five-year review?

Table 1, "Major Communication Milestones During a Five-Year Review," outlines the major communication milestones during a five-year review and a suggested time frame for conducting communication activities, especially at high profile sites or those with a strong public interest. Consult the *Superfund Community Involvement Handbook and Toolkit* to determine which activities may be best suited for your community at each stage, and for details on the time frame and effort needed for each activity. Activities may be conducted before or at the outset of your five-year review and during or close to the time of the site inspection, depending on the community needs. Activities that you should conduct for all five-year reviews are identified in Table 1 with bolded text.

Table 1: Major Communication Milestones During a Five-Year Review

When you or the CIC...		you should...
Planning the Review and Notifying the Community		
1.	review the existing CIP for potentially helpful information (the CIC should lead this effort),	begin planning immediately, so that if interaction with the community is needed, it is provided up-front.
2.	develop a communication strategy,	prepare a communication strategy before notifying the community. Circumstances and the level of public interest may change throughout the process, so refer to and update the strategy regularly.
3.	**notify the community that the five-year review will begin, using a communication activity appropriate to the specific community,**	notify the community that the five-year review process is beginning before the site inspection.
Consulting the Community		
4.	interview community members to gather additional information about the site,	plan for about one month of coordination and gathering of information, depending on whether contact with the community is via telephone, in person, etc.
Communicating the Results of the Five-Year Review		
When you or the CIC...		**you should...**
5.	plan and conduct additional communication activities tailored to community needs at each site,	plan your activities before releasing the results of the five-year review to the public. Try to complete these activities before the release of the report or within six months after the Five-Year Review report is complete.
6.	**notify the community that the Five-Year Review report is complete, prepare and distribute a brief summary of the results, and place the report in the site information repositories,**	provide this information as quickly as possible after the Five-Year Review report is completed. Consult with the CIC before preparing the summary to determine which communication mechanism is most appropriate to the community's needs.

Note: Bolded activities are required

More Information on Community Involvement

For more information on community involvement activities, please consult the following sources:

- ***The Superfund Community Involvement Handbook (OSWER Directive 9230.0-94) and Toolkit (OSWER Directive 9230.0-95).*** This two-volume handbook and toolkit includes guidance on community involvement policy throughout the Superfund pipeline, including special chapters on working at Federal facilities, risk communication, and multimedia sites. The toolkit components describe and provide over 100 tools that CICs can use to make their jobs easier, such as electronic and hard copy templates for public notices, press releases, fact sheets, communication strategies, etc.

- ***The Superfund Community Tools Home Page.*** There are a number of information resources available on the EPA Web Site. Point your Web browser to http://www.epa.gov/superfund/action/community/index.htm to access the Superfund Community Tools Home Page.

Appendix B
Document Review

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Document Review

The following six sections provide examples of potential documents to be reviewed as part of a five-year review. Each section addresses a different aspect of the document review. Documents commonly reviewed are displayed in a table in each section. Every site is different, so it may be necessary to review additional documents, such as relevant Memoranda of Understanding, to fully understand the remedial actions at a site. The tables and text below should be used as a guide.

- Basis for the Response Action;
- Implementation of the Response;
- Operation and Maintenance;
- Remedy Performance;
- Legal Documentation; and
- Community Involvement.

Basis for the Response Action

Remedy decision documents, and Federal and State laws and regulations, provide the basis upon which the remedy was selected or modified. The documents in the table below identify the background and goals of the remedy and any changes in laws and regulations that may affect the remedy. Other sources of remedy decision information are the Remedial Investigation/Feasibility Study (RI/FS) Report, toxicological and chemical characteristics databases, and transcripts of public meetings.

Non-remedial responses have other types of documentation. For instance, removal actions frequently are documented through an Action Memorandum. You should adapt your review of those documents to the circumstances at your site.

Document	Purpose of Document	Use During the Five-Year Review
Decision Documents – RODs – ROD Amendments – Explanations of Significant Differences – Action Memoranda	– records remedial decision or other actions, and significant changes from the original remedy	– goals of the remedy – background information on the site – basis for action – cleanup levels and applicable or relevant and appropriate requirements (ARARs) – community concerns and preferences

Document	Purpose of Document	Use During the Five-Year Review
Federal Environmental Laws and Regulations	– statutory and regulatory requirements that may affect the judgement as to whether the remedy protects human health and the environment	– changes in standards identified as ARARs in the ROD that provide a basis for cleanup levels/protectiveness of the remedy (only ARARs related to protectiveness need be reviewed) – pertinent laws and regulations promulgated since the signing of the ROD that are potentially applicable or relevant and appropriate and that potentially bear on the protectiveness of the remedy
State Environmental Laws and Regulations	– statutory and regulatory requirements that may affect the judgement as to whether the remedy protects human health and the environment	– more stringent State environmental laws and regulations have the same standing under the National Contingency Plan (NCP) as Federal laws and regulations, and should be reviewed in the same manner when they may call into question whether the remedy protects human health and the environment (the State typically should perform this component of the review)

Implementation of the Response

Implementation documents furnish information about design assumptions, design plans or modifications, and documentation of the completion of construction at operable units (OUs) and the site. Design reports, plans, and specifications are other documents that provide further information.

Document	Purpose of Document	Use During the Five-Year Review
Remedial Action Reports (both interim and final)	– documents that for a single operable unit all construction activities are complete, the remedy is operational and functional, and that cleanup levels have been achieved – Interim Remedial Action Reports are used for long-term actions where cleanup levels have not yet been achieved	– detailed history and status of remedial actions
As-built drawings	– documents changes/modifications to the original design which occurred during the construction	– documentation of completed action and/or implemented remedy

Document	Purpose of Document	Use During the Five-Year Review
Close Out Reports (Preliminary and Final)	– the preliminary report documents that all physical construction for all operable units at a site is complete – the final report documents cleanup levels have been met	– background information and the status of the remedial actions at the site

Remedy Performance

Monitoring data, progress reports, and performance evaluation reports provide information that can be used to determine whether the remedial action continues to operate and function as designed (*e.g.*, extent of groundwater plume is well defined and update plume maps confirm containment), and has achieved, or is expected to achieve, cleanup levels. The data presented in these documents can also provide trend analysis which can be used to determine how well the remedy is performing and how long it will take to achieve remediation goals. These reports can also indicate whether monitoring activities are adequate to ensure the effectiveness of the remedy (*e.g.*, wells in locations that can show contaminant plume is contained and not migrating) and whether these activities are being conducted.

Document	Purpose of Document	Use During the Five-Year Review
Monitoring Information/Records/Progress Reports (information could include air sampling, groundwater monitoring data, survey/settlement monument records, and gas generation records data/performance evaluation)	– records monitoring data and other information, including contaminant levels – trend analysis – containment evaluation	– to check whether contaminant levels are within established criteria – whether cleanup levels will be achieved – (for containment remedies) contaminant plumes are being contained

Operation and Maintenance (O&M)

O&M documents describe the ongoing measures at a site to ensure the remedy remains protective. (Long-term response actions to restore groundwater and surface water during the remedial phase are referred to as "system operations" in this guidance. Although this section refers to O&M documents, similar documents should be reviewed to assess system operations.) They provide the structure for O&M at the site and confirm that O&M is proceeding as planned. O&M documents that may be helpful are the O&M Manual, O&M Plan, the O&M Contract, O&M and Occupational Safety and Health Administration (OSHA) Training Records, permits and service agreements, and access and security logs. Other types of O&M data to be reviewed include permit compliance data such as air or water discharge sampling results, facilities operation data such as treatment train operational records, gas monitoring and leachate collection data, maintenance records and logs, and O&M cost data. These data demonstrate the proper O&M of the remedy.

Document	Purpose of Document	Use During the Five-Year Review
O&M Manual	– contains technical information necessary to operate and maintain the remedy	– purpose and function of the equipment and systems which comprise the overall facility
O&M Reports	– documents O&M activities, data, and costs	– to check whether O&M is proceeding as planned
Discharge Permits and Deviations*	– notes contaminant levels for the discharge permits – notes contaminant levels for deviations	– to check whether the remedy is operating within design parameters

* Permits are not required for actions taken on site. Reviewer should focus on ensuring compliance with substantive requirements of otherwise permitted activities.

Legal Documentation

Legal documentation pertinent to the site may specify responsibilities for conducting remedial actions, implementing institutional and access controls, O&M activities, and performing elements of the five-year reviews.

Document	Purpose of Document	Use During the Five-Year Review
Enforcement Documents – Consent Decrees – Unilateral Administrative Orders – Administrative Orders on Consent	– commitments/ agreements regarding implementation and operation of the remedy, and conduct of studies – access agreements that are needed	– responsibilities of the PRP for conducting remedial activities at various stages of site cleanup – O&M requirements (when these documents are used to enforce the performance of O&M, they may incorporate O&M documents, such as the O&M Manual)
Institutional Controls (deed notices, easements, other conditions, covenants or restrictions on deeds, and groundwater and land use restriction documents)	– means to restrict the use of a parcel or an associated resource, such as groundwater	– status of institutional controls
Superfund State Contracts and Cooperative Agreements	– State assurance letters to conduct O&M – State authorities responsible for O&M – specific O&M requirements – agreements with Indian Tribes	– O&M implementation and reporting requirements – roles of different agencies
Interagency Agreements and Federal Facility Agreements	– responsibilities of other agencies	– O&M guidelines and rules in effect (sometimes other agencies adopt their own guidelines and rules, which must be consistent with those established by EPA)

Community Involvement

The Community Involvement Plan (CIP) may give you a better understanding of the history of community involvement, and of other activities at the site. In addition, the CIP may help you identify community members who would be valuable resources during the interview process.

Document	Purpose of Document	Use During the Five-Year Review
Community Involvement Plan	– site communication strategy that specifies outreach activities	– community concerns/issues and identification of appropriate community members for interviews

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Appendix C
Five-Year Review Interviews

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Five-Year Review Interviews

Information gathered from interviews during the site inspection may be key to understanding site status. Interviews should be conducted with various individuals or groups, including the operation and maintenance (O&M) site manager, O&M staff, local regulatory authorities and response agencies, community action groups or associations, site neighbors, and other stakeholders.

When conducting an interview, the interviewer should note the date of the interview, and the name, title, and affiliation of the person interviewed. The interviewer should also indicate whether the interview was conducted at the site, the office, or by phone. Written documentation of the interview should briefly summarize the discussion, address any problems or successes with the implementation of the remedy, and provide suggestions for future reference. Forms to use during interviews are provided at the end of this appendix.

The following tables provide lists of potential individuals to interview and the type of information which may be obtained during the interviews. The potential individuals to be interviewed are categorized by their ability to provide the following types of information:

- Background information;
- State and local considerations;
- Construction considerations; and
- Performance, Operation and maintenance problems.

All of these individuals may be contacted during the five-year review. In most cases interviewing only a few key individuals will provide sufficient information for the review.

Background Information

The individuals listed below may provide information concerning previous and current concerns about the site, influences that affected the remedy decision, and further clarification on decisions made during remedy selection.

Interview	Information Sought
Previous EPA Staff/Management	– staff members may offer insight and clarification on decisions made during remedy selection and implementation
Nearest Neighbors	– neighbors may provide insight into the enforcement of institutional controls, changes in land use, trespassing, and unusual or unexpected activity at the site

Interview	Information Sought
Community Representatives*	– members of the community may provide a broader view of site activities and issues than can be obtained during the site inspection

* Several types of individuals may be interviewed: residents/businesses adjacent to or on the site; residents/businesses within the path of migration; local civic leaders, local officials, Community Advisory Group (CAG), Technical Assistance Grant (TAG) group, and local environmental groups; and other audiences listed in the community profile in the Community Involvement Plan.

Some example interview questions are given below.

1. What is your overall impression of the project? (general sentiment)

2. What effects have site operations had on the surrounding community?

3. Are you aware of any community concerns regarding the site or its operation and administration? If so, please give details.

4. Are you aware of any events, incidents, or activities at the site such as vandalism, trespassing, or emergency responses from local authorities? If so, please give details.

5. Do you feel well informed about the site's activities and progress?

6. Do you have any comments, suggestions, or recommendations regarding the site's management or operation?

State and Local Considerations

State and local authorities may provide you with information about changes in State laws and regulations and present and prospective land uses and restrictions.

Interview	Information Sought
State Contacts (including those responsible for State water quality, hazardous waste, and environmental health issues)	– changes in State laws and regulations that may impact protectiveness – whether the site has been in compliance with permitting or reporting requirements – information on site activities, status, and issues
Local Authorities (such as police, emergency response or fire departments, and local environmental or planning offices)	– status of institutional controls, site access controls, new ordinances in place, changes in actual or projected land use, complaints being filed, and unusual activities at the site

Some example interview questions are given below.

1. What is your overall impression of the project? (general sentiment)

2. Have there been routine communications or activities (site visits, inspections, reporting activities, etc.) conducted by your office regarding the site? If so, please give purpose and results.

3. Have there been any complaints, violations, or other incidents related to the site requiring a response by your office? If so, please give details of the events and results of the responses.

4. Do you feel well informed about the site's activities and progress?

5. Do you have any comments, suggestions, or recommendations regarding the site's management or operation?

Construction Considerations

It is important for you to determine the status of construction at the site and to ensure that health and safety concerns are addressed.

Interview	Information Sought
Construction Contractor	– progress of project and changes in design due to field conditions – revisions to the O&M Manual, implementation of the Health and Safety Plan/Contingency Plan – insight into potential O&M problems
Construction Manager	– overview of all contractor construction activities at the site, health and safety issues, site protectiveness during construction, and the quality of the construction
Local Emergency Response Officials	– adequacy of contractor's Health and Safety Plan and the contractor's implementation of the Plan – adequacy of contractor's emergency response duties as outlined in the Contingency Plan or Emergency Response Plan of the Health and Safety Plan

Some example interview questions for remedial actions still under construction are given below.

1. What is your overall impression of the project? (general sentiment)

2. What is the current status of construction (*e.g.*, budget and schedule)?

3. Have any problems been encountered which required, or will require, changes to this remedial design or this ROD?

4. Have any problems or difficulties been encountered which have impacted construction progress or implementability?

5. Do you have any comments, suggestions, or recommendations regarding the project (i.e., design, construction documents, constructability, management, regulatory agencies, etc.)?

Performance, Operation And Maintenance Problems

The following individuals may provide information to you regarding the performance of the remedy and status of O&M at the site so that the team can assess the progress of the implementation and effectiveness of the remedy, and any O&M problems.

Interview	Information Sought
O&M Manager/Operating Contractor	– O&M status of the remedy, compliance with permit and reporting requirements, and complaints filed – effectiveness of the O&M Plan – information about any potential causes for concern about the remedy – progress and performance of the remedy
O&M Staff	– effectiveness of the O&M Manual – information about any potential causes for concern about the remedy – Recommendations for adjusting the mode of operation or optimizing the operations protocol
Remedial Design/Remedial Action Consultant	– original concepts behind the O&M of the remedy – questions about remedial design parameters, expected performance and cost, and changes that have occurred during implementation

Some example interview questions are given below.

1. What is your overall impression of the project? (general sentiment)

2. Is the remedy functioning as expected? How well is the remedy performing?

3. What does the monitoring data show? Are there any trends that show contaminant levels are decreasing?

4. Is there a continuous on-site O&M presence? If so, please describe staff and activities. If there is not a continuous on-site presence, describe staff and frequency of site inspections and activities.

5. Have there been any significant changes in the O&M requirements, maintenance schedules, or sampling routines since start-up or in the last five years? If so, do they affect the protectiveness or effectiveness of the remedy? Please describe changes and impacts.

6. Have there been unexpected O&M difficulties or costs at the site since start-up or in the last five years? If so, please give details.

7. Have there been opportunities to optimize O&M, or sampling efforts? Please describe changes and resultant or desired cost savings or improved efficiency.

8. Do you have any comments, suggestions, or recommendations regarding the project?

INTERVIEW DOCUMENTATION FORM

The following is a list of individual interviewed for this five-year review. See the attached contact record(s) for a detailed summary of the interviews.

Name	Title/Position	Organization	Date
Name	Title/Position	Organization	Date
Name	Title/Position	Organization	Date
Name	Title/Position	Organization	Date
Name	Title/Position	Organization	Date
Name	Title/Position	Organization	Date

INTERVIEW RECORD

Site Name:		**EPA ID No.:**
Subject:		**Time:** / **Date:**
Type: ☐ Telephone ☐ Visit ☐ Other **Location of Visit:**		☐ Incoming ☐ Outgoing

Contact Made By:

Name:	**Title:**	**Organization:**

Individual Contacted:

Name:	**Title:**	**Organization:**
Telephone No: **Fax No:** **E-Mail Address:**	**Street Address:** **City, State, Zip:**	

Summary Of Conversation

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Appendix D
Five-Year Review Site Inspection Checklist

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Five-Year Review Site Inspection Checklist

Purpose of the Checklist

The site inspection checklist provides a useful method for collecting important information during the site inspection portion of the five-year review. The checklist serves as a reminder of what information should to be gathered and provides the means of checking off information obtained and reviewed, or information not available or applicable. The checklist is divided into sections as follows:

I. Site Information
II. Interviews
III. On-site Documents & Records Verified
IV. O&M Costs
V. Access and Institutional Controls
VI. General Site Conditions
VII. Landfill Covers
VIII. Vertical Barrier Walls
IX. Groundwater/Surface Water Remedies
X. Other Remedies
XI. Overall Observations

Some data and information identified in the checklist may or may not be available at the site depending on how the site is managed. Sampling results, costs, and maintenance reports may be kept on site or may be kept in the offices of the contractor or at State offices. In cases where the information is not kept at the site, the item should not be checked as "not applicable," but rather it should be obtained from the office or agency where it is maintained. If this is known in advance, it may be possible to obtain the information before the site inspection.

This checklist was developed by EPA and the U.S. Army Corps of Engineers (USACE). It focuses on the two most common types of remedies that are subject to five-year reviews: landfill covers, and groundwater pump and treat remedies. Sections of the checklist are also provided for some other remedies. The sections on general site conditions would be applicable to a wider variety of remedies. The checklist should be modified to suit your needs when inspecting other types of remedies, as appropriate.

The checklist may be completed and attached to the Five-Year Review report to document site status. Please note that the checklist is not meant to be completely definitive or restrictive; additional information may be supplemented if the reviewer deems necessary. Also note that actual site conditions should be documented with photographs whenever possible.

Using the Checklist for Types of Remedies

The checklist has sections designed to capture information concerning the main types of remedies which are found at sites requiring five-year reviews. These remedies are landfill covers (Section VII of the checklist) and groundwater and surface water remedies (Section IX of the checklist). The primary elements and appurtenances for these remedies are listed in sections which can be checked off as the facility is inspected. The opportunity is also provided to note site conditions, write comments on the facilities, and attach any additional pertinent information. If a site includes remedies beyond these, such as soil vapor extraction or soil landfarming, the information should be gathered in a similar manner and attached to the checklist.

Considering Operation and Maintenance Costs

Unexpectedly widely varying or unexpectedly high O&M costs may be early indicators of remedy problems. For this reason, it is important to obtain a record of the original O&M cost estimate and of annual O&M costs during the years for which costs incurred are available. Section IV of the checklist provides a place for documenting annual costs and for commenting on unanticipated or unusually high O&M costs. A more detailed categorization of costs may be attached to the checklist if available. Examples of categories of O&M costs are listed below.

Operating Labor - This includes all wages, salaries, training, overhead, and fringe benefits associated with the labor needed for operation of the facilities and equipment associated with the remedial actions.

Maintenance Equipment and Materials - This includes the costs for equipment, parts, and other materials required to perform routine maintenance of facilities and equipment associated with a remedial action.

Maintenance Labor - This includes the costs for labor required to perform routine maintenance of facilities and for equipment associated with a remedial action.

Auxiliary Materials and Energy - This includes items such as chemicals and utilities which can include electricity, telephone, natural gas, water, and fuel. Auxiliary materials include other expendable materials such as chemicals used during plant operations.

Purchased Services - This includes items such as sampling costs, laboratory fees, and other professional services for which the need can be predicted.

Administrative Costs - This includes all costs associated with administration of O&M not included under other categories, such as labor overhead.

Insurance, Taxes and Licenses - This includes items such as liability and sudden and accidental insurance, real estate taxes on purchased land or right-of-way, licensing fees for certain technologies, and permit renewal and reporting costs.

Other Costs - This includes all other items which do not fit into any of the above categories.

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Please note that "O&M" is referred to throughout this checklist. At sites where Long-Term Response Actions are in progress, O&M activities may be referred to as "system operations" since these sites are not considered to be in the O&M phase while being remediated under the Superfund program.

Five-Year Review Site Inspection Checklist (Template)

(Working document for site inspection. Information may be completed by hand and attached to the Five-Year Review report as supporting documentation of site status. "N/A" refers to "not applicable.")

I. SITE INFORMATION	
Site name:	**Date of inspection:**
Location and Region:	**EPA ID:**
Agency, office, or company leading the five-year review:	**Weather/temperature:**

Remedy Includes: (Check all that apply)

G Landfill cover/containment
G Access controls
G Institutional controls
G Groundwater pump and treatment
G Surface water collection and treatment
G Monitored natural attenuation
G Groundwater containment
G Vertical barrier walls
G Other_____

_____

Attachments: G Inspection team roster attached G Site map attached

II. INTERVIEWS (Check all that apply)

1. **O&M site manager** _____ _____ _____
   Name Title Date
   Interviewed G at site G at office G by phone Phone no. _____
   Problems, suggestions; G Report attached _____
   _____

2. **O&M staff** _____ _____ _____
   Name Title Date
   Interviewed G at site G at office G by phone Phone no. _____
   Problems, suggestions; G Report attached _____
   _____

3. **Local regulatory authorities and response agencies** (i.e., State and Tribal offices, emergency response office, police department, office of public health or environmental health, zoning office, recorder of deeds, or other city and county offices, etc.) Fill in all that apply.

Agency _____
Contact _____ _____ _____ _____
Name Title Date Phone no.
Problems; suggestions; G Report attached _____
_____

Agency _____
Contact _____ _____ _____ _____
Name Title Date Phone no.
Problems; suggestions; G Report attached _____
_____

Agency _____
Contact _____ _____ _____ _____
Name Title Date Phone no.
Problems; suggestions; G Report attached _____
_____

Agency _____
Contact _____ _____ _____ _____
Name Title Date Phone no.
Problems; suggestions; G Report attached _____
_____

4. **Other interviews** (optional) G Report attached.

III. ON-SITE DOCUMENTS & RECORDS VERIFIED (Check all that apply)

1. **O&M Documents**
   - G O&M manual — G Readily available — G Up to date — G N/A
   - G As-built drawings — G Readily available — G Up to date — G N/A
   - G Maintenance logs — G Readily available — G Up to date — G N/A

   Remarks_____

2. **Site-Specific Health and Safety Plan** — G Readily available — G Up to date — G N/A
   - G Contingency plan/emergency response plan — G Readily available — G Up to date — G N/A

   Remarks_____

3. **O&M and OSHA Training Records** — G Readily available — G Up to date — G N/A

   Remarks_____

4. **Permits and Service Agreements**
   - G Air discharge permit — G Readily available — G Up to date — G N/A
   - G Effluent discharge — G Readily available — G Up to date — G N/A
   - G Waste disposal, POTW — G Readily available — G Up to date — G N/A
   - G Other permits_____ — G Readily available — G Up to date — G N/A

   Remarks_____

5. **Gas Generation Records** — G Readily available — G Up to date — G N/A

   Remarks_____

6. **Settlement Monument Records** — G Readily available — G Up to date — G N/A

   Remarks_____

7. **Groundwater Monitoring Records** — G Readily available — G Up to date — G N/A

   Remarks_____

8. **Leachate Extraction Records** — G Readily available — G Up to date — G N/A

   Remarks_____

9. **Discharge Compliance Records**
   - G Air — G Readily available — G Up to date — G N/A
   - G Water (effluent) — G Readily available — G Up to date — G N/A

   Remarks_____

10. **Daily Access/Security Logs** — G Readily available — G Up to date — G N/A

    Remarks_____

IV. O&M COSTS

1. **O&M Organization**

G State in-house G Contractor for State
G PRP in-house G Contractor for PRP
G Federal Facility in-house G Contractor for Federal Facility
G Other_____

2. **O&M Cost Records**

G Readily available G Up to date
G Funding mechanism/agreement in place
Original O&M cost estimate_____ G Breakdown attached

Total annual cost by year for review period if available

From_____ To_____ _____ G Breakdown attached
Date Date Total cost
From_____ To_____ _____ G Breakdown attached
Date Date Total cost
From_____ To_____ _____ G Breakdown attached
Date Date Total cost
From_____ To_____ _____ G Breakdown attached
Date Date Total cost
From_____ To_____ _____ G Breakdown attached
Date Date Total cost

3. **Unanticipated or Unusually High O&M Costs During Review Period**

Describe costs and reasons: _____

V. ACCESS AND INSTITUTIONAL CONTROLS G Applicable G N/A

A. Fencing

1. **Fencing damaged** G Location shown on site map G Gates secured G N/A
Remarks_____

B. Other Access Restrictions

1. **Signs and other security measures** G Location shown on site map G N/A
Remarks_____

C. Institutional Controls (ICs)

1. **Implementation and enforcement**

Site conditions imply ICs not properly implemented G Yes G No G N/A
Site conditions imply ICs not being fully enforced G Yes G No G N/A

Type of monitoring (*e.g.*, self-reporting, drive by) _____
Frequency _____
Responsible party/agency _____
Contact _____ _____ _____ _____
Name Title Date Phone no.

Reporting is up-to-date G Yes G No G N/A
Reports are verified by the lead agency G Yes G No G N/A

Specific requirements in deed or decision documents have been met G Yes G No G N/A
Violations have been reported G Yes G No G N/A
Other problems or suggestions: G Report attached

_____
_____
_____
_____

2. **Adequacy** G ICs are adequate G ICs are inadequate G N/A
Remarks_____
_____
_____

D. General

1. **Vandalism/trespassing** G Location shown on site map G No vandalism evident
Remarks_____
_____

2. **Land use changes on site** G N/A
Remarks_____
_____

3. **Land use changes off site** G N/A
Remarks_____
_____

VI. GENERAL SITE CONDITIONS

A. Roads G Applicable G N/A

1. **Roads damaged** G Location shown on site map G Roads adequate G N/A
Remarks_____
_____

B. Other Site Conditions

Remarks_____

_____

_____

_____

_____

_____

VII. LANDFILL COVERS G Applicable G N/A

A. Landfill Surface

1. **Settlement** (Low spots) G Location shown on site map G Settlement not evident
   Areal extent_____ Depth_____
   Remarks_____
   _____

2. **Cracks** G Location shown on site map G Cracking not evident
   Lengths_____ Widths_____ Depths_____
   Remarks_____
   _____

3. **Erosion** G Location shown on site map G Erosion not evident
   Areal extent_____ Depth_____
   Remarks_____
   _____

4. **Holes** G Location shown on site map G Holes not evident
   Areal extent_____ Depth_____
   Remarks_____
   _____

5. **Vegetative Cover** G Grass G Cover properly established G No signs of stress
   G Trees/Shrubs (indicate size and locations on a diagram)
   Remarks_____
   _____

6. **Alternative Cover (armored rock, concrete, etc.)** G N/A
   Remarks_____
   _____

7. **Bulges** G Location shown on site map G Bulges not evident
   Areal extent_____ Height_____
   Remarks_____
   _____

8. **Wet Areas/Water Damage** G Wet areas/water damage not evident
   G Wet areas G Location shown on site map Areal extent_____
   G Ponding G Location shown on site map Areal extent_____
   G Seeps G Location shown on site map Areal extent_____
   G Soft subgrade G Location shown on site map Areal extent_____
   Remarks_____

9. **Slope Instability** G Slides G Location shown on site map G No evidence of slope instability
   Areal extent_____
   Remarks_____

B. Benches G Applicable G N/A
(Horizontally constructed mounds of earth placed across a steep landfill side slope to interrupt the slope in order to slow down the velocity of surface runoff and intercept and convey the runoff to a lined channel.)

1. **Flows Bypass Bench** G Location shown on site map G N/A or okay
   Remarks_____

2. **Bench Breached** G Location shown on site map G N/A or okay
   Remarks_____

3. **Bench Overtopped** G Location shown on site map G N/A or okay
   Remarks_____

C. Letdown Channels G Applicable G N/A
(Channel lined with erosion control mats, riprap, grout bags, or gabions that descend down the steep side slope of the cover and will allow the runoff water collected by the benches to move off of the landfill cover without creating erosion gullies.)

1. **Settlement** G Location shown on site map G No evidence of settlement
   Areal extent_____ Depth_____
   Remarks_____

2. **Material Degradation** G Location shown on site map G No evidence of degradation
   Material type_____ Areal extent_____
   Remarks_____

3. **Erosion** G Location shown on site map G No evidence of erosion
   Areal extent_____ Depth_____
   Remarks_____

4. **Undercutting** G Location shown on site map G No evidence of undercutting
Areal extent_____ Depth_____
Remarks_____

5. **Obstructions** Type_____ G No obstructions
G Location shown on site map Areal extent_____
Size_____
Remarks_____

6. **Excessive Vegetative Growth** Type_____
G No evidence of excessive growth
G Vegetation in channels does not obstruct flow
G Location shown on site map Areal extent_____
Remarks_____

D. Cover Penetrations G Applicable G N/A

1. **Gas Vents** G Active G Passive
G Properly secured/lockedG Functioning G Routinely sampled G Good condition
G Evidence of leakage at penetration G Needs Maintenance
G N/A
Remarks_____

2. **Gas Monitoring Probes**
G Properly secured/lockedG Functioning G Routinely sampled G Good condition
G Evidence of leakage at penetration G Needs Maintenance G N/A
Remarks_____

3. **Monitoring Wells** (within surface area of landfill)
G Properly secured/lockedG Functioning G Routinely sampled G Good condition
G Evidence of leakage at penetration G Needs Maintenance G N/A
Remarks_____

4. **Leachate Extraction Wells**
G Properly secured/lockedG Functioning G Routinely sampled G Good condition
G Evidence of leakage at penetration G Needs Maintenance G N/A
Remarks_____

5. **Settlement Monuments** G Located G Routinely surveyed G N/A
Remarks_____

E. Gas Collection and Treatment G Applicable G N/A

1. **Gas Treatment Facilities**
   G Flaring G Thermal destruction G Collection for reuse
   G Good condition G Needs Maintenance
   Remarks_____

2. **Gas Collection Wells, Manifolds and Piping**
   G Good condition G Needs Maintenance
   Remarks_____

3. **Gas Monitoring Facilities** (*e.g.*, gas monitoring of adjacent homes or buildings)
   G Good condition G Needs Maintenance G N/A
   Remarks_____

F. Cover Drainage Layer G Applicable G N/A

1. **Outlet Pipes Inspected** G Functioning G N/A
   Remarks_____

2. **Outlet Rock Inspected** G Functioning G N/A
   Remarks_____

G. Detention/Sedimentation Ponds G Applicable G N/A

1. **Siltation** Areal extent_____ Depth_____ G N/A
   G Siltation not evident
   Remarks_____

2. **Erosion** Areal extent_____ Depth_____
   G Erosion not evident
   Remarks_____

3. **Outlet Works** G Functioning G N/A
   Remarks_____

4. **Dam** G Functioning G N/A
   Remarks_____

H. Retaining Walls G Applicable G N/A

1. **Deformations** G Location shown on site map G Deformation not evident
   Horizontal displacement_____ Vertical displacement_____
   Rotational displacement_____
   Remarks_____

2. **Degradation** G Location shown on site map G Degradation not evident
   Remarks_____

I. Perimeter Ditches/Off-Site Discharge G Applicable G N/A

1. **Siltation** G Location shown on site map G Siltation not evident
   Areal extent_____ Depth_____
   Remarks_____

2. **Vegetative Growth** G Location shown on site map G N/A
   G Vegetation does not impede flow
   Areal extent_____ Type_____
   Remarks_____

3. **Erosion** G Location shown on site map G Erosion not evident
   Areal extent_____ Depth_____
   Remarks_____

4. **Discharge Structure** G Functioning G N/A
   Remarks_____

VIII. VERTICAL BARRIER WALLS G Applicable G N/A

1. **Settlement** G Location shown on site map G Settlement not evident
   Areal extent_____ Depth_____
   Remarks_____

2. **Performance Monitoring** Type of monitoring_____
   G Performance not monitored
   Frequency_____ G Evidence of breaching
   Head differential_____
   Remarks_____

IX. GROUNDWATER/SURFACE WATER REMEDIES G Applicable G N/A

A. Groundwater Extraction Wells, Pumps, and Pipelines G Applicable G N/A

1. **Pumps, Wellhead Plumbing, and Electrical**
   G Good condition G All required wells properly operating G Needs Maintenance G N/A
   Remarks_____

2. **Extraction System Pipelines, Valves, Valve Boxes, and Other Appurtenances**
   G Good condition G Needs Maintenance
   Remarks_____

3. **Spare Parts and Equipment**
   G Readily available G Good condition G Requires upgrade G Needs to be provided
   Remarks_____

B. Surface Water Collection Structures, Pumps, and Pipelines G Applicable G N/A

1. **Collection Structures, Pumps, and Electrical**
   G Good condition G Needs Maintenance
   Remarks_____

2. **Surface Water Collection System Pipelines, Valves, Valve Boxes, and Other Appurtenances**
   G Good condition G Needs Maintenance
   Remarks_____

3. **Spare Parts and Equipment**
   G Readily available G Good condition G Requires upgrade G Needs to be provided
   Remarks_____

C. Treatment System G Applicable G N/A

1. **Treatment Train** (Check components that apply)
   G Metals removal G Oil/water separation G Bioremediation
   G Air stripping G Carbon adsorbers
   G Filters_____
   G Additive (*e.g.*, chelation agent, flocculent)_____
   G Others_____
   G Good condition G Needs Maintenance
   G Sampling ports properly marked and functional
   G Sampling/maintenance log displayed and up to date
   G Equipment properly identified
   G Quantity of groundwater treated annually_____
   G Quantity of surface water treated annually_____
   Remarks_____
   _____

2. **Electrical Enclosures and Panels** (properly rated and functional)
   G N/A G Good condition G Needs Maintenance
   Remarks_____
   _____

3. **Tanks, Vaults, Storage Vessels**
   G N/A G Good condition G Proper secondary containment G Needs Maintenance
   Remarks_____
   _____

4. **Discharge Structure and Appurtenances**
   G N/A G Good condition G Needs Maintenance
   Remarks_____
   _____

5. **Treatment Building(s)**
   G N/A G Good condition (esp. roof and doorways) G Needs repair
   G Chemicals and equipment properly stored
   Remarks_____
   _____

6. **Monitoring Wells** (pump and treatment remedy)
   G Properly secured/lockedG Functioning G Routinely sampled G Good condition
   G All required wells located G Needs Maintenance G N/A
   Remarks_____
   _____

D. Monitoring Data

1. Monitoring Data
   G Is routinely submitted on time G Is of acceptable quality

2. Monitoring data suggests:
   G Groundwater plume is effectively contained G Contaminant concentrations are declining

D. Monitored Natural Attenuation

1. **Monitoring Wells** (natural attenuation remedy)
G Properly secured/lockedG Functioning G Routinely sampled G Good condition
G All required wells located G Needs Maintenance G N/A
Remarks_____

X. OTHER REMEDIES

If there are remedies applied at the site which are not covered above, attach an inspection sheet describing the physical nature and condition of any facility associated with the remedy. An example would be soil vapor extraction.

XI. OVERALL OBSERVATIONS

A. Implementation of the Remedy

Describe issues and observations relating to whether the remedy is effective and functioning as designed. Begin with a brief statement of what the remedy is to accomplish (i.e., to contain contaminant plume, minimize infiltration and gas emission, etc.).

B. Adequacy of O&M

Describe issues and observations related to the implementation and scope of O&M procedures. In particular, discuss their relationship to the current and long-term protectiveness of the remedy.

C.	**Early Indicators of Potential Remedy Problems**
	Describe issues and observations such as unexpected changes in the cost or scope of O&M or a high frequency of unscheduled repairs, that suggest that the protectiveness of the remedy may be compromised in the future.
D.	**Opportunities for Optimization**
	Describe possible opportunities for optimization in monitoring tasks or the operation of the remedy.

Appendix E
Five-Year Review Report Template

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Five-Year Review Report Template

This appendix provides a suggested checklist and a format for Five-Year Review reports. The checklist appears first, followed by the report template. You are encouraged to follow the template to ensure national consistency in the structure of Five-Year Review reports. However, each report should take into account site-specific circumstances, and you should modify the report format and content accordingly. For example, in some cases the report may be clearer if organized by operable unit (OU), or you may need to include site-specific questions that do not appear in this appendix.

The suggested format for Five-Year Review reports includes three main components: cover material, summary information, and the report body. Templates for each of these components follow. These templates provide suggested standard formats, boilerplate text, subheadings, checklists, example tables, and protectiveness statements. Suggested boilerplate text is presented in text boxes. Within the boilerplate section, text enclosed in brackets ("[ ]") should be added as appropriate, and *italicized* text denotes discussions that the reviewer should add.

You should use both the checklist and report template as guides for the types of information that should appear in the different sections of your Five-Year Review report. You should include information that is relevant to your site and needed to ensure that the rationale behind the protectiveness determination is adequately documented.

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Content Checklist For Five-Year Review Reports

This checklist may be used by you, your managers, etc., to verify that you have included all of the appropriate information in your Five-Year Review report. Depending on site-specific circumstances, some items may not be applicable. For example, a report for a site just beginning construction will generally contain less data than for a site that has reached construction completion.

General Report Format

- ☐ Signed concurrence memorandum (as appropriate)
- ☐ Title page with signature and date
- ☐ Completed five-year review summary form (page E-15)
- ☐ List of documents reviewed
- ☐ Site maps (as appropriate)
- ☐ List of tables and figures
- ☐ Interview report (as appropriate)
- ☐ Site inspection checklist
- ☐ Photos documenting site conditions (as appropriate)

Introduction

- ☐ The purpose of the five-year review
- ☐ Authority for conducting the five-year review
- ☐ Who conducted the five-year review (lead agency) and when
  - ☐ Organizations providing analyses in support of the review (*e.g.*, the contractor supporting the lead agency)
  - ☐ Other review participants or support agencies
- ☐ Review number (*e.g.*, first, second)
- ☐ Trigger action and date
- ☐ Number, description, and status of all operable units at the site
- ☐ If review covers only part of a site, explain approach
  - ☐ Define which areas are covered in the five-year review
  - ☐ Summarize the status of other areas of the site that are not covered in the present five-year

Site Chronology

- ☐ List all important site events and relevant dates (*e.g.*, date of initial discovery of problem, dates of pre-NPL responses, date of NPL listing, etc.)

Background

- ☐ General site description (*e.g.*, size, topography, and geology)
- ☐ Former, current, and future land use(s) of the site and surrounding areas
- ☐ History of contamination
- ☐ Initial response (*e.g.*, removals)
- ☐ Basis for taking remedial action (*e.g.*, contaminants)

Remedial Actions

- ☐ Regulatory actions (*e.g.*, date and description of Records of Decision, Explanations of Significant Difference, Administrative Orders on Consent, Consent Decrees and Action Memorandum)
- ☐ Remedial action objectives
- ☐ Remedy description
- ☐ Remedy implementation (*e.g.*, status, history, enforcement actions, performance)
- ☐ Systems operations/Operations & Maintenance
  - ☐ Systems operations/O&M requirements
  - ☐ Systems operations/O&M operational summary (*e.g.*, history, modifications, problems, and successes)
  - ☐ Summary of costs of system operations/O&M effectiveness (*i.e.*, are requirements being met and are activities effective in maintaining the remedy?)

Progress Since Last Five-Year Review (if applicable)

- ☐ Protectiveness statements from last review
- ☐ Status of recommendations and follow-up actions from last review
- ☐ Results of implemented actions, including whether they achieved the intended effect
- ☐ Status of any other prior issues

Five-Year Review Process

- ☐ Administrative Components
  - ☐ Notification of potentially interested parties of initiation of review process
  - ☐ Identification of five-year review team members (as appropriate)
  - ☐ Outline of components and schedule of your five-year review
- ☐ Community Involvement
  - ☐ Community notification (prior and post review)
  - ☐ Other community involvement activities (*e.g.*, notices, fact sheets, etc., as appropriate)
- ☐ Document review
- ☐ Data review
- ☐ Site inspection
  - ☐ Inspection date
  - ☐ Inspection participants

Five-Year Review Process, cont'd.

- ☐ Site inspection scope and procedures
- ☐ Site inspection results, conclusions
- ☐ Inspection checklist

☐ Interviews

- ☐ Interview date(s) and location(s)
- ☐ Interview participants (name, title, etc.)
- ☐ Interview documentation
- ☐ Interview summary

Technical Assessment

☐ Answer Question A: Is the remedy functioning as intended by the decision documents?

- ☐ remedial action performance (*i.e.*, is the remedy operating as designed?)
- ☐ system operations/O&M
- ☐ cost of system operations/O&M
- ☐ opportunities for optimization
- ☐ early indicators of potential issues
- ☐ implementation of institutional controls and other measures

☐ Answer Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?

- ☐ changes in standards, newly promulgated standards, TBCs
- ☐ expected progress towards meeting RAOs
- ☐ changes in exposure pathways
- ☐ changes in land use
- ☐ new contaminants and/or contaminant sources
- ☐ remedy byproducts
- ☐ changes in toxicity and other contaminant characteristics
- ☐ risk recalculation/assessment (as applicable)

☐ Answer Question C: Has any other information come to light that could call into question the protectiveness of the remedy?

- ☐ new or previously unidentified ecological risks
- ☐ natural disaster impacts
- ☐ any other information that could call into question the protectiveness of the remedy

☐ Technical Assessment Summary

Issues

- ☐ Issues identified during the technical assessment and other five-year review activities
- ☐ Determination of whether issues affect current or future protectiveness

Issues, cont'd.

- ☐ A discussion of unresolved issues raised by support agencies and the community (States, Tribes, other Federal agencies, local governments, citizens, PRPs, other interested parties), if applicable

Recommendations and Follow-up Actions

- ☐ Required/suggested improvements to identified issues or to current site operations
- ☐ Note parties responsible for actions
- ☐ Note agency with oversight authority
- ☐ Schedule for completion of actions related to resolution of issues

Protectiveness Statements

- ☐ Protective statement(s) for each OU (If the remedy is not protective of human health and/or the environment, have you provided supporting discussion and information in the report to make this determination, such as current threats or level of risk?)
- ☐ Comprehensive protectiveness statement covering all of the remedies at the site (if applicable)

Next Review

- ☐ Expected date of next review
- ☐ If five-year reviews will no longer be done, provide a summary of that portion of the technical analysis presented in the report that provides the rationale for discontinuation of five-year reviews

Five-Year Review Report

(First, Second, etc.) Five-Year Review Report

for

Site Name

City

County, State

Month, Year

PREPARED BY:

Lead Agency
Name and
Location

Approved by: Date:

_____ _____

[Name]
[Title]
[Affiliation]

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Five-Year Review Report

The following Table of Contents notes typical major divisions and subheadings for Five-Year Review reports. Subheadings can be included as appropriate for a given review report. This is only a general example.

Table of Contents

Tables

Attachments

Appendix

List of Acronyms

You should include a list of acronyms used in the report here.

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Executive Summary

You should include an Executive Summary at the beginning of the report. The Executive Summary should be brief, and should include a reiteration of the protectiveness statements included in Section X of the Five-Year Review report.

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Five-Year Review Summary Form

SITE IDENTIFICATION

Site name ***(from WasteLAN)*****:**

EPA ID ***(from WasteLAN)*****:**

Region:	**State:**	**City/County:**

SITE STATUS

NPL status: G Final G Deleted G Other (specify) _____

Remediation status (choose all that apply): G Under Construction G Operating G Complete

Multiple OUs?* G YES G NO	**Construction completion date:** ___ / ___ / _____

Has site been put into reuse? G YES G NO

REVIEW STATUS

Lead agency: G EPA G State G Tribe G Other Federal Agency _____

Author name:

Author title:	**Author affiliation:**

Review period:** ___ / ___ / _____ to ___ / ___ / _____

Date(s) of site inspection: ___ / ___ / _____

Type of review:

G Post-SARA G Pre-SARA G NPL-Removal only
G Non-NPL Remedial Action Site G NPL State/Tribe-lead
G Regional Discretion

Review number: G 1 (first) G 2 (second) G 3 (third) G Other (specify) _____

Triggering action:

G Actual RA Onsite Construction at OU #____ G Actual RA Start at OU#____
G Construction Completion G Previous Five-Year Review Report
G Other (specify) _____

Triggering action date ***(from WasteLAN)*****:** ___ / ___ / _____

Due date ***(five years after triggering action date)*****:** ___ / ___ / _____

* ["OU" refers to operable unit.]
** [Review period should correspond to the actual start and end dates of the Five-Year Review in WasteLAN.]

Five-Year Review Summary Form, cont'd.

Issues:

Summarize issues (see Chapter 3).

Recommendations and Follow-up Actions:

Summarize recommendations and follow-up actions (see Chapter 3).

Protectiveness Statement(s):

Include individual operable unit protectiveness statements. For sites that have reached construction completion and have more than one OU, include an additional and comprehensive protectiveness statement covering all of the remedies at the site (see Chapter 4).

Other Comments:

Make any other comments here.

Five-Year Review Report

I. Introduction

Provide a synopsis of "who, what, where, when, and why." Detail the following:

- *The purpose of the review;*
- *The authority for conducting the five-year review;*
- *Who conducted the review, when, and for what site or portion of the site;*
- *Whether it is the first review or a subsequent review at the site;*
- *What action triggered the review; and*
- *A brief status of areas of a site not addressed in the current review and/or the status of five-year reviews for other areas of the entire site.*

Further explanation and boilerplate text are provided below. Additional explanation on the following topics is provided in Chapter 1.

The Purpose of the Review

State the purpose of the five-year review specific to the site or portion of the site addressed in the review.

The purpose of five-year reviews is to determine whether the remedy at a site [is/is expected to be] protective of human health and the environment. The methods, findings, and conclusions of reviews are documented in Five-Year Review reports. In addition, Five-Year Review reports identify issues found during the review, if any, and recommendations to address them.

Authority for Conducting the Five-Year Review

The Agency is preparing this five-year review pursuant to CERCLA §121 and the National Contingency Plan (NCP). CERCLA §121 states: *If the President selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site, the President shall review such remedial action no less often than each five years after the initiation of such remedial action to assure that human health and the environment are being protected by the remedial action being implemented. In addition, if upon such review it is the judgment of the President that action is appropriate at such site in accordance with section [104]*

> *or [106], the President shall take or require such action. The President shall report to the Congress a list of facilities for which such review is required, the results of all such reviews, and any actions taken as a result of such reviews.*
>
> The agency interpreted this requirement further in the National Contingency Plan (NCP); 40 CFR §300.430(f)(4)(ii) states:
>
> *If a remedial action is selected that results in hazardous substances, pollutants, or contaminants remaining at the site above levels that allow for unlimited use and unrestricted exposure, the lead agency shall review such action no less often than every five years after the initiation of the selected remedial action.*

Who Conducted the Five-Year Review

If the U.S. Army Corps of Engineers (USACE) or a contractor has conducted an analysis in support of a five-year review, you should include their name and the date of the analysis. When a contractor for a potentially responsible party (PRP) conducts analyses or provides information in support of a five-year review, you should identify the a contractor and their affiliation with the PRP in the Five-Year Review report. You should also identify who conducted the site inspection.

Boilerplate text for the explanation of who conducted the review is provided in the box below. This text is written as though EPA is the lead agency and should be adapted when another agency or department serves as the lead agency.

> The United States Environmental Protection Agency (EPA) Region [number] has conducted a five-year review of the remedial actions implemented at the [name] site in [location]. This review was conducted from [month, year] through [month, year]. This report documents the results of the review. [Please identify any party providing an analysis in support of the five-year review; also indicate the contractual arrangements under which this was done.]

Other Review Characteristics

State whether the review is the first or a subsequent five-year review for the site, what action or event "triggered" the review, and the date of this action. See Chapter 1, Section 1.2 of this guidance for a discussion of triggering events for the five-year review and indicate in your report whether the trigger for the current five-year review has been met.

Boilerplate text for the explanation of other review characteristics is provided in the box below. Select text from brackets as appropriate.

This is the [first/second/etc.] five-year review for the [name] site. The triggering action for this review is the date of the [triggering action], as shown in EPA's WasteLAN database: [date]. [This discussion should also mention what is specifically activating the review, *i.e.*, that hazardous substances, pollutants, or contaminants are or will be left on site above levels that allow for unlimited use and unrestricted exposure.]

In addition, if separate five-year reviews are conducted for different areas of a site, you should include the following in this section:

- *An explanation of this approach;*
- *A description of which areas are covered by this five-year review; and*
- *A brief synopsis of the remedial activities and the status of remedial measures and/or five-year reviews for other areas.*

II. Site Chronology

List all important site events and relevant dates in the site chronology, such as those shown in Table 1. The identified events are illustrative, not comprehensive.

Table 1: Chronology of Site Events

Event	Date
Initial discovery of problem or contamination	
Pre-NPL responses	
NPL listing	
Removal actions	
Remedial Investigation/Feasibility Study complete	
ROD signature	
ROD Amendments or ESDs	
Enforcement documents (CD, AOC, Unilateral Administrative Order)	
Remedial design start	
Remedial design complete	

Table 1: Chronology of Site Events

Event	Date
Superfund State Contract, Cooperative Agreement, or Federal Facility Agreement signature	
Actual remedial action start	
Construction dates (start, finish)	
Construction completion date	
Final Close-out Report	
Deletion from NPL	
Previous five-year reviews	

III. Background

Describe the fundamental aspects of the site, providing a clear, succinct description of site characteristics. The purpose of this section is to identify the threat posed to the public and environment at the time of the ROD, so that the performance of the remedy can be easily compared with the site conditions the remedy was intended to address. Include all major site activities prior to the signing of the ROD. In addition to text, you may use site maps to help clarify the discussion. The following checklist may assist you in developing the text for this section.

	Background Checklist
Physical Characteristics *Present the site's location and characteristics, including the following:*	
	Area of site, relation to parcel(s), extent and location of sources
	Whether site is located in a populated area or is near populated areas
	Whether site is located in an environmentally sensitive area or is near environmentally sensitive areas, where applicable
Land and Resource Use *Discuss the following:*	
	Former, current and projected land uses for the site, as identified in the ROD or other decision document
	Current and projected land uses for the area surrounding the site, at the time of the five-year review
	Human and ecological past, present and known future use of resources (*e.g.*, groundwater or surface water as a drinking water supply) and any other current uses of the site not already addressed, as applicable

	Background Checklist
History of Contamination *Discuss the following:*	
	The historical activities that caused contamination, including the type of activity or process, when it took place, the specific type of hazardous substances, and their volumes/proportions, if known
	How contamination was discovered and problems resulting from contamination
Initial Response *Describe any pre-ROD cleanup activities at the site:*	
	CERCLA removal actions, non-CERCLA removals/responses, closures, the ceasing of operations, as well as governing agreements and parties involved in these activities
Basis for Taking Action *Describe the contaminants found at the site by appropriate media type (soil, groundwater, surface water, air). Note the effect or potential effect of the contamination on people, resources they use, or the environment. Examples of elements of this discussion include the following:*	
	Contaminated media and structures (summary of remedial investigation)
	Resources/targets that have been or could potentially be affected, results of risk assessments, determination of primary health threat

IV. Remedial Actions

Discuss initial plans, implementation history, and current status of the remedy. Explain events identified in the chronology, and generally include discussions of remedy selection, remedy implementation, remedy performance, and system operations/O&M. Present – accurately, adequately, and concisely – relevant site activities from the signing of the ROD to the present. You should delineate all remedial measures, for instance, include monitoring, fencing, and institutional controls. Discuss any changes to or problems with remedial components. The following checklist may assist you in developing the text for this section.

	Remedial Actions Checklist
Remedy Selection *Describe the remedial action objectives and the selected remedy. This discussion should explain the following:*	
	Scope and role of actions including definition of OUs related to each ROD and how they relate to each other
	Source documents listing remedial action objectives and the remedy (e.g., RODs, ESDs), including signature/filing date
	Statement of remedial action objectives, related to each OU or ROD
	Description of remedial actions/remedy, related to each OU or ROD, noting media addressed; all components of the remedy, including engineering controls, access controls, institutional controls, cleanup measures, treatment types, and required monitoring should be described

	Remedial Actions Checklist
Remedy Implementation *Discuss the history of and plans for implementation of the remedy. Discuss enforcement actions if applicable. The text may be presented either chronologically or by OU, and should include the following:*	
	Dates when remedial designs were started and completed
	Difficulties or changes that occurred during remedial design
	Dates when remedial actions were started and completed
	The performance of each remedial action since implementation
	Enforcement agreements, and parties involved in these agreements
	CERCLA removal actions or non-CERCLA removals/responses since the ROD
System Operations/O&M *Describe system operations/O&M requirements, activities to date, any problems that have arisen, and costs:*	
	System operations/O&M requirements, as noted in the system operations/O&M plan, system operations/O&M manual, enforcement documents, and monitoring plans
	System operations/O&M activities to date
	Problems in the implementation of system operations/O&M
	Originally estimated annual O&M costs
	Actual annual O&M costs over the review period
	Reasons for any unanticipated or unusually high O&M costs

A table, such as Table 2, should be used to document total annual system operations/O&M costs during the period preceding the current five-year review. In the text, you should discuss significant variations from anticipated costs or between operating years.

Table 2: Annual System Operations/O&M Costs

Dates		Total Cost rounded to nearest $1,000
From	To	

At the end of the remedial actions section, it is sometimes helpful for you to add a brief discussion of the current status of each of the components of the remedy. This discussion can be particularly helpful for large, complex sites.

V. Progress Since the Last Review

Progress since the last review should be discussed when follow-up actions which impact protectiveness were noted in the previous Five-Year Review report. The following checklist may assist you in developing the text for this section.

	Progress Since the Last Review Checklist
Describe progress toward accomplishing recommendations and follow-up actions since the last five-year review was completed. Include the following:	
	Protectiveness statements from the last review
	Status of recommendations and follow-up actions from last review
	Results of implemented actions, including whether they achieved the intended effect
	Status of any other prior issues

Table 3 below presents one approach for providing information on the recommendations and follow-up actions stated in the past review and subsequent actions. The accompanying text should also discuss why any recommendations and follow-up actions have not been implemented if that is the case, and whether implemented actions achieved desired results.

Table 3: Actions Taken Since the Last Five-Year Review

Issues from Previous Review	Recommendations/ Follow-up Actions	Party Responsible	Milestone Date	Action Taken and Outcome	Date of Action

VI. Five-Year Review Process

Describe activities performed during the five-year review process and provide a summary of findings when appropriate. The following checklist may assist you in developing the text for this section.

	Five-Year Review Process Checklist
Administrative Components of the Five-Year Review Process	
	Notify potentially interested parties of start of five-year review
	Identify members of the review team
	Develop a review schedule

	Five-Year Review Process Checklist
Community Notification and Involvement	
	Community notification
	Other community involvement activities
Document Review See Appendix B for a full discussion of the document review	
	What documents were reviewed
	Identify document source of RAOs, ARARs and cleanup levels
Data Review *Discuss and present the following:*	
	What data were reviewed
	Relevant trends and levels, noting levels which are not currently compliant and whether future compliance can be expected without additional action
	Tables summarizing monitoring and sampling data
	Increase and/or decrease or non-presence of specific chemical compounds and recommended changes for future monitoring programs
Site Inspection *Summarize the site inspection and site conditions:*	
	Date of site inspection (if more than one inspection was conducted to allow for monitoring or further inspection, list all inspections and activities conducted, and the reasons for conducting each inspection)
	Who conducted and/or attended the inspection
	Activities conducted (scope and procedures)
	Summary of site conditions, inspection results, conclusions
Interviews *Discuss the following:*	
	Interviews conducted (name, title, organization, date, location(S))
	Interview documentation
	Interview summary
	Successes/problems in the implementation of access and institutional controls
	Successes/problems with the construction of the remedy
	Successes/problems with system operations/O&M
	Unusual situations or problems at the site

VII. Technical Assessment

Discuss how each of the three questions asked in the technical assessment were answered (e.g., yes, yes, no or a variation of this) and provide the information that presents the basis for each answer as a framework for your protectiveness determination(s). Explain the conclusions of

your review, based on the information presented in the previous section. As explained in Chapter 4, the assessment should focus on answering three key questions:

- *Question A: Is the remedy functioning as intended by the decision documents?*
- *Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of remedy selection still valid?*
- *Question C: Has any other information come to light that could call into question the protectiveness of the remedy?*

Each question, and the associated information to be discussed, is presented in its own checklist which may assist you in developing the text for this section. Checklist items shown may be supplemented or modified based on site-specific circumstances.

	Checklist for Question A: Is the remedy functioning as intended by the decision documents?
Remedial Action Performance *Discuss the following:*	
	Whether the remedial action continues to be operating and functioning as designed
	Whether the remedial action is performing as expected and cleanup levels are being achieved
	Whether containment is effective
System Operations/O&M *Discuss the following:*	
	Whether operating procedures, as implemented, will maintain the effectiveness of response actions
	Whether large variances in O&M costs could indicate a potential remedy problems or remedy issues
Opportunities for Optimization *Discuss the following:*	
	Whether opportunities exist to improve the performance and/or reduce costs of monitoring, sampling, and treatment systems
Early Indicators of Potential Issues *Discuss the following:*	
	Whether frequent equipment breakdowns or changes indicate a potential issue
	Whether issues or problems could place protectiveness at risk
Implementation of Institutional Controls and Other Measures *Discuss the following:*	
	Whether access controls are in place and prevent exposure (*e.g.*, fencing and warning signs)
	Whether institutional controls are in place and prevent exposure
	Whether other actions (*e.g.*, removals) necessary to ensure that immediate threats have been addressed are complete

	Checklist for Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of remedy selection still valid?
Changes in Standards and TBCs *Discuss the following:*	
	Whether standards identified in the ROD have been revised and call into question the protectiveness of the remedy
	Whether newly promulgated standards call into question the protectiveness of the remedy
	Whether TBCs used in selecting cleanup levels at the site have changed and could affect the protectiveness of the remedy
Changes in Exposure Pathways *Discuss the following:*	
	Whether land use or expected land use on or near the site changed
	Whether human health or ecological routes of exposure or receptors have been newly identified or changed in a way that could affect the protectiveness of the remedy
	Whether there are newly identified contaminants or contaminant sources
	Whether there are unanticipated toxic byproducts of the remedy not previously addressed by the decision documents
	Whether physical site conditions or the understanding of these conditions have changed in a way that could affect the protectiveness of the remedy
Changes in Toxicity and Other Contaminant Characteristics *Discuss the following:*	
	Whether toxicity factors for contaminants of concern at the site have changed in a way that could affect the protectiveness of the remedy
	Whether other contaminant characteristics have changed in a way that could affect the protectiveness of the remedy
Changes in Risk Assessment Methods *Discuss the following:*	
	Whether standardized risk assessment methodologies have changed in a way that could affect the protectiveness of the remedy
Expected Progress Towards Meeting RAOs	
	Whether the remedy is progressing as expected

When a standard or requirement has changed, a table can be used to record the nature of the change. Tables 4, 5, and 6 below demonstrate potential ways for you to note changes in chemical-specific, action-specific, or location-specific requirements, respectively.

Table 4: Changes in Chemical-Specific Standards

Contaminant	Media	Cleanup Level	Standard		Citation/Year
Chemical A	*e.g.*, groundwater	*e.g.*, 0.XX mg/L	Previous	*e.g.*, 0.XX mg/L	*e.g.*, SDWA 1988
			New	*e.g.*, 0.YY mg/L	*e.g.*, SDWA 1995
Chemical B			Previous		
			New		

Table 5: Changes in Action-Specific Requirements

Action	Requirement		Prerequisite	Citation/Year
Action A (*e.g.*, landfill)	Previous	Include original ARAR here; if none applies, state "None"		
	New			

Table 6: Changes in Location-Specific Requirements

Location	Requirement		Prerequisite	Citation/Year
Location A (*e.g.*, critical habitat upon which endangered or threatened species depend)	Previous	Include original ARAR here; if none applies, state "None"		
	New			

	Checklist for Question C: Has any other information come to light that could call into question the protectiveness of the remedy?
Other Information *Discuss the following:*	
	Whether newly identified ecological risks been found
	Whether there are impacts from natural disasters
	Whether any other information has come to light which could affect the protectiveness of the remedy

Technical Assessment Summary

Discuss how each of the three questions were answered and provide the information that presents the basis for each answer as a framework for your protectiveness determination(s).

VIII. Issues

Detail issues related to current site operations, conditions, or activities, noting which issue, if any, currently prevent the remedy from being protective. You may use a table such as Table 7 to note the issues identified.

Table 7: Issues

Issues	Affects Current Protectiveness (Y/N)	Affects Future Protectiveness (Y/N)

IX. Recommendations and Follow-up Actions

Specify the required and suggested improvements to current site operations, activities, remedy, or conditions. Note the parties responsible for actions, milestone dates, and which agencies have oversight authority. At a minimum, address all issues that currently affect current and/or future protectiveness. Table 8 illustrates one way to include the necessary information.

Table 8: Recommendations and Follow-up Actions

Issue	Recommendations and Follow-up Actions	Party Responsible	Oversight Agency	Milestone Date	Affects Protectiveness (Y/N)	
					Current	Future

X. Protectiveness Statement(s)

Include a protectiveness statement for each OU at which a remedial action has begun. For sites that have reached construction completion and have more than one OU, you should develop and include an additional comprehensive site-wide protectiveness statement covering all of the remedies at the site. You should not include this additional protectiveness statement until construction completion because, until then, all remedies at the site have not necessarily been selected and constructed.

In order to promote consistency, you are strongly encouraged to model your protectiveness statements on the sample protectiveness statements provided in Chapter 4, Exhibits 4-6 and 4-7. Your Five-Year Review report should present the protectiveness statements at the beginning of a

discussion that should explain and provide the supporting rationale of the protectiveness determination.

Suggested statements are as follows:

If the remedial action at the OU is under construction, then use this statement:

Protective or will be protective:

"The remedy at OU X is expected to be protective of human health and the environment upon completion, and in the interim, exposure pathways that could result in unacceptable risks are being controlled."

Not protective:

"The remedy at OU X is not protective because of the following issues [describe the issue(s)]. The following actions need to be taken [describe the actions needed to ensure protectiveness]."

Protectiveness deferred:

"A protectiveness determination of the remedy at OU X cannot be made at this time until further information is obtained. Further information will be obtained by taking the following actions [describe the actions]. It is expected that these actions will take approximately [insert time frame] to complete, at which time a protectiveness determination will be made."

If the remedial action at the OU is operating or completed:

Protective:

"The remedy at OU X is expected to be or is protective of human health and the environment, and in the interim, exposure pathways that could result in unacceptable risks are being controlled."

Protective in the short-term:

"The remedy at OU X currently protects human health and the environment because [describe the elements of the remedy that protect human health and the environment in the short term]. However, in order for the remedy to be protective in the long-term, the following actions need to be taken [describe the actions needed to ensure long-term protectiveness]."

Not protective:

"The remedy at OU X is not protective because of the following issue(s) [describe the issue(s)]. The following actions need to be taken [describe the actions needed to ensure protectiveness].

Protectiveness deferred:

"A protectiveness determination of the remedy at OU X cannot be made at this time until further information is obtained. Further information will be obtained by taking the following actions [describe the actions]. It is expected that these actions will take approximately [insert time frame] to complete, at which time a protectiveness determination will be made."

For Sites That Have Reached Construction Completion:

If the remedy(s) is/are protective then use:

"Because the remedial actions at all OUs are protective, the site is protective of human health and the environment."

If the remedy is not protective then use:

"The remedial actions at OUs X and Y are protective. However, because the remedial action at OU Z is not protective, the site is not protective of human health and the environment at this time. The remedial action at OU Z is not protective because of the following issue(s) [describe the issue(s)]. The following actions need to be taken [describe the actions needed to ensure protectiveness]."

XI. Next Review

Discuss whether another five-year review will be conducted and the date on which that report will be due. If no additional five-year reviews are to be conducted, explain why and provide a justification for discontinuation of reviews.

Attachments

Site Maps (if not included in the body of the report)
List of Documents Reviewed
Tables and Figures Documenting Remedy Performance and Changes in Standards
(If not included in the body of the report)
Interview Report (as appropriate)
Photos Documenting Site Conditions

Appendix

Comments received from Support Agencies and/or the community

Appendix F
Sample Five-Year Review Report

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Five-Year Review Report

First Five-Year Review Report
for
Acme Superfund Site
Town of Riverside
Waters County, Massachusetts

September 2000

PREPARED BY:

United States Environmental Protection Agency
Region 1
Boston, Massachusetts

(This is a hypothetical site. However, the site characteristics were taken from an actual site in the Superfund program.)

Approved by: Date:

Robert Webster *September 11, 2000*

Robert Webster
Superfund Division Director
U.S. EPA, Region 1

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Table of Contents

List of Acronyms

ARAR	Applicable or Relevant and Appropriate Requirement
CAMU	Corrective Action Management Unit
CD	Consent Decree
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
EPA	United States Environmental Protection Agency
CFR	Code of Federal Regulations
DEQE	Massachusetts Department of Environmental Quality Engineering
ESD	Explanation of Significant Difference
MADEP	Massachusetts Department of Environmental Protection
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
NCP	National Contingency Plan
NPL	National Priorities List
O&M	Operation and Maintenance
PAH	Polyaromatic Hydrocarbon
PCB	Polychlorinated Biphenyl
PRP	Potentially Responsible Party
PSD	Performing Settling Defendant
RA	Remedial Action
RAO	Remedial Action Objective
RD	Remedial Design
RI/FS	Remedial Investigation/Feasibility Study
ROD	Record of Decision
SDWA	Safe Drinking Water Act
VOC	Volatile Organic Compound

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Executive Summary

The remedy for the Acme Superfund site in Riverside, Massachusetts included stabilization and capping of contaminated soils and sediments on site, institutional controls, and monitored natural attenuation of contaminated groundwater. The site achieved construction completion with the signing of the Preliminary Close Out Report on August 28, 1998. The trigger for this five-year review was the actual start of construction on September 12, 1995.

The assessment of this five-year review found that the remedy was constructed in accordance with the requirements of the Record of Decision (ROD). One Explanation of Significant Difference (ESD) was issued to change the cap design and the treatment approach of soils and sediments. The remedy is functioning as designed. The immediate threats have been addressed and the remedy is expected to be protective when groundwater cleanup goals are achieved through monitored natural attenuation, which is expected to require 10 years.

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Five-Year Review Summary Form

SITE IDENTIFICATION		
Site name *(from WasteLAN)*: Acme Superfund Site		
EPA ID *(from WasteLAN)*: MADXXXXXXX		
Region: 1	**State:** MA	**City/County:** Riverside/Waters
SITE STATUS		
NPL status: ☒ Final ☐ Deleted ☐ Other (specify)		
Remediation status (choose all that apply): ☐ Under Construction ☐ Operating ☒ Complete		
Multiple OUs?* ☐ YES ☒ NO	**Construction completion date:** 8 / 28 / 1998	
Has site been put into reuse? ☐ YES ☒ NO		
REVIEW STATUS		
Lead agency: ☒ EPA ☐ State ☐ Tribe ☐ Other Federal Agency _____		
Author name: Mary Jones		
Author title: Remedial Project Manager	**Author affiliation:** U.S. EPA, Region 1	
Review period:** 3 / 1 / 2000 to 8 / 31 / 2000		
Date(s) of site inspection: 3 / 12 / 2000 & 5 / 23 / 2000		
Type of review: ☒ Post-SARA ☐ Pre-SARA ☐ NPL-Removal only ☐ Non-NPL Remedial Action Site ☐ NPL State/Tribe-lead ☐ Regional Discretion)		
Review number: ☒ 1 (first) ☐ 2 (second) ☐ 3 (third) ☐ Other (specify)		
Triggering action: ☐ Actual RA On-site Construction at OU #___ ☒ Actual RA Start at OU# NA ☐ Construction Completion ☐ Previous Five-Year Review Report ☐ Other (specify)		
Triggering action date *(from WasteLAN)*: 9 / 12 / 1995		
Due date *(five years after triggering action date)*: 9 / 12 / 2000		

* ["OU" refers to operable unit.]

** [Review period should correspond to the actual start and end dates of the Five-Year Review in WasteLAN.]

Five-Year Review Summary Form, cont'd.

Issues:

Burrowing animals were observed to have left minor tunnels in cap soil, and a portion of the constructed wetlands have not been properly maintained.

Failure to maintain a portion of the constructed wetlands due to restricted access to the property.

Inadequate monitoring to verify that the plume is not migrating.

Recommendations and Follow-up Actions:

The burrows are scheduled to be repaired. The State and Potentially Settling Defendants (PSDs) are actively seeking an alternate location for wetlands development.

Identify an alternate location for wetlands development.

Increase monitoring frequency for MW-103; Investigate groundwater discharge to river; sample sediments and groundwater at discharge points.

Protectiveness Statement(s):

All immediate threats at the site have been addressed, and the remedy is expected to be protective of human health and the environment after the groundwater cleanup goals are achieved through MNA in an estimated 10 years.

Long-term Protectiveness:

Long-term protectiveness of the remedial action will be verified by obtaining additional groundwater samples to fully evaluate potential migration of the contaminant plume downgradient from the treatment area and towards the river. Current data indicate that the plume remains on site. Additional sampling and analysis will be completed within the next six months. Current monitoring data indicate that the remedy is functioning as required to achieve groundwater cleanup goals.

Other Comments:

The problems encountered in maintaining the wetlands result from access issues that will be resolved once an alternative location for development of wetlands is identified. This issue does not impact protectiveness and is expected to be resolved within the current year.

Acme Superfund Site
Riverside, Massachusetts
First Five-Year Review Report

I. Introduction

The purpose of the five-year review is to determine whether the remedy at a site is protective of human health and the environment. The methods, findings, and conclusions of reviews are documented in Five-Year Review reports. In addition, Five-Year Review reports identify issues found during the review, if any, and identify recommendations to address them.

The Agency is preparing this Five-Year Review report pursuant to CERCLA §121 and the National Contingency Plan (NCP). CERCLA §121 states:

> *If the President selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site, the President shall review such remedial action no less often than each five years after the initiation of such remedial action to assure that human health and the environment are being protected by the remedial action being implemented. In addition, if upon such review it is the judgement of the President that action is appropriate at such site in accordance with section [104] or [106], the President shall take or require such action. The President shall report to the Congress a list of facilities for which such review is required, the results of all such reviews, and any actions taken as a result of such reviews.*

The Agency interpreted this requirement further in the NCP; 40 CFR §300.430(f)(4)(ii) states:

> *If a remedial action is selected that results in hazardous substances, pollutants, or contaminants remaining at the site above levels that allow for unlimited use and unrestricted exposure, the lead agency shall review such action no less often than every five years after the initiation of the selected remedial action.*

The United States Environmental Protection Agency (EPA), Region 1, conducted the five-year review of the remedy implemented at the Acme Superfund Site in Riverside, Massachusetts. This review was conducted by the Remedial Project Manager (RPM) for the entire site from March 2000 through August 2000. This report documents the results of the review.

This is the first five-year review for the Acme Site. The triggering action for this statutory review is the initiation of the remedial action on September 12, 1995. The five-year review is required due to the fact that hazardous substances, pollutants, or contaminants remain at the site above levels that allow for unlimited use and unrestricted exposure.

II. Site Chronology

Table 1 - Chronology of Site Events

Event	Date
Waste oil and solvent recovery activities at the site	1974 - 1978
Massachusetts Department of Environmental Quality Engineering (DEQE) (now Massachusetts Department of Environmental Protection or MADEP), initiates actions against facility owners resulting in closing of facility	1978
Removal activities - removing drums, liquids and sludge from tanks	1978 - 1984
Final listing on EPA National Priorities List	9/1983
Interim removal activities - Demolition and removal of remaining storage tanks and waste material contained in tanks	1986
Remedial Investigation/Feasibility Study (RI/FS) made available to public	1/1992
Proposed plan identifying EPA's preferred remedy presented to public; start of public comment period.	3/1992
ROD selecting the remedy is signed	9/30/1992
Consent Decree finalizing settlement for responsible party performance of remedy entered by Federal Court	9/18/1994
Start of on-site construction for building/structures demolition and decontamination (1st phase of site Remedial Action and date that triggers a five-year review).	9/12/1995
Completion of on-site construction for building/structures demolition and decontamination	12/28/1995
ESD issued by EPA, primarily changing soil and sediment stabilization from "in-situ" to "ex-situ", and changing cap design	11/26/1996
PRP Remedial Design approved by EPA	3/5/1997
Start of on-site construction for stabilization remedy (2nd phase of site Remedial Action)	3/11/1997
Pre-final inspection of Phase II remedial action	11/19/1997
Preliminary Close Out Report signed	8/28/1998
O & M Plan approved by EPA	9/18/1998

III. Background

Physical Characteristics

The Acme Site property includes a four-acre facility located on Canal Street adjacent to and upgradient of the Green River in Riverside, Massachusetts. Riverside is a community of approximately 12,000 residents, located in Waters County. In addition to the facility, the site includes the adjacent wetlands, wooded area, and the immediately adjacent portion of the river. The facility is located 200 feet northeast of the Green River and is within the river's 100-year flood zone. The site is bordered by Canal Street, wetlands and woodlands, the Green River, and a soccer field. Residential and commercial properties are located across Canal Street from the site (See Attachment 1).

Land and Resource Use

The historic land use of the site has involved some petroleum- or solvent-related industry since at least 1900. From at least 1974 until operations ceased in 1978, activities at the site included waste oil and solvent recovery and disposal. Since 1978, the facility has been inactive.

The current land use for the surrounding area is residential, commercial and recreational (the adjacent soccer field). The Green River is used for swimming and fishing. Although there have been a number of zoning changes over the years, it is anticipated that a mix of land uses similar to that described will continue into the future. In establishing cleanup requirements for the site, EPA considered the theoretical possibility of residential development at the site. The site itself is currently fenced and the treated, stabilized soils and sediments are contained within the fenced area under an impermeable cap.

The groundwater aquifer underlying the site is currently not used as a drinking water source. The dominant groundwater flow direction is to the southwest toward the Green River.

History of Contamination

The Acme facility reclaimed used oils and solvents from State collection points, treated them with a heat process, and sold them as lube oil and heavy fuel mixtures. In the course of these operations, spills occurred causing contamination of soils, sediments, and groundwater. Contamination in groundwater at the site consists primarily of volatile organic compounds (VOCs), including benzene and methylene chloride. Contaminants in soils and sediments include polychlorinated biphenyls (PCBs), polyaromatic hydrocarbons (PAHs), VOCs, and other organics and lead. Contamination at the site was discovered in the course of several property inspections conducted by the State which documented improper maintenance, as well as waste oil and hazardous materials spills. Millions of gallons of waste were left behind in tanks and lagoons when the owner abandoned the facility in 1978.

Initial Response

From 1978 to 1984, as a result of State enforcement efforts, approximately 1.5 million gallons of waste material were removed from the site during a number of separate events. In 1982, the State requested assistance from EPA's Superfund program. EPA discovered several leaking tanks and contaminated ditches, as well as saturated soils. The site was proposed for the National Priorities List

(NPL) on December 30, 1982, and finalized on the NPL in March 1983. In 1986, interim measures were taken to establish complete fencing of the site, demolish and dispose of 19 storage tanks, dispose of the oil and water contained in the tanks, and dispose of sludge generated during the cleaning of tanks. In January 1992, the Remedial Investigation/Feasibility Study was made available to the public. In March 1992, the Proposal Plan identifying EPA's preferred remedy was presented to the public, starting the period for public comment.

Basis for Taking Action

Contaminants

Hazardous substances that have been released at the site in each media include:

Soil

PCBs
PAHs
1,1-Dichloroethane
Cis-1,2-Dichloroethylene
Trans-1,2-Dichloroethylene
1,1,1-Trichlorethane
Trichloroethylene
Tetrachloroethylene
Benzene
Lead

Groundwater

Bis (2-ethylhexyl) Phthalate
Vinyl Chloride
1,1-Dichloroethane
Cis-1,2-Dichloroethylene
Trans-1,2-Dichloroethylene
1,1,1-Trichlorethane
Methylene Chloride
Trichloroethylene
Tetrachloroethylene
Benzene
2-Butanone (MEK)
Acetone
Lead

Lagoon Sediment

Bis (2-ethylhexyl) Phthalate
PAHs
1,1-Dichlorethane
1,1,1-Trichloroethane
Trichloroethylene
Tetrachloroethylene
Methylene Chloride
Benzene
Acetone
Lead

Wetland Sediment

PCBs
PAHs
Arsenic
Lead
Zinc

Exposures to soil, groundwater, wetland sediment, and lagoon sediment are associated with significant human health risks, due to exceedance of EPA's risk management criteria for either the average or the reasonable maximum exposure scenarios. The carcinogenic risks were highest for exposures to lagoon sediments due to the high concentrations of carcinogenic polyaromatic hydrocarbons (PAHs). Non-

carcinogenic hazards were highest for exposure to wetland sediment due to the high concentrations of lead detected in the medium. Risks from exposure to soil were significant due to the presence of TCE, PCE, and PCBs. Potential risks associated with exposure to groundwater are attributed to the presence of a variety of VOC contaminants that exist at concentrations that exceed State and Federal MCLs.

IV. Remedial Actions

Remedy Selection

The ROD for the Acme Site was signed on September 30, 1992. Remedial Action Objectives (RAOs) were developed as a result of data collected during the Remedial Investigation to aid in the development and screening of remedial alternatives to be considered for the ROD. The RAOs for Acme were divided into the following groups:

Source Control Response Objectives

- Minimize the migration of contaminants from the property soils and lagoon sediment that could degrade groundwater quality;
- Reduce risks to human health by preventing direct contact with, and ingestion of, contaminants in the property soils, wetland sediments, and lagoon sediments, and by preventing potential ingestion of contaminated groundwater;
- Reduce risks to the environment by preventing direct contact with, and ingestion of, contaminants in the wetland sediments; and
- Minimize the migration of contaminants (*i.e.*, from property soils, lagoon sediments, and wetland sediments) that could result in surface water concentrations in excess of Ambient Water Quality Criteria.

Management of Migration Response Objectives

- Eliminate or minimize the threat posed to human health and the environment by preventing exposure to groundwater contaminants;
- Prevent further migration of groundwater contamination beyond its current extent; and
- Restore contaminated groundwater to Federal and State applicable or relevant and appropriate requirements (ARARs), including drinking water standards, and to a level that is protective of human health and the environment within a reasonable period of time.

The major components of the source control remedy selected in the ROD include the following:

1. Decontamination, demolition, and off-site disposal of property structures; treatment and discharge of lagoon surface water;
2. Consolidation of contaminated property soils with lagoon and wetland sediments on site property;
3. In-situ mixing and stabilization of property soils/sediments with treatment agents to bind

contaminants into a stable matrix;
4. Construction of a permeable cap over stabilized property soils and sediments, and grading and planting of the cap's surface;
5. Restoration of wetlands;
6. Implementation of institutional controls on groundwater use and land development; and
7. Long-term monitoring of groundwater, wetland sediments, and Green River water and sediments.

The major components of the management of migration remedy selected in the ROD include:

1. Use of monitored natural attenuation (MNA) to achieve groundwater cleanup levels;
2. Groundwater monitoring of existing wells on the Acme property and of monitoring wells adjacent to the property;
3. Sediment sampling of portions of the wetland and the Green River, and where groundwater discharges to the wetland and the Green River;
4. Surface water sampling in areas adjacent to the wetland and in the Green River; and
5. Five-year site reviews to assess site conditions, contaminant distributions, and any associated site hazards.

An ESD was issued on November 26, 1996. Subsurface conditions including the existence of building foundations and low soil workability rendered in-situ stabilization impracticable. Additionally, Potentially Responsible Parties (PRPs) suggested adding a geosynthetic layer to the cap that would make it an impermeable cap rather than a soil cap. EPA approved the recommended change. The primary changes documented in the ESD were:

- Ex-situ stabilization instead of in-situ; and
- Construction of an impermeable cap instead of a permeable cap.

The change to ex-situ stabilization led to the necessity of designating a Corrective Action Management Unit (CAMU) at the site concurrent with the ESD. This designation allowed the handling and temporary storage of contaminated soils and sediments.

Institutional controls are required for the Acme property as well as for the adjacent Town-owned property, the only properties on or near the site requiring institutional controls. These institutional controls are established through the Access and Institutional Controls Agreement between the Performing Settling Defendants (PSDs) and the Town of Riverside, dated October 20, 1994, and recorded on June 19, 1997 in the Waters County Registry of Deeds.

Remedy Implementation

In a Consent Decree (CD) signed with EPA on September 18, 1994, 112 PSDs agreed to perform the remedial design/remedial action (RD/RA) and pay past costs for cleaning up the site. The Remedial Design (RD) was conducted in conformance with the ROD as modified by the ESD. The RD was approved by EPA on March 5, 1997.

The Remedial Action (RA) took place in two phases. The first phase entailed the decontamination, demolition and off-site disposal at a non-hazardous waste landfill of property structures. The activities for this phase were initiated on September 12, 1995 and were completed on December 28, 1995. The major

components of this phase of the RA were the following:

- Decontamination of the buildings and structures on the property;
- Removal, treatment, and discharge to the Green River of water from the basement of one building and water collected from decontamination;
- Collection and analyses of composite samples of buildings and structures;
- Demolition and off-site disposal as non-hazardous waste of property buildings and structures and off-site disposal of miscellaneous debris from the property;
- Removal and off-site disposal of two underground storage tanks and their contents; and
- Restoration of demolition areas to match existing grade.

The second phase entailed all other remedial activities. Components 2 through 7 of the Source Control Remedy constituted the primary activities performed as the second phase of the RA. The activities for the second phase of the RA were formally initiated on March 11, 1997 when the PSDs awarded the RA contract. The contractor conducted remedial activities as planned and EPA and the State conducted a pre-final inspection on November 19, 1997. During this period, 1,606 cubic yards of lagoon sediment, 1,187 cubic yards of wetland sediment, and 8,000 cubic yards of soil were treated, stabilized, and placed under the impermeable cap. In addition, a fence with warning signs and surface water drainage structures were built. At this time, the preparation for the wetland restoration (grading and backfilling of clean sediment material) and the planting of new replacement wetland species was accomplished. The pre-final inspection concluded that construction had been completed in accordance with the remedial design plans and specifications and did not result in the development of a punch list.

The site achieved construction completion status when the Preliminary Close Out Report was signed on August 28, 1998.

EPA and the State have determined that all RA construction activities, including the implementation of institutional controls, were performed according to specifications. It is expected that cleanup levels for all groundwater contaminants will have been reached within approximately ten years. After groundwater cleanup levels have been met, EPA will issue a Final Close Out Report.

System Operation/Operation and Maintenance

The PSDs are conducting long-term monitoring and maintenance activities according to the operation and maintenance (O&M) plan that was approved by EPA on September 8, 1998. The primary activities associated with O&M include the following:

- Visual inspection of the cap with regard to vegetative cover, settlement, stability, and any need for corrective action. In addition, the cap is scheduled to be mowed semi-annually;
- Inspection of the drainage swale for blockage, erosion and instability, and any need for corrective action;

- Inspection of the condition of groundwater monitoring wells;
- Environmental monitoring: Quarterly monitoring of groundwater, wetland surface water and sediment, and Green River surface water and sediment; and
- Engineered wetlands inspection and assessment: Inspections are conducted primarily for the purposes of assessing both weed control needs and the survival of plantings. Assessments are performed specifically to determine if the engineered wetlands are meeting the performance standards regarding the survival and density of desired wetland species.

The primary cleanup of the Acme Site took place during the construction phase of the Remedial Action (*i.e.* the stabilization of contaminated soil and sediments). The other remaining component of cleanup is the natural attenuation of groundwater, as the source of groundwater contamination in soil and sediment has been removed. Therefore, as indicated in the planned elements above, the primary O&M activities have been geared towards monitoring groundwater, surface water, sediments, wetlands, inspections, and maintenance of the cap.

A currently evolving issue exists with regard to the engineered wetlands. The total area of engineered wetlands at the Acme Site is 0.7 acres. This area encompasses wetland habitats that were replanted with appropriate wetland plant species following the removal of contaminated sediments during the RA. As previously mentioned, there are performance standards with regard to density of desired plant species and to minimization of weeds and other undesirable species. The PSDs are obligated to meet these standards. During the course of the O&M period, there have been repeated access issues involving the property abutting the southern border of the Acme property. During the RA, contaminated sediments were removed from this property, clean sediment was backfilled, and wetland plants were planted. Since completion of the RA, the owner of this property has prevented PSD contractors from performing maintenance (weeding and replanting, as necessary) in an area that is highly at risk from invasive species. The area affected by this issue is 0.32 acres. EPA, the Riverside Conservation Commission, and the PSDs are working together to determine if there is additional wetland acreage at the site which may be amenable to restoration or enhancement. If an appropriate area is found, it may be substituted for the 0.32 acre area that is not accessible for maintenance. The failure to provide proper maintenance for the wetlands does not impact the protectiveness of the site.

O&M costs include cap and drainage structure maintenance, sampling and monitoring efforts, monitoring well maintenance, and wetlands maintenance. In the first year, costs were higher due to an extra effort required to establish the vegetative cover on the cap and to establish wetlands. Less effort was required the second year and the PSDs were denied access by a property owner and were not able to maintain all of the wetlands. Costs are expected to rise when additional wetlands are identified and developed. The O&M costs for the first two years are consistent with the originally estimated annual costs of $20,000 per year.

Table 2 - Annual System Operations/O&M Costs

Dates		Total Cost rounded to nearest $1,000
From	To	
9/1998	9/1999	$22,000.00
9/1999	9/2000	$17,000.00

V. Progress Since the Last Five-Year Review

This was the first five-year review for the site.

VI. Five-Year Review Process

Administrative Components

Members of the PSDs and the MADEP were notified of the initiation of the five-year review on February 1, 2000. The Acme Five-Year Review team was led by Mary Jones of EPA, Remedial Project Manager (RPM) for the Acme Site, and included members from the Regional Technical Advisory staff with expertise in hydrology, biology, and risk assessment. Tom McDuff of the State assisted in the review as the representative for the support agency.

From March 1 to March 15, 2000, the review team established the review schedule whose components included:

- Community Involvement;
- Document Review;
- Data Review;
- Site Inspection;
- Local Interviews; and
- Five-Year Review Report Development and Review.

The schedule extended through August 31, 2000.

Community Involvement

Activities to involve the community in the five-year review were initiated with a meeting in early January 2000 between the RPM and the Community Involvement Coordinator (CIC) for the Acme Superfund site. A notice was sent to two local newspapers that a five-year review was to be conducted and that there would be a public meeting on April 20, 2000. A letter stating the same was sent to the Community Advisory Group (CAG), the Waters County Department of Health, the Fire and Rescue Department of Riverside, the County Commissioner's office, and the residents of properties adjacent to the Acme Superfund site. The letter invited the recipients to submit any comments to EPA.

During the public meeting, representatives of the CAG and local residents expressed concerns that work be completed as soon as possible at the site as they were concerned about the stigma that may be

attached to the property in the future, limiting its availability for redevelopment. None of the attendees expressed any concerns over the protectiveness of the remedy.

On September 11, 2000, a notice was sent to the same local newspapers that announced that the Five-Year Review report for the Acme Superfund site was complete, and that the results of the review and the report were available to the public at the Riverside Town Library and the EPA Region 1 office.

Document Review

This five-year review consisted of a review of relevant documents including O&M records and monitoring data (See Attachment 3). Applicable groundwater cleanup standards, as listed in the 1992 Record of Decision, were reviewed (See Attachment 4).

Data Review

Groundwater Monitoring

Groundwater monitoring has been conducted at the Acme Site since the late 1980s. In general, most contaminants were detected at their highest levels early in the Removal/Remedial history of the site (1989 to 1990). This high level followed by a drop in contaminant levels may well have been the result of removal activities eliminating significant source material.

The evaluation of the natural attenuation processes at the site was achieved by evaluating four indicators that are recommended in the *Use of Monitored Natural Attenuation at Superfund, RCRA Corrective Action, and Underground Storage Tank Sites* (OSWER Directive No. 9200.4-17P, April 21, 1999) for evaluating the performance of an MNA remedy. The four indicators are:

- Demonstrate that natural attenuation is occurring according to expectations;
- Detect changes in environmental conditions that may reduce the efficacy of the natural attenuation processes;
- Identify any potentially toxic or mobile transformation products; and
- Verify that the plume is not expanding either downgradient, laterally, or vertically.

Since construction completion in 1997, 8 of the 13 contaminants for which groundwater cleanup levels have been established, remained below their respective cleanup goals in all sampling events. Furthermore, for the five contaminants that have exceeded their cleanup goals in recent sampling events, there is a marked trend downward in concentrations. Recent monitoring results for the five contaminants are shown in Table 3. MW-104b, MW-104c, and MW-105b are located on the southern end of the treatment area which is the downgradient side. Therefore, trends in contaminant levels in these wells are good indicators of the fate of contaminants remaining in the groundwater near to the original source areas. In MW-104b and MW-104c, there is a clear downward trend in benzene concentrations, although concentrations remain above the cleanup goals. There is a clear indication that concentrations of TCE and the daughter products, cis 1,2-DCE and vinyl chloride are trending downward in MW-105b and MW-104c. This monitoring record indicates that the groundwater attenuation process conceptualized in the ROD is proceeding essentially as expected.

Table 3 - Quarterly Comparison of Groundwater Concentrations

Contaminant	Well No.	MCL (ppb)	Concentration in ppb				
			3/1999	6/1999	9/1999	12/1999	3/2000
Benzene	104b	5	110*	130*	310 (est)*	120*	58*
Benzene	104c	5	2,300*	4,900*	530*	190*	39*
Benzene	103c	5	100*	130*	130*	100*	NS
Trichlorethene	105b	5	15 (est)*	5.5*	ND	0.29 (est)	0.014 (est)
Vinyl chloride	105b	2	13*	5.2*	ND	ND	5.9 (est)*
cis-1,2,-Dicloroethene	104c	70	ND	78*	7.4 (est)	5.8	0.88
Lead	104c	0.015	0.005 (est)	0.004 (est)	0.017*	ND	0.003 (est)

* = Exceeds Cleanup Level
(est) = Estimated Value
ND = Not Detected
NS = Not Sampled

No monitoring of environmental conditions that may affect the efficacy of the MNA remedy is being conducted at this time. Given that contaminant concentrations continue to decline, such monitoring may not be necessary, as attenuation processes appear to be functioning as expected.

No potentially toxic or mobile transformation products have been identified during sampling events that were not already present at the time of the ROD, and therefore have cleanup goals specified in the ROD.

Regarding plume migration, there is some concern that the plume may be migrating downgradient toward the Green River. Concentrations of benzene in MW-103c have remained relatively stable since March 1999, lacking the downward trend in concentrations for this contaminant seen in other wells. This well is located downgradient from the treatment area and is closest to the river. This may be an indication that the plume is being pulled toward the river. The lack of a sampling point for the March 2000 event, due to the area of the well being flooded, gives rise to further concern. In the future, if it is not possible to obtain a sample during a scheduled monitoring event, provisions have been made to return to the site at a later date to obtain the sample and ensure that the monitoring record is complete.

Surface Water and Sediment Monitoring

Quarterly analysis of surface water samples taken in areas adjacent to the wetland and in the Green River found that all levels of contaminants of concern were below detection. Analysis of sediment samples taken in portions of the wetland and the Green River where groundwater discharges to the surface found contaminant levels also below detection limits.

Site Inspection

Inspections at the site were conducted on March 12, and May 23, 2000, by the RPM and an EPA biologist (See Attachment 5). The purpose of the inspections was to assess the protectiveness of the remedy, including the presence of fencing to restrict access, the integrity of the cap and the condition of the restored wetlands. Institutional controls were evaluated by visiting the County Planning Office to review zoning maps and by visiting the County Department of Health to review information on the site. A visit to the County Office of Public Records to review the property deed confirmed that a deed covenant had been filed.

No significant issues have been identified at any time regarding the cap, the drainage structures, or the fence. Examination of the cap revealed that there had been some slight burrowing of small animals. Another minor issue was trespassing and its effect on plantings within restored wetlands. As noted, a joint effort between the governments and the PSDs is being made to potentially change some of the wetland areas which are subject to restoration. In addition, the use of additional fencing is being considered within the site property boundaries to inhibit trespassing and better protect restored wetland plantings.

The institutional controls that are in place include prohibitions on the use or disturbance of groundwater until cleanup levels are achieved, excavation activities, disturbance of the cap, and any other activities or actions that might interfere with the implemented remedy. No activities were observed that would have violated the institutional controls. The cap and the surrounding area were undisturbed, and no new uses of groundwater were observed.

Interviews

Interviews were conducted with various parties connected to the site. Marjorie Edwards, owner of nearby Pliny Products, was interviewed on June 17, 2000. Two nearby residents, Alice Parsons and Michael Smith, were interviewed on July 18, 2000. No significant problems regarding the site were identified during the interviews. However, Mr. Smith and Ms. Parsons did note that occasional passers by have walked through the site. Paul Wainwright, a representative of the Riverside Conservation Commission, was interviewed on July 18, 2000, and expressed concern that requirements for wetland mitigation were not being observed. Mr. Wainwright was, however, confident that the problem would be resolved when a parcel of neighboring land would be selected for the establishment of new wetlands. During the May inspection, EPA interviewed the staff of the Fire and Rescue Department of Riverside, MA. None of the staff were able to identify any concerns regarding the site and there had not been any emergency responses at the site since the end of remedial construction.

VII. Technical Assessment

Question A: Is the remedy functioning as intended by the decision documents?

The review of documents, ARARs, risk assumptions, and the results of the site inspection indicates that the remedy is functioning as intended by the ROD, as modified by the ESD. The stabilization and capping of contaminated soils and sediments has achieved the remedial objectives to minimize the migration of contaminants to groundwater and surface water and prevent direct contact with, or ingestion

of, contaminants in soil and sediments. The effective implementation of institutional controls has prevented exposure to, or ingestion of, contaminated groundwater.

Operation and maintenance of the cap and drainage structures has, on the whole, been effective. A few small areas showed evidence of burrowing of small animals. The burrows did not penetrate beyond the soil layer, and so did not affect protectiveness. The PSDs were arranging for filling of the burrows and will include the task of inspection and repair of small animal burrows in future O&M routines. O&M annual costs are consistent with original estimates and there are no indications of any difficulties with the remedy.

Where the PSDs have had access to wetlands, the maintenance of the wetlands has been good. A 0.32-acre portion of the wetlands has not been maintained because the property owner where the wetlands are located has denied access to the PSDs. EPA, the Riverside Conservation Commission, and the PSDs are currently working to identify an alternate location where wetlands can be developed. The failure to meet the wetlands mitigation requirements for the site does not affect the potential for release of contaminants and does not affect protectiveness for the site.

There were no opportunities for system optimization observed during this review. The monitoring well network provides sufficient data to assess the progress of natural attenuation within the plume, and maintenance on the cap is sufficient to maintain it's integrity. There is some concern that the plume may be migrating downgradient toward the Green River. Concentrations of benzene in MW-103c have remained relatively stable since March 1999, lacking the downward trend in concentrations for this contaminant seen in other wells. This well is located downgradient from the treatment area and is closest to the river. This may be an indication that the plume is being pulled toward the river. The lack of a sampling point for the March 2000 event, due to the area of the well being flooded, gives rise to further concern.

The institutional controls that are in place include prohibitions on the use or disturbance of groundwater until cleanup levels are achieved, and prohibitions on excavation activities, disturbance of the cap, and any other activities or actions that might interfere with the implemented remedy. No activities were observed that would have violated the institutional controls. The cap and the surrounding area were undisturbed, and no new uses of groundwater were observed. The fence around the site is intact and in good repair.

Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?

There have been no changes in the physical conditions of the site that would affect the protectiveness of the remedy.

Changes in Standards and To Be Considereds

As the remedial work has been completed, most ARARs for soil contamination cited in the ROD have been met. ARARs that still must be met at this time and that have been evaluated include: the Safe Drinking Water Act (SDWA) (40 CFR 141.11-141.16) from which many of the groundwater cleanup levels were derived - [Maximum Contaminant Levels (MCLs), and MCL Goals (MCLGs)]; ARARs related

to wetland protection; and ARARs related to post-closure monitoring. A list of ARARs is included in Attachment 3. There have been no changes in these ARARs and no new standards or TBCs affecting the protectiveness of the remedy.

Changes in Exposure Pathways, Toxicity, and Other Contaminant Characteristics

The exposure assumptions used to develop the Human Health Risk Assessment included both current exposures (older child trespasser, adult trespasser) and potential future exposures (young and older future child resident, future adult resident and future adult worker). There have been no changes in the toxicity factors for the contaminants of concern that were used in the baseline risk assessment. These assumptions are considered to be conservative and reasonable in evaluating risk and developing risk-based cleanup levels. No change to these assumptions, or the cleanup levels developed from them is warranted. There has been no change to the standardized risk assessment methodology that could affect the protectiveness of the remedy. The remedy is progressing as expected and it is expected that all groundwater cleanup levels will be met within approximately 10 years.

Question C: Has any other information come to light that could call into question the protectiveness of the remedy?

No ecological targets were identified during the baseline risk assessment and none were identified during the five-year review, and therefore monitoring of ecological targets is not necessary. All sediment and surface water samples analyzed found no contamination of wetlands or surface water. No weather-related events have affected the protectiveness of the remedy. There is no other information that calls into question the protectiveness of the remedy.

Technical Assessment Summary

According to the data reviewed, the site inspection, and the interviews, the remedy is functioning as intended by the ROD, as modified by the ESD. There have been no changes in the physical conditions of the site that would affect the protectiveness of the remedy. Most ARARs for soil contamination cited in the ROD have been met. There has been no changes in the toxicity factors for the contaminants of concern that were used in the baseline risk assessment, and there have been no change to the standardized risk assessment methodology that could affect the protectiveness of the remedy. There is no other information that calls into question the protectiveness of the remedy.

VIII. Issues

Table 4 - Issues

Issue	Currently Affects Protectiveness (Y/N)	Affects Future Protectiveness (Y/N)
Evidence of small animal burrows at a few locations on the southwest corner of the cap.	N	N

Issue	Currently Affects Protectiveness (Y/N)	Affects Future Protectiveness (Y/N)
Failure to maintain 0.32 acres of the total 0.7 acres of wetlands constructed to comply with wetlands mitigation requirements for the site.	N	N
Inadequate monitoring data to verify that the plume is not migrating	N	Y

IX. Recommendations and Follow-Up Actions

Table 5 - Recommendations and Follow-Up Actions

Issue	Recommendations/ Follow-up Actions	Party Responsible	Oversight Agency	Milestone Date	Affects Protectiveness? (Y/N)	
					Current	Future
Animal burrows in cap	Repair current burrows; establish O&M task to ensure future burrows are identified and repaired	PSDs	State/EPA	6/30/2001	N	N
0.32 acres of wetlands not maintained due to access problems	Identify alternate location at or near the site for wetlands development	PSD, Riverside Conservation Commission	State/EPA	9/30/2001	N	N
Inadequate monitoring data	1) Increase monitoring frequency for MW-103 cluster; 2) Investigate groundwater recharge to river; and 3) Sample sediments and groundwater flux at recharge points.	PSDs	State/EPA	9/30/2001	N	Y

X. Protectiveness Statement

The remedy is expected to be protective of human health and the environment upon attainment of groundwater cleanup goals, through natural attenuation, which is expected to require 10 years to achieve. In the interim, exposure pathways that could result in unacceptable risks are being controlled and institutional controls are preventing exposure to, or the ingestion of, contaminated groundwater. All threats at the site have been addressed through stabilization and capping of contaminated soil and sediments, the installation of fencing and warning signs, and the implementation of institutional controls.

Long-term protectiveness of the remedial action will be verified by obtaining additional groundwater samples to fully evaluate potential migration of the contaminant plume downgradient from the treatment area and towards the river. Current data indicate that the plume remains on site. Additional sampling and analysis will be completed within the next six months. Current monitoring data indicate that the remedy is functioning as required to achieve groundwater cleanup goals.

XI. Next Review

The next five-year review for the Acme Superfund Site is required by September 2005, five years from the date of this review.

ATTACHMENTS

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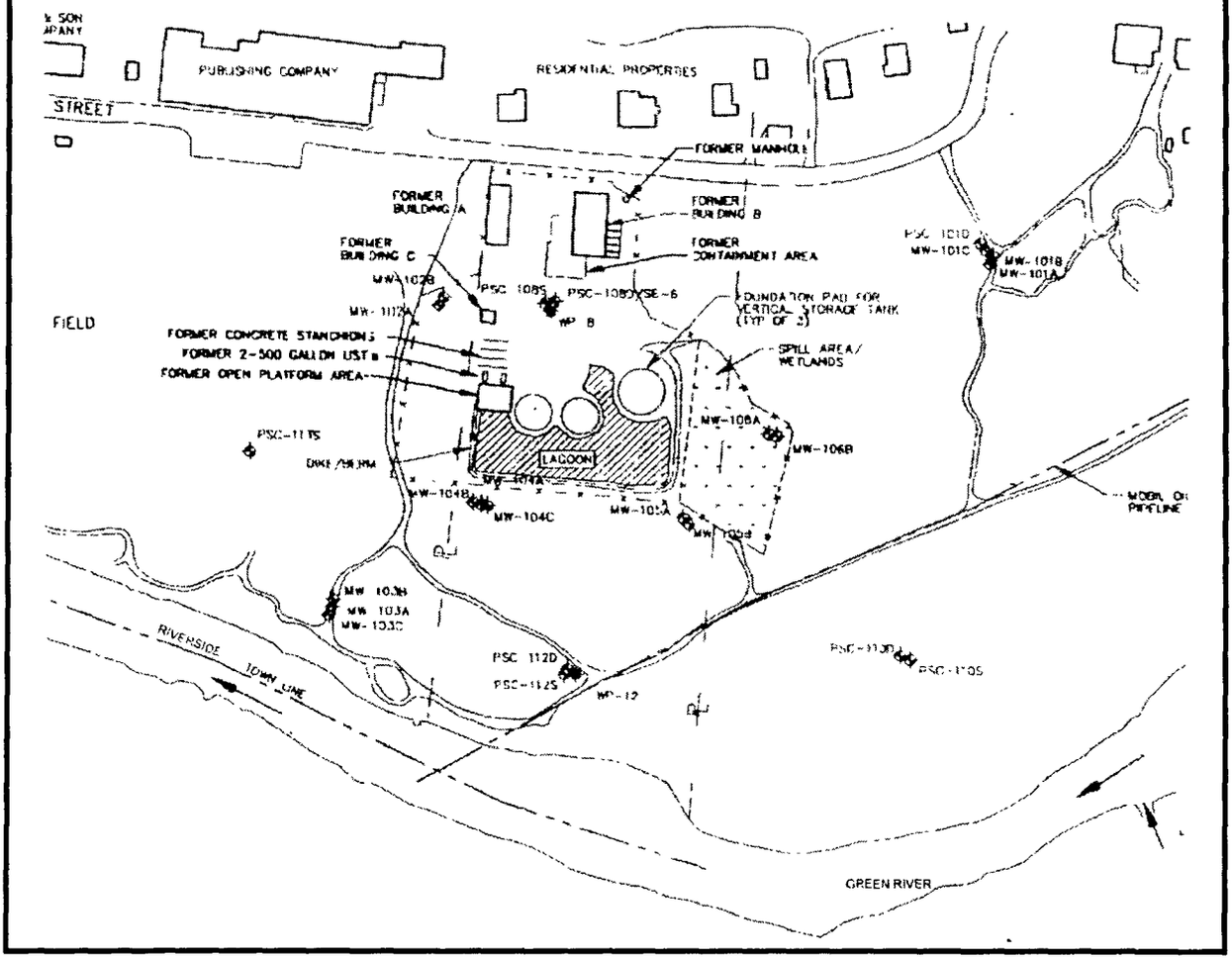
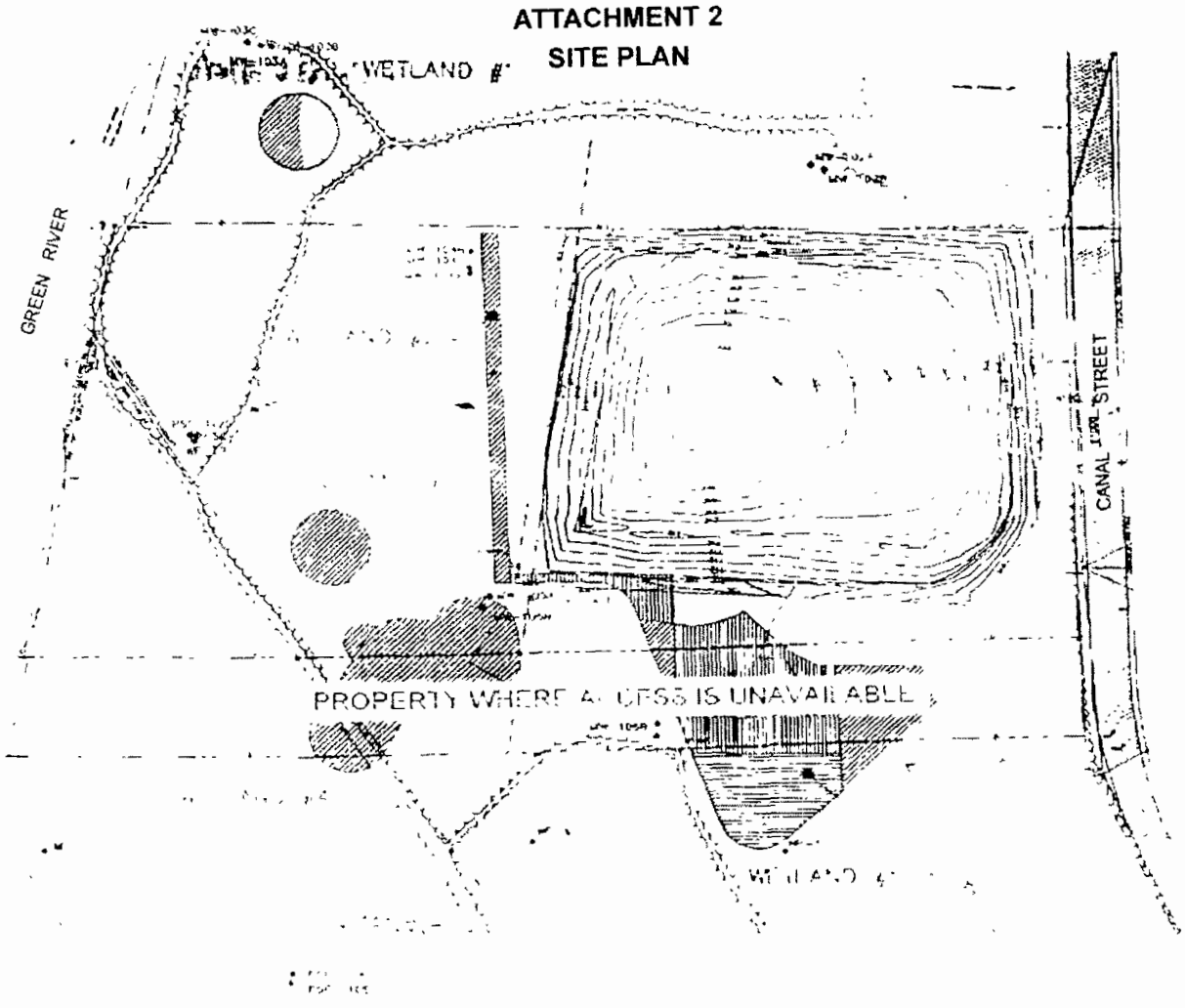
ATTACHMENT 1

SITE LOCATION MAP



ATTACHMENT 2
SITE PLAN



ACME SUPERFUND SITE
RIVERSIDE, MASSACHUSSETTS

SITE PLAN

5819005-008
NOVEMBER 1999

100 0 100
SCALE IN FEET

ATTACHMENT 3

List of Documents Reviewed

Acme Remedial Design for Stabilization and Containment of Contaminated Soils and Sediments, Riverside, MA, March 5, 1997

Acme Superfund Site Operations & Maintenance Plan, September 18, 1998

Acme Superfund Site PSDs/EPA Settlement Agreement, September 18, 1994

Acme Superfund Site Quarterly Groundwater Monitoring Reports, 1998 and 1999

Acme Superfund Site Record of Decision, September 30, 1992

Explanation of Significant Difference, Remedial Design, Acme Superfund Site, November 26, 1996

Riverside Wetlands Mitigation Plan, Riverside Conservation Commission, Riverside, MA, March 31, 1997

ATTACHMENT 4

Applicable or Relevant and Appropriate Requirements (ARARs)

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Groundwater/ SDWA	Federal - SDWA - Maximum Contaminant Levels (MCLs) (40 CFR Part 141.11-141.16) and non-zero Maximum Contaminant Level Goals (MCLGs)	Relevant and Appropriate	Standards (MCLs) have been adopted as enforceable standards for public drinking water systems: goals (MCLGs) are non-enforceable levels for such systems.	Remediation of contaminated material in soils and sediment will eliminate ongoing discharges of contaminants to groundwater. MCLs and non-zero MCLGs will be attained in groundwater at the point of compliance.

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Surface Water/CWA	Federal - CWA - Ambient Water Quality Criteria (AWQC)- Protection of Freshwater Aquatic Life, Human Health, Fish Consumption	Relevant and Appropriate	AWQC are developed under the Clean Water Act (CWA) as guidelines from which states develop water quality standards. CERCLA §121(d)(2) requires compliance with such guidelines when they are relevant and appropriate. A more stringent AWQC for aquatic life may be found relevant and appropriate rather than an MCL, when protection of aquatic organisms is being considered at a site. Federal AWQC are health-based criteria which have been developed for 95 carcinogenic compounds; these criteria consider exposure to chemicals from drinking water and/or fish consumption. Acute and chronic exposure levels are established.	The selected remedy will attain AWQC in the wetland surface waters and river water after completion of remedial activities.
Groundwater/ CWA	State Department of Environmental Protection (DEP) - Massachusetts Groundwater Quality Standards (314 CMR 6.00)	Applicable	State groundwater quality standards have been promulgated for a number of contaminants. When the state levels are more stringent than federal levels, the state levels will be used.	The selected remedy will attain State standards in the groundwater at the point of compliance after completion of remedial activities.

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Groundwater/ SDWA	State - 310 CMR 22.06 Maximum Contaminant Levels for Inorganic Chemicals in Drinking Water	Relevant and Appropriate	Maximum contaminant levels are established for inorganic chemical contaminants under 310 CMR 22.06. All public water systems must comply with the levels of inorganic contaminants which are listed in Table 1 of 310 CMR 22.06.	The selected remedy will attain State MCLs for inorganics in the groundwater at the point of compliance.
Groundwater/ SDWA	State - 310 CMR 22.07 Maximum Organic Chemical Contaminant Levels in Drinking Water	Relevant and Appropriate	310 CMR 22.07 establishes maximum contaminant levels for selected chlorinated hydrocarbons, pesticides and herbicides.	The selected remedy will attain State MCLs for organic contaminants in the groundwater at the point of compliance.
Air/CAA	Federal - CAA - National Emissions Standards for Hazardous Air Pollutants (NESHAP) (40 CFR Part 61)	Applicable	NESHAP standards have been promulgated for two organic compounds present at the site, benzene and vinyl chloride.	Remediation technologies which emit air contaminants regulated under NESHAPs will attain the appropriate standard during operation.
Soil/ Sediments/ RCRA	Federal - Resource Conservation and Recovery Act (RCRA) - Criteria for Classification of Solid Waste Disposal and Practices (40 CFR Part 257)	Relevant and Appropriate	Solid wastes containing PCBs greater than 10 ppm must not be incorporated into the soil (or mixed with surface soil) applied to land used for food chain or pasture crop production.	Any debris, soil, or sediment which contains greater than 10 ppm PCBs will be excavated and stabilized. Institutional controls will prohibit the use of the site for agriculture.

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Air/CAA	Federal - CAA - National Ambient Air Quality Standards (NAAQS) (40 CFR Part 50)	Applicable	NAAQS define levels of primary and secondary levels for six common air contaminants [sulfur dioxide, particulate matter (PM_{10}), carbon monoxide, ozone, nitrogen dioxide and lead].	The levels established for these six air contaminants will be used as target levels which may not be exceeded by air release from on-site activities.
Surface Water/CWA	State Operation and Maintenance and Pretreatment Standards for Wastewater Treatment Works and Indirect Discharge (314 CMR 12.00)	Applicable	Regulations to ensure proper operation and maintenance of wastewater treatment facilities and sewer systems within the State.	Remedial activities will comply with all provisions of this regulation.
Air/OSHA	Federal - Occupational Health and Safety Act (OSHA) (29 CFR Part 1910.1000 - Air Contaminants)	To be Considered	Acceptable employee exposure levels have been promulgated for an extensive list of materials to control air quality in workplace environments.	Action levels for volatile and semi-volatile air contaminants will be established for implementation during on-site remedial actions. Exposure levels will also be used in the risk assessment to determine overall site risk.

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Groundwater/ CWA	Federal - (Guidance) Groundwater Classification Guidelines	To be Considered	Classifies groundwater by its potential beneficial uses such as special groundwater (Class 1) which is "highly vulnerable to contamination because of the hydrological characteristics of the areas in which it occurs and characterized by either of the following factors: – The groundwater is irreplaceable; no reasonable alternative source of drinking water is available to substantial populations. – The groundwater is ecologically vital; the aquifer provides the base flow for a particularly sensitive ecological system that, if polluted, would destroy a unique habitat. Class 2 groundwater is classified as a current and potential source of drinking water and waters having other beneficial uses. All groundwater which does not fit under Class 1 and which is not heavily saline (total dissolved solids (TDS) > 10,000 mg/l) are considered Class 2 groundwater.	The groundwater aquifer will meet the standards under the SDWA for the appropriate classification of groundwater after completion of remedial activities.

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Sediments/ CWA	Federal - NOAA Technical Memorandum NOS OMA 52	To be Considered	The memorandum identifies reference doses for various contaminants in sediments and their potential biological effects on biota exposed to the contaminants.	Contaminated sediments will be remediated.
Wetlands/ CWA	Federal - CWA Section 404(b)(1); 40 CFR Part 230, 33 CFR Parts 320 - 330	Applicable	Requirements under these codes prohibit the discharge of dredged or fill material into wetlands unless those actions comply with the substantive requirements which are identified under these regulations.	Discharges to wetlands around the site will comply with these requirements.
Wetlands/ CWA	Federal Executive Orders 11990 Protection of Wetlands	Applicable	Under this regulation, Federal agencies are required to minimize the destruction, loss, or degradation of wetlands, and preserve and enhance natural and beneficial values of wetlands.	Wetlands protection considerations will be incorporated into the planning and implementation of this selected remedy.

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Floodplains/ RCRA	Federal 40 CFR Part 264.18 Location Standards	Relevant and Appropriate	This regulation identifies geological features that a proposed location for a RCRA hazardous waste treatment and/or disposal facility must avoid. Three specific geological features are identified of which two apply to the site. These features and the significance are: – Floodplain - A facility located in a 100-year floodplain must be designed, constructed, operated, and maintained to prevent washout of any hazardous waste unless the owner or operator can demonstrate to the EPA Regional Administrator that he can meet the criteria established under this subpart which exempts him from complying with this requirement.	This site is located within a 100-year floodplain and a portion of the site may be within 200 feet of a fault. On-site remediation activities will comply with the requirements of 40 CFR Parts 264.18(a) and (b).

Medium/ Authority	ARAR	Status	Requirement Synopsis	Action to be taken to Attain ARAR
Rivers/CWA	Federal - 16 USC 661 et. seq. Fish and Wildlife Coordination Act	Applicable	Mitigative actions must be taken to minimize potential adverse impacts to natural sources such as wetlands. Restoration of damaged natural features are required.	Relevant federal agencies will be contacted to help analyze impacts of the implementation of remedial alternatives on wildlife in wetlands and rivers. Restoration of impacted wetlands will occur once all excavation and stabilization activities are completed.
Wetlands/ CWA	State - Department of Environmental Protection - Wetlands Protection (310 CMR 10.00)	Applicable	These regulations are promulgated under Wetlands Protection Laws, which regulate dredging, filling, altering or polluting inland wetlands. Work within 100 feet of a wetland is regulated under this requirement. The requirement also defines wetlands based on vegetation types and requires that effects on wetlands be mitigated.	The selected remedy will include measures to mitigate and/or replace loss of habitat or hydraulic capacity in accordance with 310 CMR 10.00.

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Appendix G
Methods and Examples for Evaluating Changes in Standards and Toxicity

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Methods and Examples for Evaluating Changes in Standards and Toxicity

This appendix provides a series of flowcharts and examples that you can use to aid in evaluating changes in promulgated standards and chemical toxicity characteristics. The following tables are arranged in two sets, with a generic decision flowchart first. A hypothetical example follows with an example of the flowchart filled in according to the information in the hypothetical example.

Exhibit G-1: Evaluating Changes in Standards

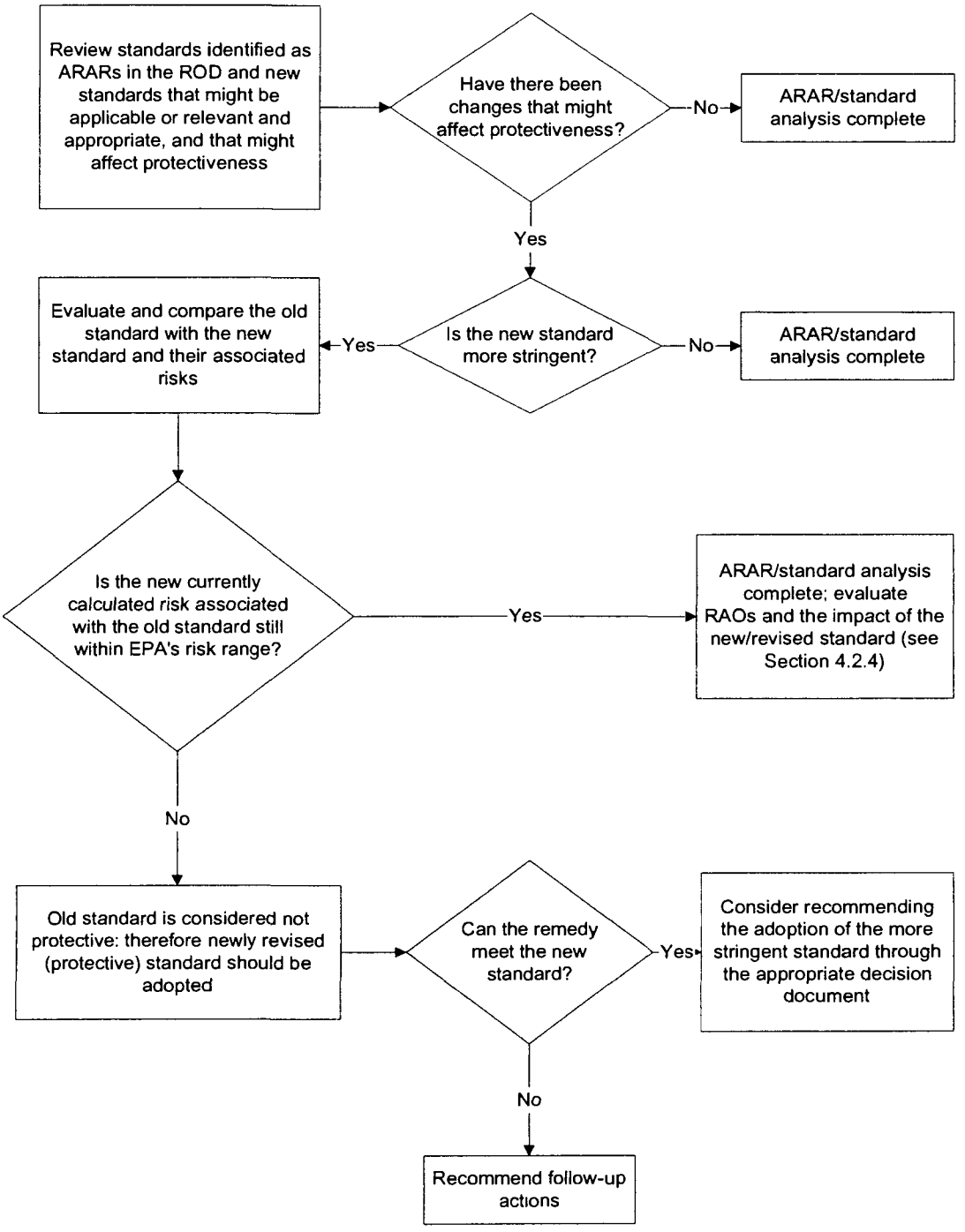


Exhibit G-2: Hypothetical Scenario for a Change in a Standard

During the 1998 Five-Year Review for the Flower Dye site in the State of Franklin, the review team learned that the State drinking water standard for 2,4-Dinitrochickenwire changed from 20 parts per billion (ppb) to 2 ppb. The Record of Decision (ROD), signed in 1988, identified the state standard for 2,4-Dinitrochickenwire as an ARAR and established a cleanup level for 2,4-Dinitrochickenwire at 20 ppb. The ROD also specified that the remedial action objective (RAO) for groundwater is to restore groundwater to drinking water standards. The remedy is to pump-and-treat groundwater using extraction and reinjection wells with air stripping.

In the ARAR/standard analysis (See Exhibit G-1) it was identified that the standard (ARAR) of 20 ppb at the time the ROD was signed had an associated risk of $5x10^{-5}$, which was within EPA's risk range. However, the current risk associated with the same level (20 ppb) now is $5x10^{-4}$ due to changes in the toxicity information that is the basis for the standard. This is generally considered outside of EPA's risk range and therefore, generally considered not protective. As part of the evaluation it was determined that the new standard (2 ppb) has an associated risk of $5x10^{-5}$, which is within EPA's risk range.

In examining the treatment records, monitoring reports, and existing groundwater modeling information, it was determined that the system can treat to 2 ppb, and potentially the remedy can achieve that level in the groundwater. Since the old standard (20 ppb) is no longer considered protective, further actions needed to be taken to ensure that the remedy achieves protectiveness. These actions included the adoption of a protective cleanup level. Therefore, the Five-Year Review report recommended that the new standard (2 ppb) be adopted through an Explanation of Significant Difference. The physical remedy did not have to be modified because it was determined that it could achieve the 2 ppb level. In addition, the RAOs would also be achieved and would not require any modification.

Exhibit G-3: Decision Process for a Hypothetical Change in Standard

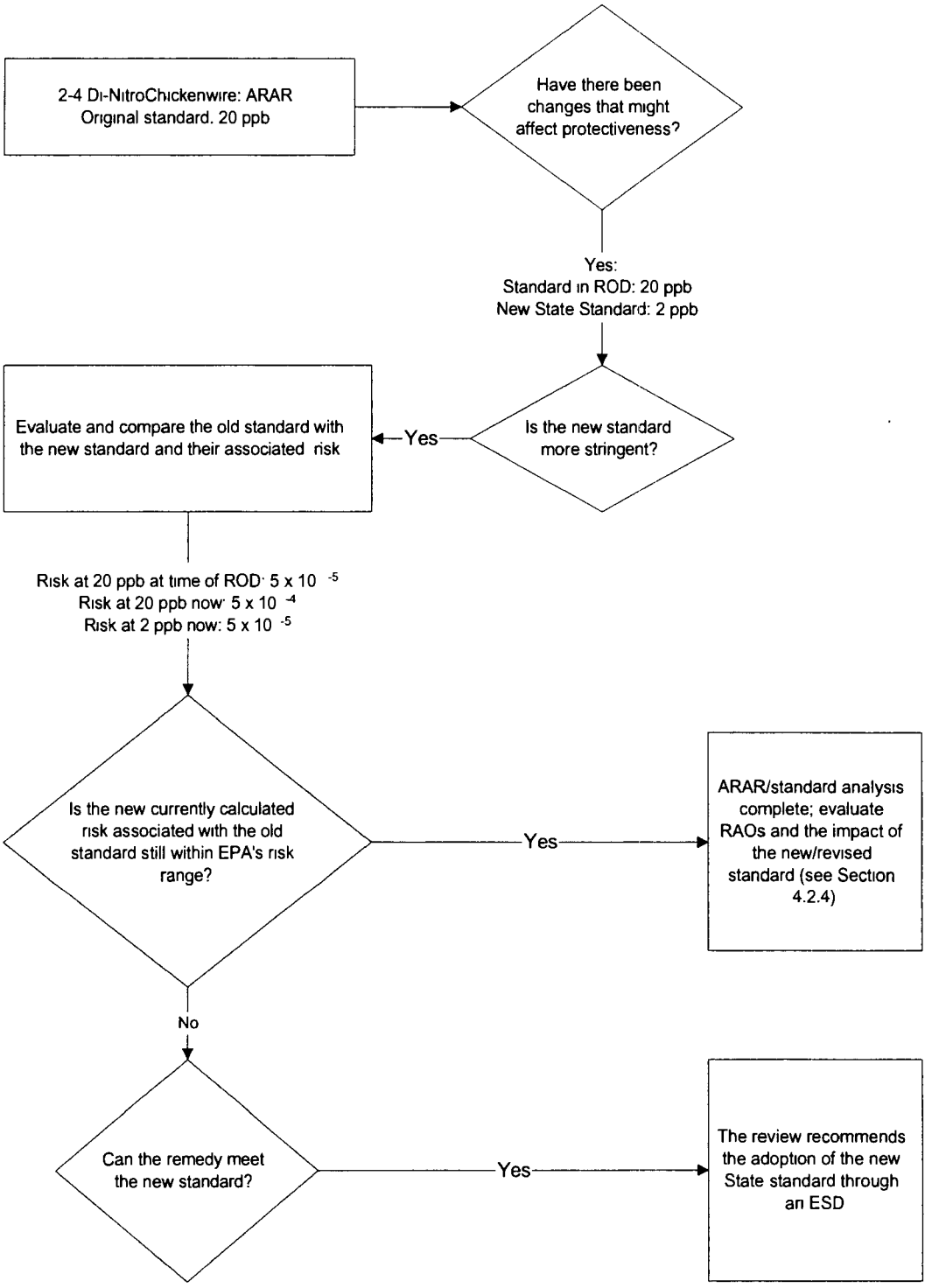


Exhibit G-4: Evaluating Changes in Toxicity and Other Contaminant Characteristics

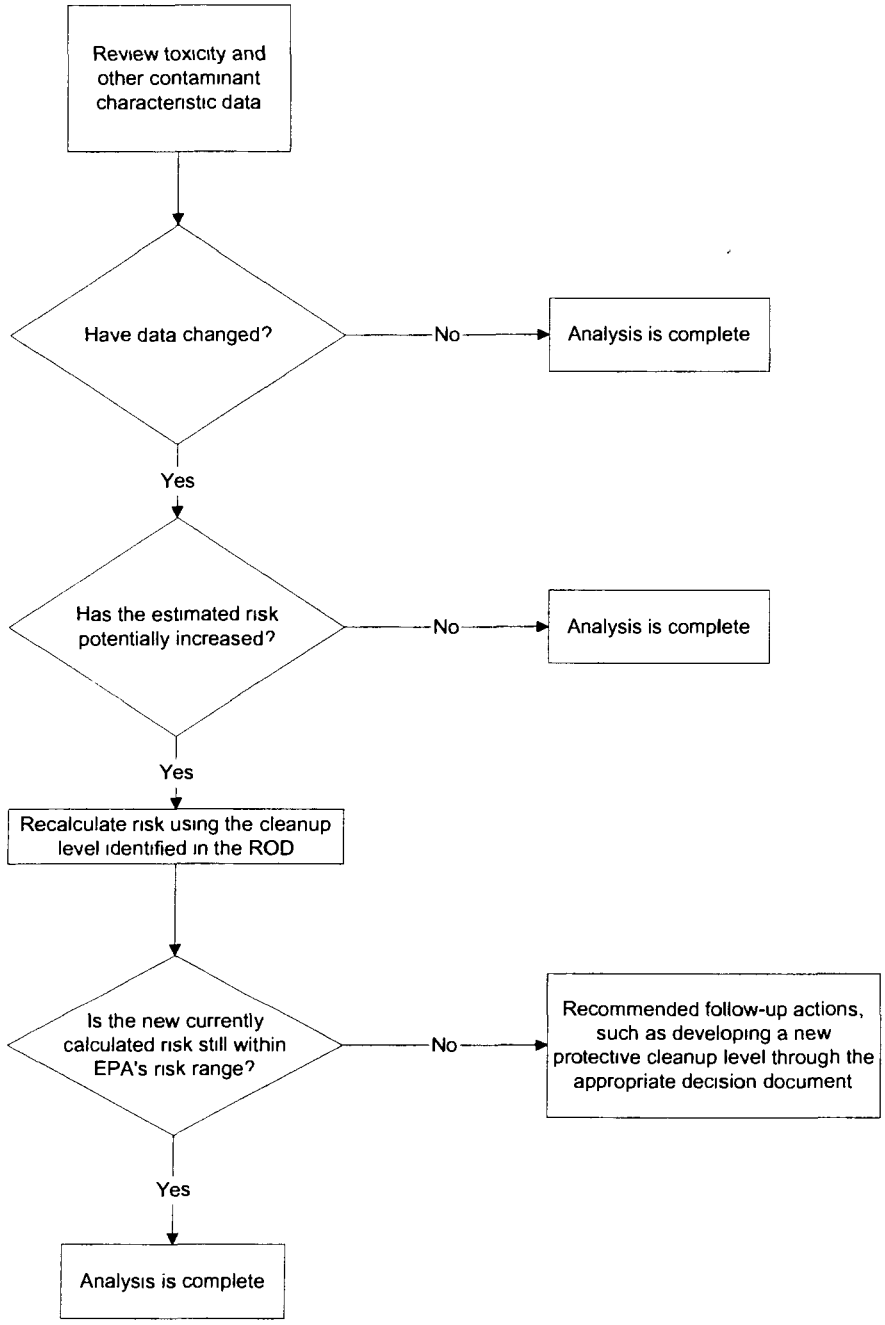


Exhibit G-5: Hypothetical Scenario for a Change in Toxicity

During the 1998 Five-Year Review at the Old Pesticide Disposal site in the State of Franklin, the review team determined that the Cancer Slope Factor (CSF) for the pesticide "Hypochem" had been increased in 1996 from 0.05 $(mg/kg\text{-}day)^{-1}$ to 20.00 $(mg/kg\text{-}day)^{-1}$. Hypochem, among other contaminants, had been found in the water supply well across the street from the Old Pesticide Disposal facility at a concentration of 0.001 mg/L. When the ROD was signed in 1986, this level was associated with a risk level less than one in one million excess cancer cases based on the following equations and site-specific exposure parameters:

$$\text{Average Daily Intake (mg/kg-day)} = (C_{Water} * IR*EF*ED)/(BW*AT) \quad (1)$$

where:

Parameter			Site Scenario
C_{Water}	=	Contaminant concentration in water (mg/L)	
IR	=	Drinking water intake (ingestion) rate (L/day)	2 L/day
EF	=	Exposure frequency (days/year)	350 days/year
ED	=	Exposure duration (years)	30 years
BW	=	Body weight (kg)	70 kg
AT	=	Average time (days)	25,550 days

$$\text{Target Risk (R)} = \text{Average Daily Intake} * \text{Cancer Slope Factor} \quad (2)$$

When equations (1) and (2) are combined, the allowable concentration of Hypochem (C_{Water}) that corresponds to a given risk level "R," can be determined by inserting the site-specific parameters into the following equation:

$$C_{water}\ (mg/L) = (R*BW*AT)\ /(CSF*IR*EF*ED) \quad (3)$$

The Old Pesticide Disposal site's original one in one million risk level R = $1x10^{-6}$) was based on the original CSF of 0.05. Thus, equation (3) yielded a health-based screening level for Hypochem of:

$$C_{Water} \text{ for R of } 1x10^{-6} = 0.001704 \text{ mg/L}$$

Since the actual concentration of Hypochem in the water in 1986 was 0.001 mg/L, and thus fell within acceptable limits, there was no need to reduce its levels. (The risk corresponded to 0.6 new cases per million people.) However, using the new CSF of 20.00 to achieve a one in one million risk level R = $1x10^{-6}$), the new health-based screening level for Hypochem becomes:

$$C_{Water} \text{ for R of } 1x10^{-6} = 0.00000426 \text{ mg/L}$$

and using the new CSF of 20.00 to achieve one in a ten thousand risk level R = $1x10^{-4}$), equation (3) yields a C_{Water} value of:

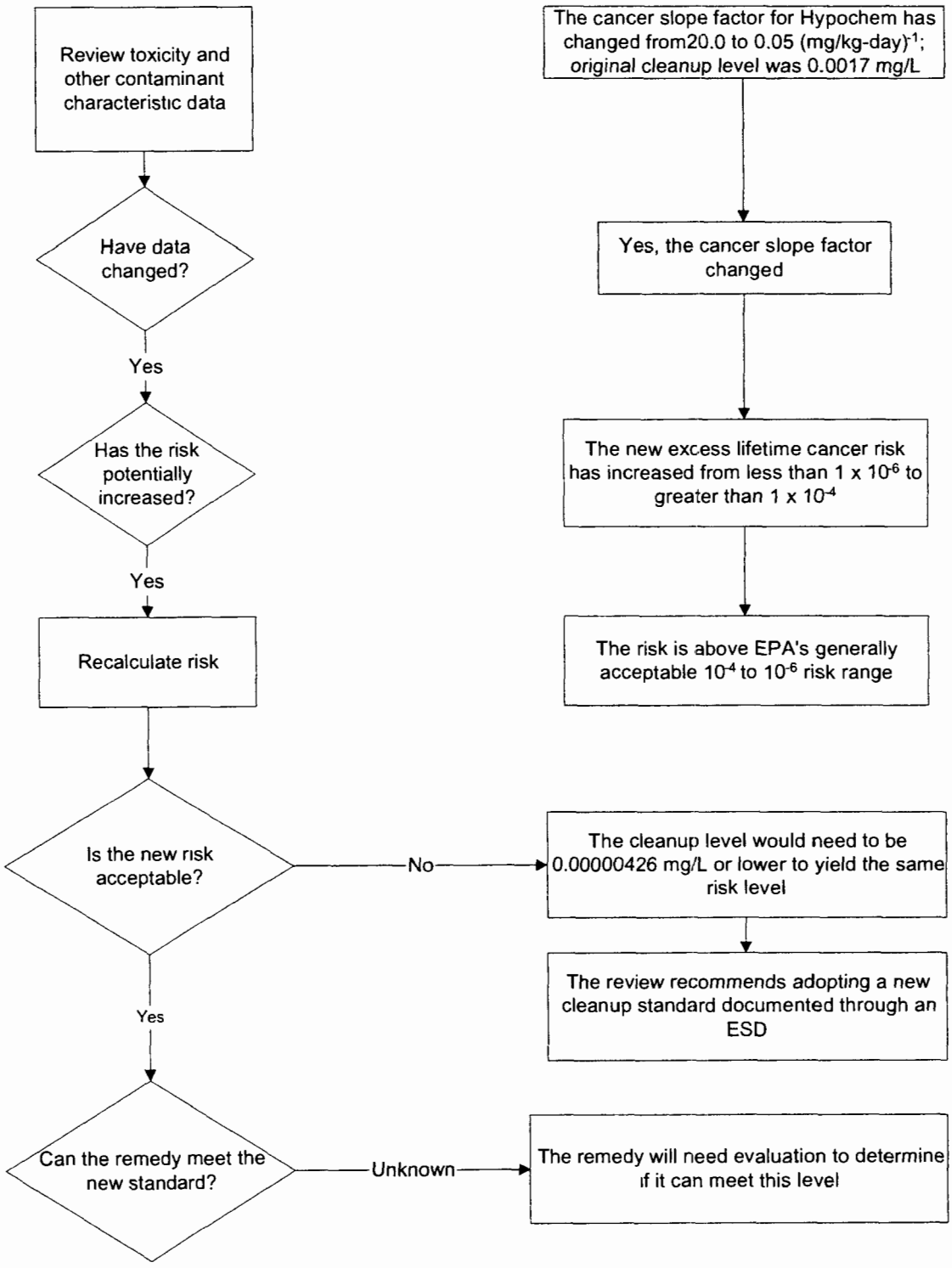
$$C_{Water} \text{ for R of } 1x10^{-4} = 0.000426 \text{ mg/L}$$

Exhibit G-5: Hypothetical Scenario for a Change in Toxicity, cont'd.

The 1986 ROD selected pumping and air stripping of the groundwater to remove solvents also found in the groundwater, and groundwater recharge. Based on sampling records of the recharge water, the stripping unit did not significantly reduce Hypochem concentrations. In fact the current concentration of Hypochem in groundwater is 0.0008 mg/L. Given the new cancer risk factor, the levels of Hypochem are not acceptable because the risk based on this new factor is greater than one in ten thousand (1×10^{-4}).

Based on this result, the Five-Year Review report recommended that a protective cleanup level be developed through the appropriate decision document. In addition, the physical remedy would have to be evaluated to determine whether the current system would be able to reduce the level of Hypochem to protective/acceptable concentrations.

Exhibit G-6: Decision Process for a Hypothetical Change in Toxicity



Attachment B

Dredging Up the Truth, Albany Times Union

TIMES UNION

Dredging up the truth

Records show GE was warned about health threats of PCBs decades before anti-dredging campaign

By Brendan J. Lyons

Updated 11:37 pm, Saturday, March 8, 2014

Photo: Paul Buckowski

Photo: Jack McKinney

A view of the former GE plant near the Hudson River on March 25, 2014 in Hudson Falls, NY. (Paul Buckowski / Times Union) and empty barrels labeled General Electric insulating oil are seen at a dump site at South Glens Falls Drag strip on July 28, 1982. (Fred McKinney/Times Union archive)

CLICK HERE FOR DOCUMENTS, GRAPHICS AND MORE COVERAGE.

Fort Edward

For years, as it fought against being forced to clean up the Hudson River, General Electric Co. argued that an oil-like insulating fluid that had seeped into the river from its Washington County capacitor plants wasn't harmful to humans.

Besides, GE officials said, the river was cleaning itself.

Yet newly uncovered documents reveal that as early as the 1960s — decades before the government ordered GE to undertake the river dredging that is scheduled to resume this spring — company officials were warned of the potential serious health threats of polychlorinated biphenyls (PCBs), which their engineers described in confidential memos as "hazardous waste."

The documents also indicate that GE flushed far more PCBs into the river than government regulators have estimated, and that nearly a million pounds a year of additional PCBs were carted away by scavenger crews, dumped with an attitude characterized by a GE engineer in 1970 as "out of sight, out of mind."

While fighting a federally mandated cleanup, the longtime CEO of the company, Jack Welch, always insisted that PCBs are safe — a position he and GE hold to this day, despite scientific evidence to the contrary.

The records were obtained by the Times Union through a series of Freedom of Information Law requests at a critical time: GE has been battling several river communities over who will pay for alternate drinking-water supplies as the corporation mops up the PCB pollution that created a nearly 200-mile environmental disaster from Hudson Falls to the Atlantic Ocean — the nation's largest Superfund site.

The federally mandated dredging of the Upper Hudson is costing GE $1 billion. Taxpayers have spent hundreds of millions more in related cleanup costs, and there are growing calls for GE to participate in funding long-stalled dredging of the Champlain Canal, which also was tainted by PCBs from GE.

The documents raise an unsettling question: Did GE brush off its own employees' assessments of the risk of PCB pollution, later engaging in a lobbying and public relations campaign to change the public's perception of the dangers?

Such questions are reflections on history, rather than current reality, GE spokesmen say.

About this article

In August 2013, the Times Union's Brendan J. Lyons, a senior writer, and J. Robert Port, a former senior editor for investigations, began a months-long examination of General Electric Co.'s history of pollution in the Hudson River. Their efforts included interviewing current and former GE employees and reviewing thousands of documents from court cases, scientific studies and public records dating back more than 40 years. A series of Freedom of Information Law requests also gave the newspaper access to hundreds of GE's internal records, which were shared through pre-trial disclosures with attorneys for several communities along the upper Hudson River that sued GE in 2009 for contaminating their water supplies.

"The fact is GE acted diligently and responsibly in dealing with PCBs, complied with the law and regulations and has done an exceptional job on the dredging project," said Mark Behan, a GE spokesman.

Misleading the regulators

The Hudson River was dying.

In the fall of 1968, a cadre of environmental specialists from the state Health Department was touring the General Electric Co.'s capacitor plants in Hudson Falls and Fort Edward to learn more about industrial pollutants that were destroying the river's ecosystem. The federal Clean Water Act was still a few years away, but the state was studying the feasibility of a regional wastewater treatment facility, after decades of allowing GE and other companies to treat the embattled river like their own toxic landfill.

But as they took their tour, the state officials were being lied to about the level of heavy metals leaking into the river from GE's plants.

"We intensionally (sic) omitted some information ... which would have greatly compounded the problem in the eyes of the regulatory people; namely, the state Department of Health authorities," a GE engineer, Kenneth H. Harvey, wrote in an internal memo dated Sept. 21, 1968, a day after the meeting.

Jack Welch, who in 1981 became GE's CEO and one of the world's most celebrated business executives, scowled as he read Harvey's memo during a deposition last year in Palm Beach, Fla.

"This guy's ass should have been fired," Welch said. "The guy that got this memo should have had this guy in and understood what the hell he was doing and why he was doing it. That's not a practice of the General Electric Co. that I knew when I worked there."

The deposition by Welch, 78, his first testimony on GE's handling of PCBs, was never made public. The 300-page transcript and a video of the proceeding were obtained by the Times Union through FOIL requests that also gave the newspaper access to hundreds of internal GE records.

Welch's testimony, and that of numerous current or former GE officials, was compelled by a lawsuit filed against GE five years ago in federal court by the Saratoga County towns of Halfmoon, Stillwater and Waterford, the village of Stillwater, and the Saratoga County Water Authority. The municipal agencies have water systems tied to the Hudson River that they claim were tainted by the ongoing dredging project and the possibly millions of pounds of PCBs that were flushed into the river from GE's capacitor plants over a 30-year period.

The records reviewed by the Times Union include internal GE reports and correspondence on the company's knowledge and handling of PCBs. Despite the unprecedented insight, thousands of other internal company records remain shielded from public disclosure, including scores of documents related to GE's multimillion-dollar public relations campaign in opposition to dredging. GE has argued in federal court that those records are privileged — that is, not subject to disclosure — including communications with Behan, a public-relations strategist and former newspaper editor.

Last week Behan said GE and three of the plaintiffs in the case — the village and town of Stillwater and the town of Waterford — had reached a "settlement in principle." He declined to elaborate.

The case records show that GE began aggressively studying the health and environmental issues related to PCBs more than 40 years ago, and curtailed use in the early 1970s of what it suspected at the time was a more dangerous type of PCBs.

"Suspected carcinogens"

GE opened its Fort Edward manufacturing plant in 1946 and its Hudson Falls plant, a mile up the river, in 1951. For decades, the plants were a backbone of employment in the hardscrabble villages. But the payoff of jobs came with long-term environmental consequences.

According to court records, GE purchased an estimated 190 million pounds of PCBs over a period of decades, using it as a dielectric fluid to insulate its capacitors from overheating. PCBs, a coal-tar byproduct, were for many years the government's preferred chemical for that purpose, in part because they were effective at preventing fires while not interfering with conductivity.

But in 1979, amid growing evidence that PCBs may cause cancer and other serious health problems, they were banned by the U.S. Environmental Protection Agency.

Much of the Hudson River's pollution occurred when PCBs were spilled onto the ground and seeped into the bedrock below the factories. They were also flushed into the capacitor plants' wastewater systems, which poured into the nearby river.

GE officials have repeatedly insisted that the company never broke any environmental laws and its toxic discharges were "permitted." In fact, there was no government permitting system for most of the period that PCBs were in use. Legal opponents of GE assert that under the federal Refuse Act, hazardous discharges to a waterway were illegal, and that the company violated state and federal permits that were required beginning in the 1970s.

No one can accurately say how many pounds of PCBs ended up in the Hudson River or the bedrock under GE's capacitor plants. A GE spokesman said the company "has not issued an estimate of the volume of PCBs that were discharged to the river."

For more than a decade, the EPA has said 1.3 million pounds of PCBs entered the river, but even the EPA cannot say where that figure originated.

"That's one we've generally been using," said David King, director of the EPA's Hudson River field office in Fort Edward. "I wouldn't say it's irrelevant but it's nothing that can be quantified with any real accuracy."

GE's internal records suggest that the EPA's estimate falls far short.

Kenneth R. Murphy, an engineer who worked in GE's environmental pollution control division in Schenectady, wrote an analysis of the potential environmental damage in June 1970. His report came as other internal records show the company was becoming increasingly aware of the environmental dangers of PCBs. Murphy warned that controlling the company's PCB waste stream would be a "major undertaking" and estimated that GE annually discharged about 500,000 pounds of PCBs "directly to bodies of water."

"The Hudson River has been the major receiving stream," he wrote in his report, which was widely distributed inside the company but never made public.

Each year, an additional 900,000 pounds of PCB waste was being taken away by contractors — "scavengers who dispose of it in an 'out of sight, out of mind' manner," Murphy wrote. "Few, if any, scavengers give consideration to proper disposal of hazardous wastes," he added.

Murphy's report estimated that another 100,000 pounds of PCB-contaminated waste was sent each year to landfills, yet "there is no indication that any consideration was given to the handling of hazardous wastes in these landfills."

In a recent interview, Murphy, now 73 and living in Virginia, didn't back away from his 1970 report. But he cautioned that he used data "more from manufacturing people, some of them with limited education and not chemists or chemical engineers."

"I just compiled data that was given to me," said Murphy, who worked at GE for 23 years. "I think the company wanted to understand the size of the problem."

By the time Murphy's report was issued, the company was aware of the potential environmental risks of PCBs. A month earlier, GE had sent a letter to its utility customers warning that PCBs, which it referred to as "Pyranol," a GE trademark, were "a matter of growing concern as to their effect on some species of wildlife."

The May 1970 letter noted that Monsanto Company, which was the only U.S. manufacturer of PCBs and sounded the alarm on the concerns, "has been reviewing procedures to be sure that these materials do not find their way into land or water environment."

There are other records outlining GE's early concerns about PCBs. In October 1969, about eight months after a California newspaper reported on a "menacing new pollutant" causing problems for marine life and birds in San Francisco Bay, a product-safety engineer at GE's Schenectady headquarters wrote a memo documenting concerns about the ecological effects and toxic hazards of PCBs. The memo, sent to a high-ranking GE engineer, cited troubling reports by Monsanto and newspaper stories noting their "hormone destroying activity" and similarities to insecticides like DDT.

"The broad class of materials we are dealing with here ... contains known or suspected carcinogens," the engineer, James S. Nelson, wrote in a six-page report.

Nelson's report said GE's "largest user" of PCBs was the Industrial and Power Capacitor Department at Hudson Falls.

In 1976, GE prepared another internal report, also never made public, that included the headline: "The Problem." The report said the industry-wide use of PCBs in 1975 was about 13 million pounds, "of which 5.6 million pounds — about 45 percent of industry usage — were used by the Department at Hudson Falls and Fort Edward ... PCBs have been of environmental concern since the late 1960s."

"Gun to your head"

Welch was at the helm when GE began battling the EPA and the state of New York in the 1970s, leading to a two-decades-long debate over the toxicity of PCBs and the effectiveness of dredging. GE vehemently opposed dredging, citing the high cost, potential ecological damage to the river, and the lack of assurances that the plan would work.

Welch holds a doctorate in chemical engineering and, after joining GE at age 24, became vice president of its chemical division in 1973 before his meteoric rise through the company's ranks. During his 20-year tenure as CEO, he was credited with turning GE into one of the world's most profitable companies. But his success was dogged by GE's pollution history, especially the Hudson River.

Welch became personally involved in the company's battle with regulators over PCBs years before he took over the company. He has recounted an incident from December of 1975, when he learned that the state Department of Environmental Conservation was holding a hearing on GE's pollution of the Hudson River, and drove to Albany from his office in Pittsfield, Mass. He said he sat incognito in the back of the hearing room, growing concerned as he watched a GE-hired biologist "coming unglued" as he appeared before regulators.

"He lacked credibility," Welch said in his deposition.

As a result, Welch took over leadership of the PCB battle. Months later, in the spring of 1976, Welch negotiated a settlement with the state of New York that released GE from state liability for the PCB pollution.

The deal called for GE to pay $3 million for monitoring the river's PCB pollution, and $1 million for research.

During his deposition last March, Welch was shown an internal company memo from April 1975.

The memo was attributed to James C. Herger, who was human resources and labor relations director at the Washington County plants in 1975, reporting to the plants' general manager, Lucas Hart. The document, carrying a header from GE's Capacitor Products Department in Hudson Falls, was stamped "strictly private."

"We are again rapidly slipping into a lethargic state of mind concerning our environmental affairs," Herger wrote. "We need to make several changes to abate the discharge of PCBs in kerosines." He also described the company's "lack of priority for environmental projects" and warned that GE's continued violation of an EPA discharge permit "could result in the shutdown of our business."

At the time Welch, a vice president, oversaw the capacitor division as part of his wide-reaching duties. He said last year, though, that he'd never seen Herger's memo and doesn't remember him.

"This fellow thinks we should have had environmental projects at the top of the priorities," Welch said. "I don't know what happened here."

At GE's request, the Herger memo, like thousands of other internal company documents, remains sealed under a court order.

Herger, 75, who left GE in 1979 and lives in Silver Spring, Md., said he met and spoke with Welch on occasion but could not recall writing the memo that's attributed to him in GE's files.

"That strikes me as a suicide note for anybody who works for that company," Herger said in a telephone interview. "Even if I had those concerns, I would not express it like that. ... Why would I have written that letter? That's the equivalent of putting a gun to your head to be an employee (of GE) and write that kind of letter."

Hiring the enemy

In 2004, four years before she became GE's vice president of corporate environmental programs, Ann R. Klee sat before a U.S. Senate environmental committee to make her pitch for confirmation as the EPA's general counsel. Klee was nominated for the position by President George W. Bush, and brought a wealth of legal talent and experience, including several years as a legislative aide on Capitol Hill.

Klee recounted "my most significant case, and certainly one of the highlights of my career" for the Senate panel. In 1990, she said, she had represented the city of Delray Beach, Fla., which had been forced to shut down its public drinking water system due to industrial pollution.

"We were able to identify the source of the contamination — a company that had been dumping used solvents on its property for years — and obtain an $8.7 million verdict ... on behalf of the city for cleanup costs and future operation and maintenance of the treatment structures," Klee told the panel, according to a copy of her statement.

In February 2008, after Klee left the EPA, GE hired her as its vice president of corporate environmental programs. Her job, in part, is overseeing management and clean-up of GE's polluted sites around the country, including the Hudson River. Klee said in a deposition two years ago that she doesn't know if her connections at the EPA were a factor in GE's decision to hire her.

At GE, Klee succeeded Stephen D. Ramsey, who was the Justice Department's environmental enforcement chief before GE hired him in 1990.

Attorneys for the river communities that sued GE have suggested in court filings that GE deliberately hired attorneys like Ramsey and Klee in part due to their government connections. They also have accused GE of plugging attorneys into key corporate positions so their work is categorized as legal advice to a client and thus won't be subject to public disclosure.

More than 20 years ago, at the height of GE's fight against dredging, the company also hired M. Peter Lanahan, a former deputy commissioner in New York's Department of Environmental Conservation. Lanahan was GE's manager of the Hudson River project and personally made presentations to his former agency on behalf of GE, records show.

"Pete was an asset to the company because ... he had great relationships throughout state government, and he was a good project manager," Ramsey said in a deposition. Welch denied that GE was looking to cash in on its executives' government connections.

"We were looking for people that had experience with the agency that they would be dealing with," Welch testified.

"No adverse health effects"

In October 2010, a year after dredging began, the project was suspended so the EPA and GE could evaluate issues that occurred that first year. A major problem was that dredging stirred up more PCBs than predicted. On at least 10 occasions the levels in the upper Hudson River, above the Troy dam, exceeded the 500 parts-per-trillion threshold set by the EPA.

As the negotiations unfolded, Klee wrote an email to the EPA and the Justice Department requesting that documents and charts used in their discussions be kept secret and not made subject to the Freedom of Information Act. The federal agencies declined the request.

"My concern was that if the documents that we exchanged in those discussions were produced in response to a FOIA request, it would be much harder for the parties to engage in an open and honest discussion," Klee testified at a September 2012 deposition in Boston.When dredging resumed, GE and the EPA had agreed to looser rules. Under the new standards, warnings were made to public water suppliers only if the 500 ppt threshold was breached at Rhinebeck — some 60 miles south of the Troy Dam.

The town of Waterford shut down its water treatment plant in 2009 when dredging started. Halfmoon, which opened a $12 million water plant along the river in 2003, kept pumping water from the river but had to turn its plant off repeatedly the first year of dredging.

In March 2010, when there was no dredging, PCB levels in the upper Hudson River spiked to 2,000 ppt, prompting Halfmoon to shut down its plant. It has not used water from the Hudson River since then.

During her 2012 deposition, Klee repeatedly said it's her "understanding" there are "no adverse health effects associated with PCBs." That view conflicts with the stance of her former employer, EPA, which considered PCBs a possible human carcinogen in the 1970s. In the 1990s, the EPA amended its position and said that PCBs are a "probable" human carcinogen.

"It's not part of my responsibility to be a PCB scientist," Klee said, when asked if she'd read reports on PCBs other than those commissioned by GE.

Welch also doesn't believe PCBs have harmful health effects, despite scientific evidence that they may. Much of his position on that, he said, comes from studies that GE commissioned as early as 1976, in which the health trends of its factory workers were studied by scientists.

Last spring, the International Agency for Research on Cancer, which first warned of PCBs' adverse health effects in 1978, declared that certain PCBs, including the type flushed into the river by GE, "have reproductive, toxic, and carcinogenic consequences." The EPA, based on past practice, is expected to adopt that finding once the World Health Organization adopts it.

"EPA," Welch scoffed, waving his hand dismissively during his deposition last year. "I was completely satisfied as to the safety of PCBs. In my time I studied it. I looked at it. I made my judgment and I was completely satisfied. ... I haven't seen any PCB studies that convince me there was another side to it."

But the studies that Welch cites have drawn scientists' questions. In one study commissioned by GE, scientists examined the health patterns of workers at the Washington County capacitor plants and determined that they had a lower rate of cancer than the general population.

"It followed people for only five years ... (and) included all the secretaries, all the people that weren't anywhere near where the PCBs were," said Dr. David O. Carpenter, who has studied PCBs for decades.

"The point about those studies is they were included in the review by the International Agency for Research on Cancer, along with all of the other studies, many of them occupational, and they were found to be unconvincing," said Carpenter, director of the Institute for Health and the Environment at the University at Albany. "The issue is that in addition to cancer, we now have such strong evidence that PCBs alter a large number of other organ systems. PCBs affect the brain and reduce learning ability. This has been demonstrated repeatedly in children exposed to PCBs, often exposed even before birth where they get PCBs from their mother's body and from breast milk."

Learning and memory functions also diminish for adults exposed to PCBs, Carpenter said. He added that some scientists suspect PCBs cause adverse effects to the thyroid glands and health risks that include diabetes, high blood pressure and effects on human reproductive systems.

"PCBs are very dangerous chemicals and anybody that says they are not dangerous simply is not telling the truth, or just does not know what studies have been done," Carpenter said.

Behan, GE's spokesman, questioned Carpenter's credibility.

"Judges in three states have found David Carpenter's opinions too unreliable for a jury to consider as evidence," Behan wrote in an email. "Given this, the Times Union is obliged to re-evaluate whether he is truly qualified to render an opinion on GE's views."

Carpenter, who was retained as an expert witness by the Saratoga County river communities suing GE, has testified in numerous other cases involving PCBs' health effects. He is the founder of the state's School of Public Health and was a member of the World Health Organization panel that last year declared PCBs are a human carcinogen. Carpenter said any compensation he receives as an expert witness is donated to the university's research program.

But not all researchers agree on the dangers of PCBs.

Paul Stewart, a PCB researcher at the State University of New York at Oswego, said the effects on the brain may not be as significant as once believed.

"I tend to see the possible effects of PCBs are far less important than I did when I started this research in 1990s and early 2000s," Stewart said. "The correlations between PCBs and cognitive development are small, even under carefully controlled conditions."

"Alter perception on PCBs"

The alarm sounded at GE in 1990 when the EPA said it was reconsidering its 1984 decision to delay dredging of the river. The federal agency noted mounting evidence that PCBs were a health concern and improvements it said were made in dredging techniques.

On Jan. 17, 1991, an internal team at GE drafted a detailed, confidential plan to take on the EPA.

The 67-page report outlined plans to mount a multimillion-dollar public relations campaign that included a strategy to "establish an intelligence network at regulatory agencies," influence media outlets, and use connections to pressure regulators and elected leaders at all levels of government.

The document, which was stamped "privileged," said the strategy was to "alter perception on PCBs and dredging" and "change regulatory treatment of PCBs."

It highlighted "positive issues" that would be a point of focus, including GE's stated position that PCBs in the Hudson River were becoming "less toxic" through "natural processes" and that "PCBs are overregulated because risk is overstated."

The "basic message," the document states, is that PCBs' "cancer potency factors" were overestimated and that they biodegrade naturally, and are therefore less toxic — notions many scientists dispute. Also, GE would take the position that "neurotoxic effects in children are not attributable to PCBs."

"What GE did was to hire scientists ... who would give GE whatever answer they wanted," said Carpenter, who was previously director of the state Health Department's Wadsworth Center for Laboratories and Research.

Carpenter said the company is correct that anaerobic bacteria remove some chlorine from the PCB molecules. But he said it doesn't destroy or reduce the PCBs. Carpenter reviewed a copy of GE's 1991 strategy report at the request of the Times Union. He pronounced it "full of lies."

"It outlines and shows an intentional strategy to deceive both the government agencies and the general public," he said. "Natural processes in the river were not creating less toxic forms of PCBs, but rather more water soluble and volatile forms that are more toxic for at least some health outcomes."

Behan challenged Carpenter's assessment and said GE's dredging of the river is serving as a national model.

"We simply disagree with his view," Behan said. "That doesn't make anything we've said misleading. We have a disagreement in principle about how he views this issue. ... GE's view is based on the research on the impact of PCBs that have been conducted on the GE workers. It's a view that's based on the interpretation of the science."

Behan also said the debate over the health effects of PCBs "is moot at this point. That ended more than a decade ago. The Hudson River dredging project is now 70 percent complete."

Still, the strategy outline raises questions about whether the company's position on the health effects of PCBs was based on business interests.

Under a section titled "Human Health Effects," the report acknowledges that even if GE could show there were inconclusive cancer studies on PCBs, there was an emerging "new area of concern" on the "neurotoxicological effects" of PCBs. The report mentioned studies showing "minor developmental deficiencies in children of mothers having PCB and other environmental contaminants."

The political targets of GE's campaign included then-Gov. Mario Cuomo, the heads of various state agencies, the EPA, the White House, members of Congress and elected officials from river towns.

On a local level, GE sought to recruit "permanent allies in most local governments above Troy," so the report listed key communities, including those in Saratoga, Washington and Warren counties. In many cases, the plan worked, and local officials initially rallied against dredging.

But whether some public officials understood the implications of their opposition to dredging is questionable.

In June 2012, Stillwater village Mayor Ernest W. Martin Sr. was questioned by a GE attorney as part of the village's lawsuit seeking $12 million for what Martin said was the cost of switching the village water system from a series of underground wells next to the river to a pipeline operated by the county water authority.

The attorney pressed Martin about letters he had written, and public statements he had made, opposing dredging and stating his belief the river was "cleaning itself."

"Well, at the time, it was out there that the river was cleaning itself and the fish and the plant life was all coming back," he testified. "And I was afraid if they stirred up the PCBs, it would just disrupt everything that's going on by cleaning itself."

Martin couldn't cite any report, scientific study or other written materials that led him to oppose dredging. "Common sense," he finally said.

The federal government disagreed.

"Looks can be deceiving," states an EPA web page on the Hudson River's PCB pollution, last updated in 2010. "Yes, the Hudson River looks clean and is teeming with fish. But, the fish and the river bottom on which they depend for food and shelter are contaminated by PCBs. ... The river is not cleaning itself. The PCBs are not safely buried in the sediment. They continue to move as the river flows and each day add to the pollution of the river."

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http://www.timesunion.com/local/article/Dredging-up-the-truth-5294643.php photo-5984598

Attachment C

ATSDR Case Studies in Environmental Medicine Polychlorinated Biphenyls (PCBs) Toxicity

ATSDR Case Studies in Environmental Medicine Polychlorinated Biphenyls (PCBs) Toxicity

U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

226072-A

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

CASE STUDIES IN ENVIRONMENTAL MEDICINE

Polychlorinated Biphenyls (PCBs) Toxicity

Course: WB 2460

Original Date: May 14, 2014

CE Renewal Date: May 14, 2016

Expiration Date: May 14, 2018

Key Concepts	• The highest human exposures to polychlorinated biphenyls (PCBs) occur via the consumption of contaminated fish and, in certain occupational settings, via contact with equipment or materials made before 1977. • Recent studies indicate that maternal consumption of PCB-contaminated fish can cause disturbances in reproductive parameters and neurobehavioral and developmental deficits in newborns and older children. • Evidence shows that exposures to high concentrations of PCBs cause adverse dermal effects in humans. On the basis of sufficient evidence of carcinogenicity in humans and experimental animals, the International Agency for Research on Cancer (IARC) classified PCBs as carcinogenic to humans (Group 1).
About This and Other Case Studies in Environmental Medicine	This educational case study document is one in a series of self-instructional modules designed to increase the primary care provider knowledge of hazardous substances in the environment and to promote the adoption of medical practices that aid in the evaluation and care of potentially exposed patients. The complete series of Case Studies in Environmental Medicine is located on the ATSDR Web site at URL: http: www.atsdr.cdc.gov csem csem.html In addition, the downloadable PDF version of this

	educational series and other environmental medicine materials provides content in an electronic, printable format, especially for those who may lack adequate Internet service.
Acknowledgements	**We gratefully acknowledge the work of the medical writers, editors, and reviewers in producing this educational resource. Contributors to this version of the Case Study in Environmental Medicine are listed below.** **Please Note: Each content expert for this case study has indicated that there is no conflict of interest that would bias the case study content.** **CDC ATSDR Author(s):** Dianyi Yu, MD **CDC ATSDR Planners:** Charlton Coles, PhD; John Doyle, MPA; Kimberly Gehle, MD; Sharon L. Hall, PhD; Michael Hatcher, DrPH; Barbara M. Riley, RN; Delene Roberts, MSA; Brian Tencza, MS; Dianyi Yu, MD. **Peer Reviewers**: Obaid Faroon, DVM, PhD; John Osterich, PhD; Avima M. Ruder, Ph.D.; Anne Sowell, PhD; Mohammed Uddin, MD, MPH.
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units (CEU) for CPH recertification credits from CDC. Please select CEU as your choice for continuing education when registering for a course on TCEOnline. Learners seeking CPH should use the guidelines provided by the NBPHE for calculating recertification credits. For assistance please contact NBPHE at http: www.NBPHE.org.

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U.S. Department of Health and Human Services

Agency for Toxic Substances and Disease Registry

Division of Toxicology and Human Health Sciences

Environmental Medicine Branch

Table of Contents

How to Use This Course

Introduction	The goal of ATSDR□S CSEM is to increase the primary health care provider□S knowledge of hazardous substances in the environment and to help evaluate and treat potentially exposed patients. This CSEM focuses on PCB toxicity.

Availability	Two versions of the PCB toxicity CSEM are available. • The HTML versionhttp: atsdr-link.cdc.gov csem csem.asp?csem 30 po 0 provides content through the Internet. This version offers interactive exercises and prescriptive feedback to the user. • The downloadable PDF version http: www.atsdr.cdc.gov csem pcb docs pcb.pdf provides content in an electronic, printable format.
Instructions	To make the most effective use of this course: • Take the Initial Check to assess your current knowledge about PCB toxicity. • Read the title, learning objectives, text, and key points in each section. • Complete the progress check exercises at the end of each section and check your answers. • Complete and submit your assessment and posttest response online if you wish to obtain free continuing education credit. You can print your continuing education certificates immediately upon completion.
Instructional Format	This course is designed to help you learn efficiently. Topics are clearly labeled so that you can skip or quickly scan sections with which you are already familiar. This labeling will also allow you to use this training material as a handy reference. To help you identify and absorb important content quickly, each section is structured in the following format.

Section Element	**Purpose**
Title	Serves as a focus question that you should be able to answer after completing the section
Learning Objectives	Describes specific content addressed in each section and focuses your attention on important points
Text	Provides the information you need to answer the focus questions and achieve the learning objectives

Key Points	Highlights important issues and helps you review
Progress Check Exercises	Enables you to test yourself to determine whether you have mastered the learning objectives
Progress Check Answers	Provides feedback to ensure you understand the content and can locate information in the text

Learning Objectives	Upon completion of the PCBs toxicity CSEM, you will be able to meet the objectives as outlined.

Content Area	**Objectives**
Overview	• Describe key characteristics of PCBs
Exposure Pathways	• Identify sources of exposure to PCBs • Identify routes of exposure to PCBs
Who is at Risk	• Identify who is at risk for exposure to PCBs
Standards and Regulations	• Identify EPA's maximum contaminant level (MCL) for PCBs in drinking water • Identify FDA's tolerance levels for PCBs in food
Biological Fate	• Describe characteristics of the metabolism of PCBs in the body
Health Effects	• Describe adverse health effects associated with exposure to PCBs
Clinical Assessment	• Describe characteristic findings on clinical assessment of patients exposed to PCBs • Describe a rational approach for evaluating a patient with a history of occupational and or environmental exposure to PCBs • Describe measurements that can help diagnose exposure to PCBs
Treatment and Management	• Describe the principal treatment strategy for managing PCB poisoning

	• Describe the measures for preventing occupational and environmental exposure to PCBs
Patient Education and Counseling	• Describe instructions appropriate for patients exposed to PCBs

Initial Check

Instructions	This Initial Check will help you assess your current knowledge about PCB toxicity. To take the Initial Check, read the case below and then answer the questions that follow.
Case	**A 48-year-old handyman has progressive cystic acne and abnormal liver function.** A 48-year-old man that you are treating for acne vulgaris returns to your clinic for a follow-up appointment. You first saw this patient about 3 weeks ago. At that time, he had multiple acneiform lesions in the malar and periorbital areas. Both cystic and comedonal lesions were present; most ranged between 3 and 6 millimeters (mm) in diameter, and some were edematous. The patient noted that he was surprised about the development of acne at his age; he never suffered from this condition during adolescence. He had used over-the-counter astringents and anti-acne medications, but they had not affected the lesions. A review of the patient□s medical history indicates that he has • **No** history of hepatitis, • **No** recollection of contact with hepatitis patients, • **No** evidence of liver difficulties, and • **No** record of blood transfusion. The patient has no family history of cardiovascular disease or cancer. The patient does not smoke; he drinks two to three bottles of beer each evening and sometimes more on weekends. He is taking no

medications other than over-the-counter dermatologic medications.

The patient is married and has three teenaged children. His wife and children are in good health. They live in a high-rise apartment building where the patient has been a handyman and part-time building manager for the past year. He spends a lot of time in the basement area, which includes

- Heating facilities,
- A laundry,
- A recreation room with pool table, and
- A workshop.

The patient is an avid fisherman who spends most weekends fishing and eating his catch with his two sons.

At the end of the patient's initial visit, you prescribed a topical antibiotic and instructed the patient on its use. After reassuring the patient that stronger prescription medications are available to treat acne, you ordered a serum biochemical and hematologic profile to document baseline values.

During today's physical examination, you note little or no improvement in the patient's acne. The ratio of cystic to comedonal lesions appears to have increased, and many lesions appear to have become more edematous and erythematous. The patient has several new comedones on his chin, and he points out what appears to be developing areas of folliculitis on his chest and forearms.

In addition to this worsening of the patient's acne, your physical examination of the patient reveals mild, non-tender hepatomegaly without jaundice. This finding causes you to review the results of the biochemical panel. You note the following abnormalities:

- Total bilirubin 2.8 milligrams per deciliter (mg dL) (normal 0 1.5),
- Direct bilirubin 0.4 mg dL (normal 0 0.4),

	• Serum glutamic-pyruvic transaminase (SGPT) (alanine aminotransferase [ALT]) 74 international units per liter (IU L) (normal 0□50), • Serum glutamic-oxaloacetic transaminase (SGOT) (aspartate aminotransferase [AST]) 88 IU L (normal 0□50), • Glutamyl transpeptidase (GGTP or GGT) 190 IU L (normal 15□85), and • Lactate dehydrogenase (LDH) 230 IU L (normal 50□225). The results of all other tests, including the complete blood count, alkaline phosphatase (ALP), blood urea nitrogen, creatinine, and urinalysis, are within normal limits.

Initial Check Questions

1. What should be included in the patient's problem list?

2. What is a differential diagnosis for the patient's altered liver enzymes?

3. What tests would help you arrive at a diagnosis?

4. You persistently pose detailed questions to the patient regarding his work, hobbies, recreational activities, and possible contact with hepatotoxins.

   The patient reveals that he frequently wipes up a "dark, oily discharge" near a large electrical transformer in the work area in the basement workshop. The discharge has produced a gummy residue on tools and other surfaces. He mentions he sometimes feels dizzy and nauseated after working in the basement all day.

   Is this additional information related to the clinical findings?

5. Is there a need to be concerned about exposure to PCBs when the clinical effects in this patient seem so limited?

6. Are other sources of PCB exposure likely for this patient?

7. What confirming laboratory test can be conducted to establish the diagnosis of PCB exposure?

8. The doctor requests a serum PCB analysis. The laboratory reports a level of 125 parts per billion (ppb), with no normal range indicated. How will you interpret this report?

9. What steps should be recommended to patients when PCB-related health effects are suspected?

10. What additional steps should be taken for the situation described in the case study?

Initial Check Answers

1. The patient's problem list includes acne vulgaris, which is atypical because of the location of the lesions and their late onset with no history of outbreaks during adolescence. The mildly altered liver functions are nonspecific but clinically unexpected.

   More information for this answer can be found in the "Clinical Assessment – Signs and Symptoms" section.

2. The combination of asymptomatic hepatomegaly and mild nonspecific elevations of hepatic enzymes suggests a chronic inflammatory liver process or hepatitis. Hepatitis can be drug-induced, toxic, infectious, genetic, or caused by connective tissue disease.

   The major cause of liver disease in the United States is ethanol ingestion. Less commonly, environmental exposures cause either acute or chronic toxic hepatitis. Some connective tissue diseases such as lupus erythematosus are associated with a specific type of hepatitis. Infectious hepatitis includes those attributed to the viruses such as A, B, C, and other possible agents of non-A, non-B hepatitis. Hepatitis can also occur with Epstein-Barr virus and cytomegalovirus infections. Infiltrative diseases such as sarcoidosis or amyloidosis, and rare genetic diseases such as Wilson disease, primary hemochromatosis, and alpha-1-antitrypsin deficiency should be excluded as causes of hepatitis also.

   More information for this answer can be found in the "Clinical Assessment – Laboratory Tests" section.

3. Repeat ALT, AST, GGT, and bilirubin testing; test ALP and prothrombin time; and test for hepatitis viral serologies, heterophil antibody (anti-EBV capsid IgM), anti-nuclear antibody, anti-smooth muscle antibody, and anti-mitochondrial antibody. Consider hemachromatosis (serum ferritin, iron,

and iron binding capacity), Wilson disease (serum copper and ceruloplasmin), and parasitic hepatitis as possible causes of chronic hepatitis.

Assays for suspected hepatotoxins and biopsy of adipose tissue might also be of value. Further evaluation might include ultrasound and percutaneous liver biopsy if other tests do not provide sufficient information.

More information for this answer can be found in the "Clinical Assessment – Laboratory Tests" section.

4. Older electrical transformers and capacitors can contain PCBs as a dielectric and heat-transfer fluid. Leaks in this equipment could allow PCBs to volatilize under conditions of increased temperature. A person with chronic exposure to the vapors or residue could eventually receive a significant PCB dose through both dermal and inhalation routes.

   More information for this answer can be found in the "How Are People Exposed to PCBs?" section.

5. Notably, potential carcinogenicity is the main reason PCB production was banned in the United States. EPA has determined that PCBs are probable human carcinogens and has assigned them the cancer weight-of-evidence classification B2. DHHS concluded that PCBs are reasonably anticipated to be carcinogenic in humans, based on sufficient evidence of carcinogenicity in animals. In February 2013, 26 experts from 12 countries met at the International Agency for Research on Cancer (IARC), Lyon, France, to reassess the carcinogenicity of polychlorinated biphenyls (PCBs). On the basis of sufficient evidence of carcinogenicity in humans and experimental animals, the IARC classified PCBs as carcinogenic to humans (Group 1). The classification is based on

consistent association between PCB exposure and increased risk of melanoma in humans.

More information for this answer can be found in the "What Are the Physiologic Effects of PCBs?" section.

6. In addition to possible dermal and inhalation exposure, the patient might be exposed by consuming contaminated fish, a potential source of PCBs.

   More information for this answer can be found in the "How Are People Exposed to PCBs?" section.

7. Select laboratories have the capability to perform PCB analyses on human tissue. The lipophilic nature of PCBs causes them to accumulate in fat; consequently, analysis of adipose tissue obtained by biopsy has been advocated as a measure of long-term exposure. Serum PCB analysis, which is less invasive than fat biopsy, is readily available. Health risks are not consistent necessarily with PCB levels, but a serum measurement is useful for gauging the patient□s exposure.

   More information for this answer can be found in the "Clinical Assessment – Laboratory Tests" section.

8. A correlation between increasing levels of serum PCBs and dermatologic findings, including chloracne, has not been found consistently in human epidemiologic studies. However, statistically significant associations between dermatologic effects and plasma levels of higher chlorinated PCB congeners have been reported.

   PCB compounds generally can be found at the parts per trillion (ppt) levels in the lipid stores of humans, especially persons living in an industrialized society. The general population is exposed to PCB compounds primarily through the ingestion of high-fat foods such as dairy products,

eggs, animal fats, and some fish and wildlife [CDC 2009b; Hopf et al. 2009; Patterson et al. 2008]. By comparison, the case study patient's PCB serum level of 125 ppb is consistent with PCB exposure as a cause for his unusual acne, and PCB exposure might be contributing to the hepatic effects noted.

More information for this answer can be found in the "Clinical Assessment – Laboratory Tests" section.

9. The first objective should be to stop the exposure. In this case, the patient should stay away from the basement until the transformer is repaired and the basement area is cleaned by a professional familiar with PCB removal. He should also check with his state advisory on PCB-fish contamination and not eat fish from contaminated areas until his PCB level normalizes and the fish are declared uncontaminated. Many states issue advisories on fish consumption based on where the fish are caught. Fish advisories also provide guidance on how to choose fish that are safer to eat and on safer ways to prepare and cook fish. Avoiding exposure is especially important because no specific treatment exists for PCB accumulation. The need to avoid other hepatotoxic substances, including ethanol, should be stressed. Confirmation of exposure with a serum PCB level should be obtained.

   More information for this answer can be found in the "How Should Patients Exposed to PCBs Be Treated and Managed?" section.

10. Because stopping exposure is of prime importance, the physician can be most helpful by advising the patient that proper abatement by professionals is necessary. In this case, the owner of the building should be notified of the potential health hazard and should contact the local public health agency. This might require the assistance of local, state, or federal agencies such as the department of public health and EPA. These agencies can cooperate with

entities involved to ensure remediation of the harmful exposure. It is important to prevent other persons from using the basement areas until cleanup is complete. In addition, the patient should be informed of the availability of fishing and game advisories particular to his state, and he should be encouraged to observe the recommendations of these advisories.

More information for this answer can be found in the "How Should Patients Exposed to PCBs Be Treated and Managed?" section.

What Are Polychlorinated Biphenyls (PCBs)?

Learning Objective

Upon completion of this section, you will be able to

- Describe key characteristics of PCBs.

Definition

PCBs are chemicals formed by attaching one or more chlorine atoms (at the Xs in Figure 1 below) to a pair of connected benzene rings.

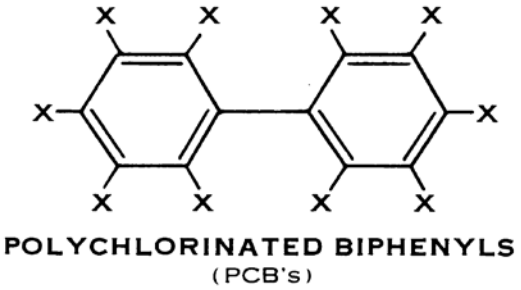

Figure 1: Polychlorinated Biphenyls (PCBs)

Depending on the number and position of chlorine atoms attached to the biphenyl ring structure, 209 different PCB congeners can be formed. PCB congeners can be divided into the coplanar, the mono-ortho-substituted PCBs, and other non-dioxin-like PCBs. The significance of this designation is that coplanar and some of the mono-ortho-substituted PCBs have dioxin-like toxicologic effects.

Chemical Properties: Dioxin-like vs. Non-dioxin-like	The chlorination pattern of the PCBs determines the toxicity of the substance. A number of PCB congeners show **dioxin-like** toxicity. These PCBs have no more than one chlorine atom at the ortho-position (polychlorinated non-ortho and mono-ortho biphenyls). The phenyl rings of these molecules can rotate and adopt a coplanar structure, which leads to the same toxicity as the polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The toxic effects of these dioxin-like PCBs are discussed in detail in later sections of this document. A number of PCB congeners, however, have two or more of the ortho-positions in the biphenyl molecules occupied by chlorine molecules. For these, the two phenyl rings are not in the same plane, and these PCBs express **non-dioxin-like** toxicity. Commercial PCB products are mixtures of different PCB congeners and contain small amounts of PCDFs or PCDDs. Contamination is a concern because the toxicity of these contaminants is generally much greater than that of PCBs [ATSDR 2000].
Uses	Because of their insulating and nonflammable properties, PCBs were marketed for nearly 50 years between 1929 and 1977. They were used in making • Diffusion pump oils, • Extenders for pesticides, • Heat exchange and dielectric fluids in transformers and capacitors, • Hydraulic and lubricating fluids, and • As ingredients in caulking compounds, paints, adhesives, flame retardants, and plasticizers. In 1977, the United States banned production of PCBs because of their potential carcinogenicity.
Trade Names	The following trade names are used for commercial PCB mixtures: • Aroclor

	• Asbestol • Askarel • Clorphen • Diaclor • Dykanol • EEC-18 • Eucarel • Nepolin • Pyranol
Key Points	• PCBs are manufactured chemicals that were produced for nearly 50 years in the United States before they were banned in 1977. • PCBs were banned because of their potential carcinogenicity.
Progress Check	1. Select the best answer from the following choices: A. Commercial PCB products are mixtures of different PCB congeners. B. Commercial PCB products commonly are contaminated with small amounts of polychlorinated dibenzofurans (furans) or polychlorinated dibenzodioxins (dioxins). C. PCB manufacturing was banned in the United States in 1977. D. All of the above. *To review relevant content, see "Chemical Properties and Uses" in this section.*

Where Are PCBs Found?

Learning Objective	Upon completion of this section, you will be able to • Identify sources of exposure to PCBs.

Introduction	No known natural sources of PCBs exist. The United States banned production of these chemicals in 1977, when their ability to accumulate in the environment and to cause harmful effects became apparent [ATSDR 2000]. Today, the major source of exposure to ambient PCBs is environmental cycling of PCBs previously released into the environment.
Released Into the Environment	Between 1929 and 1977, more than 1.25 billion pounds of PCBs were produced in the United States [ATSDR 2000]. PCBs can be released into the general environment by or from • Disposal of PCB-containing consumer products in municipal landfills • Illegal or improper dumping of waste that contained PCBs, such as transformer fluids • Leaks (fugitive emissions) from electrical transformers and capacitors containing PCBs • Poorly maintained toxic waste sites Once released into the environment, PCBs • bioaccumulate and biomagnify as they move up the food chain, • degrade relatively slowly, and • are cycled and transported within the ecosystem [ATSDR 2000; Safe 2007]. PCBs have been identified in at least 500 of the 1,598 hazardous waste sites on the EPA□S National Priorities List, and low levels of PCBs can be found throughout the world [ATSDR 2000].

Environmental Contamination	Once released into the environment, PCBs adsorb strongly to soil and sediment. As a result, these compounds tend to persist in the environment, with half-lives for most congeners ranging from months to years. PCBs leach from soil slowly, particularly the more highly chlorinated congeners, and translocate to plants via soil insignificantly. Cycling of PCBs through the environment involves volatilization from land and water surfaces into the atmosphere, with subsequent removal from the atmosphere by wet or dry deposition, then revolatilization. In the general population, inhalation of these airborne PCBs is one route of exposure, in addition to the food source of exposure to PCBs.
Key Points	• Environmental contamination from PCBs has been caused by accidental releases, careless disposal practices, and leaks from industrial facilities or chemical waste-water disposal sites. • PCBs degrade very slowly, are cycled and transported within the ecosystem, and bioaccumulate as they move up the food chain.
Progress Check	2. Once released into the environment, PCBs may undergo all of the following **EXCEPT** A. Volatilization from land and water surfaces into the atmosphere B. Biotransformation into more complex undefined mixtures C. Fast degradation within the ecosystem D. Strong adsorption to soil and sediment *To review relevant content, see "Released into Environment" in this section.*

What Are Routes of Exposure for PCBs?

Learning Objective	Upon completion of this section, you will be able to

	• Identify routes of exposure to PCBs.
Introduction	Although PCBs are no longer manufactured in the United States, people can still be exposed to them. The two main sources of exposure to PCBs are the environment and the workplace. Because they are resistant to degradation, highly chlorinated PCB compounds can persist in the environment for decades. However, over the past two decades, concentrations of PCBs in most environmental media generally have decreased.
Non-Occupational Exposure: • **General Population**	Food is the main source of exposure to PCBs for the general population (CDC 2009b). Exposure occurs primarily by ingesting high-fat foods□such as dairy products, eggs, and animal fats□and some fish and wildlife [ATSDR 2000; CDC 2009b; Fisher 1999; Gunderson and Gunderson 1988; Hopf et al. 2009; Patterson et al. 2008]. CDC publishes the National Report on Human Exposure to Environmental Chemicals. This report is an ongoing assessment of the exposure to environmental chemicals in the general U.S. population. The Fourth Report (CDC 2009b) contains data for years 1999□2000, 2001□2002, and 2003□2004 from participants in National Health and Nutrition Examination Survey (NHANES). Detailed information on NHANES is available at http: www.cdc.gov nchs nhanes about□nhanes.htm . Serum concentrations of PCBs were found to reflect cumulative past exposure in the general U.S. population [ATSDR 2011].

• **Bio-accumula-tion and Dietary Exposure**	In aquatic environments, the high lipophilicity of PCBs causes these compounds to partition out of the water and become adsorbed preferentially to sediments. Although sediment adsorption helps prevent the contamination of drinking-water supplies, the partitioning of PCBs to sediments plays a role in the tendency of these compounds to become concentrated in aquatic organisms. Bottom-feeding fish ingest and accumulate PCBs from sediment. The resistance of these compounds to biodegradation causes PCBs to become more concentrated as they move upward through the food chain from the bottom-feeding organisms. As a result of this bioconcentration and biomagnification, PCB levels in aquatic organisms can be as much as one million times higher than the levels in the aquatic environment [ATSDR 2000]. In the National Study of Chemical Residues in Fish conducted between 1986 and 1989 [EPA 1992a, 1992b], the mean concentration of PCBs in bottom-feeding and game fish was 1.9 parts per million (ppm). However, PCB levels as high as 20 ppm have been detected in game fish taken from waters near hazardous waste sites [ATSDR 2000].
• **FDA Studies**	The U.S. Food and Drug Administration (FDA)□S Total Diet Studies have revealed that total PCB levels have shown a downward trend in concentration from the middle 1970s to the middle 1980s and a relatively steady intake from 1982 to 1997. For example, total diet studies conducted from 1982 to 1984 for adults between the ages of 25 and 30 indicated that the mean daily intake of PCBs was 0.001 micrograms kilograms (µg/kg) body weight day while in the 1997 study, the mean was 0.002 µg/kg body weight/day [ATSDR 2000].
Non-Occupational Exposure: • **Special populations**	People living near incinerators, other PCB-disposal facilities, or NPL hazardous waste sites where PCBs have been detected may receive higher PCB exposures than the general population. These exposures may be through ingestion, inhalation, or skin contact [ATSDR 2000].

	Do-it-yourselfers repairing or removing older construction materials, including plaster, paint, and caulk, that contain PCBs.
Occupational Exposures:	Occupational exposure to PCBs occurs mainly via the inhalation and dermal routes.
• **Inhalation and Dermal**	Commercial PCB mixtures vary from colorless to dark brown oils, and from viscous liquids to sticky resinous semisolids. Although PCBs evaporate slowly at room temperature, the volatility of PCBs increases dramatically with even a small rise in temperature. Equipment that contains PCBs can overheat and vaporize significant quantities of these compounds, creating an inhalation hazard that can be magnified by poor ventilation. Because of their highly lipophilic nature, PCBs also can be absorbed through the skin following contact with contaminated equipment, water, or soil. Products that contain PCBs are no longer manufactured, thus occupational exposure no longer occurs in those settings. However, it might occur • During the maintenance or repair of old equipment that contains PCBs, • As a result of accidents involving such equipment [Schecter AJ and Charles 1991; Wolff 1985], or • During waste-site cleanup or disposal activities [ATSDR 2000; Luotamo et al. 1993; Schecter A et al. 1994]. • During repair or removal of older construction materials, including plaster, paint, and caulk that contain PCBs. Today, PCBs are found mainly in transformers and capacitors manufactured before 1977. Such transformers and capacitors might be found in • Old industrial equipment (e.g., welding equipment), • Medical equipment (e.g., x-ray machines), and

	• Household appliances (e.g., refrigerators, microwaves and televisions).
Key Points	• The primary route of exposure to PCBs in the general population is the consumption of contaminated foods, particularly meat, fish, and poultry. • Occupational exposure to PCBs occurs mainly via the inhalation and dermal routes. o Although occupational exposure no longer occurs as a result of the manufacture of PCB-containing products, it might still occur during the maintenance or repair of equipment manufactured before 1977 that may contain PCBs or as a result of accidents involving such equipment. o Occupational exposure might also occur during waste-site cleanup or disposal activities.
Progress Check	3. Which of the following statements is **NOT CORRECT**? A. The primary route of exposure to PCBs in the general population is consuming contaminated foods. B. Over the past two decades, the general overall trend is decreasing concentrations of PCBs in most environmental media. C. Over the past two decades, PCB body burdens in humans have shown no changes. D. Occupational exposure to PCBs occurs mainly via the inhalation and dermal routes. *To review relevant content, see "Non-occupational and Occupational Exposure Routes" in this section.*

Who Is at Risk of Exposure to PCBs?

Learning Objective	Upon completion of this section, you will be able to • Identify who is at risk of exposure to PCBs.

Introduction	People with potentially high exposures to PCBs include • Recreational and subsistence fishers who typically consume larger quantities of locally caught fish than the general population, • Children with in utero and lactational exposure to PCBs from mothers who eat large quantities of contaminated fish during pregnancy and while nursing, • Certain farmers and their families who consume PCB-contaminated food via their own farm-raised beef and dairy cattle, and • People living near incinerators, other PCB-disposal facilities, or NPL hazardous waste sites where PCBs have been detected. Although PCBs are no longer manufactured in the United States, workplace exposure potentially may exist. In occupational settings, persons who repair and maintain equipment with capacitors and transformers manufactured before 1977 could be exposed to PCBs.
Recreational and Subsistence Fishers	Due to the factors of culture and lifestyles, sport anglers and subsistence fishers may consume 10 times more fish and seafood than average U.S. consumers. Many of these subsistence fishers are American Indian, ethnic minority, or immigrant populations. The special dietary practices of these populations can lead to significant exposures to persistent pollutants [Hovinga et al. 1993; Judd et al. 2004].

Children with Maternal Exposures	**Children of mothers who eat large quantities of contaminated fish may be exposed to PCBs prenatally and while breastfeeding.** Several studies have reported that prenatal exposure to PCBs has been confirmed among children of consumers of contaminated fish and certain other groups [Fein et al. 1984; Jacobson JL et al. 1990b; Jacobson SW et al. 1985; Swain and Swain 1991]. Other studies have indicated that lactating women whose diets are high in PCB-contaminated fish potentially can increase the PCB exposure for their breastfeeding infants [Dewailly et al. 1989; Fitzgerald et al. 1998; Greizerstein et al. 1999; Rogan et al. 1985]. Fetuses and neonates are more sensitive to PCBs than are adults. During these early life stages, the hepatic microsomal enzyme systems that facilitate the metabolism and excretion of PCBs are not fully functional.
Farming Families	**Farmers and their families who consume PCB-contaminated food via their own farm-raised beef and dairy cattle may be exposed.** During the 1940s and 1950s, the insides of concrete silos on many farms in the Midwest United States were coated with sealants containing PCBs. Over time, these sealants peeled off and became mixed with silage used to feed beef and dairy cattle. Farmers and their families who lived on these farms and who regularly ate farm-raised beef and dairy products were exposed to PCBs. Although most of these silos have been dismantled and removed, the remaining silos represent a potential source of exposure to PCBs [Hansen 1987; Humphrey 1983; Schantz et al. 1994].

People Living Near PCB Contaminated Sites	**Persons living near incinerators, other PCB-disposal facilities, or NPL hazardous waste sites where PCBs have been detected may be exposed.** Persons living near incinerators, other PCB-disposal facilities, or any of the 500 current or former hazardous waste sites on the NPL sites where PCBs have been found may be also at increased risk for exposure to PCBs [ATSDR 1987; Hazdat 2000; Hermanson and Hites 1989; Robertson and Ludewig 2011; Stehr-Green et al. 1988; Wester et al. 1993].
Persons with Impaired Hepatic Function	PCBs are metabolized mainly in the liver, thus, persons with impaired hepatic function might be at increased risk because their ability to detoxify and excrete these compounds is diminished. Persons with incompletely developed glucuronide conjugation mechanisms (such as Gilbert syndrome or Crigler-Najjar syndrome) have impaired liver function, as do persons with chronic liver diseases such as cirrhosis or hepatitis B [Calabrese et al. 1977; Lester et al. 1964]. Similarly, because hepatic function normally declines with age, elderly persons may be more susceptible to the effects of exposure to PCBs.
Children's Susceptibility	Infants and young children consume a greater amount of food per kilogram of body weight than do adults. Therefore, they have proportionately greater exposure to PCBs than do adults eating food with the same level of contamination [ATSDR 2000]. In addition, as mentioned earlier, fetuses and neonates are potentially more sensitive to PCBs than are adults because their hepatic microsomal enzyme systems that facilitate the metabolism and excretion of PCBs are not fully functional.
Exposure in the Workplace	PCB levels in blood and body tissues were 10–1,000 times higher in persons exposed to PCBs in the workplace than in non-occupationally exposed persons [Kreiss and Kreiss 1985; Wolff 1985; Yakushiji et al. 1978].

The United States no longer produces PCBs or products containing PCBs (e.g., capacitors, transformers, and electrical equipment), thus occupational exposure to PCBs no longer occurs in those settings. However, workers can have inhalation or dermal contact with PCBs when repairing or performing routine maintenance on older equipment or electrical transformers, and during accidents or spills involving PCBs [Fait et al. 1989; Schecter AJ and Charles 1991; Welsh 1995; Wolff 1985]. Exposure can also occur during the disposal of materials containing PCBs at hazardous waste sites, waste-site cleanup, or demolishing buildings containing PCBs [Luotamo et al. 1993; Robertson and Ludewig 2011].

Specific occupations with risk for exposure to PCBs in the National Occupational Exposure Survey (NOES)[NIOSH 1989] include

- Construction work,
- Demolition work,
- Electric cable repair,
- Electroplating,
- Emergency response,
- Firefighting,
- Hazardous waste hauling or site operation,
- Heat exchange equipment repair,
- Maintenance or cleaning,
- Medical laboratory technician or technologist,
- Metal finishing,
- Non-cellulose fiber industry,
- Paving and roofing,
- Pipefitting or plumbing,
- Semiconductor and related industries,
- Timber products manufacturing,
- Transformer or capacitor repair, and
- Waste-oil processing.

Key Points

- Recreational and subsistence fishers who consume large amounts of contaminated fish may be at increased risk for high-level exposure to PCBs.
- Populations with increased exposure to PCBs include

- Children of mothers who eat large quantities of contaminated fish during pregnancy and while nursing;
- Farm families who eat PCB-contaminated food; and
- Persons who live near incinerators, other PCB-disposal facilities, or NPL hazardous waste sites where PCBs have been detected.

- Persons with compromised hepatic function might metabolize PCBs less efficiently than healthy persons.
- Although the United States no longer manufactures PCBs, workers can be exposed to PCBs during repair of equipment manufactured before 1977, accidents or spills involving PCB, and waste-site cleanup or disposal activities.

Progress Check

4. Of the following, who may be at increased risk of high-level exposure to PCBs?

   A. Sport anglers and subsistence fishers.
   B. Workers whose jobs include routine maintenance of equipment or electrical transformers manufactured before 1977.
   C. The children of mothers who eat large quantities of contaminated fish during pregnancy and while nursing.
   D. All of the above.

   To review relevant content, see all topics in this section.

What Standards and Regulations Exist for PCB Exposure?

Learning Objective

Upon completion of this section, you will be able to

- Describe EPA's maximum contaminant level (MCL) for PCBs in drinking water.
- Describe FDA's tolerance levels for PCBs in food.

Introduction	The U.S. government has developed standards and regulations for PCBs that are designed to protect the public and workers from potential adverse health effects.
Workplace Standards	**Air** The Occupational Safety and Health Administration (OSHA)☒ permissible exposure limit (PEL) is a time-weighted average (TWA) airborne concentration of 1.0 milligrams per cubic meter (mg m^3) for PCBs containing 42☐ chlorine (average molecular formula of $C_{12}H_7Cl_3$). The PEL for PCBs with 54☐ chlorine and an average molecular formula of $C_{12}H_5Cl_5$ is 0.5 mg m^3 (OSHA 1998a). Both standards encompass all physical forms of these compounds: • Aerosols, • Vapor, • Mist, • Sprays, and • PCB-laden dust particles. OSHA recognizes that PCBs can be absorbed through intact skin; therefore, both dermal and inhalation exposure routes should be evaluated by an industrial hygienist. The National Institute for Occupational Safety and Health (NIOSH)FDA recommends a 10-hour TWA of 1.0 micrograms per cubic meter (µg/m^3) based on the minimum reliable detectable concentration and the potential carcinogenicity of PCBs [NIOSH 2005]. NIOSH also recommends that all workplace exposures be reduced to the lowest feasible level.

Environmental Standards

Drinking Water

EPA considers PCBs a probable human carcinogen and prohibits industrial discharges under the Clean Water Act Effluent Guidelines.

EPA's goal for drinking water's maximum contaminant level is zero, and the enforceable MCL for PCBs in public water systems is 0.0005ppm [EPA 2001].

EPA requires that PCB spills or accidental releases into the environment of 1 pound or more be reported to EPA [ATSDR 2000].

Food

FDA mandates tolerances of 0.2–3.0 ppm PCBs for all foods, with a tolerance level in fish of 2 ppm. FDA also limits PCBs in paper food-packaging materials to 10 ppm [FDA 1996c].

The Food and Agriculture Organization (FAO) and the World Health Organization (WHO) allow a daily PCB intake of 6 µg/kg per day [AAP 2003].

Table 1. Standards, regulations, and recommendations for PCBs

Agency	Focus	Level	Comments
OSHA	Air: workplace	1.0 mg m^3 for PCBs with 42% Cl 0.5 mg m^3 for PCBs with 54% Cl	Enforceable; TWA, PEL Both standards encompass all physical forms of aerosols, vapor, mist, sprays, and PCB-laden dust particles.
NIOSH	Air: workplace	1.0 µg/m^3	Advisory; TWA (10-hour)

	EPA	Drinking water: environment	0.0005 ppm	Enforceable MCL□
	FDA	Food: environment	0.2□3.0 ppm (all foods) 2.0 ppm (fish) 10 ppm (paper food-packaging materials)	Enforceable; Tolerance level
	WHO FAO	Food: environment	6.0 µg/kg per day	Allowable daily intake
	□TWA (time-weighted average): TWA concentration for a normal workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed □PEL (permissible exposure limit): highest level of PCBs in air to which a worker may be exposed, averaged over an 8-hour workday □MCL (maximum contaminant level): enforceable level for drinking water µg/kg: microgram per kilogram µg/m^3: microgram per cubic meter ppm: parts per million			
Key Points	• OSHA□s PEL is 1,000 µg/m^3 for PCB mixtures 42□ chlorinated and 500 µg/m^3 for compounds 54□ chlorinated. • EPA□s enforceable MCL for PCBs in public drinking-water systems is 0.0005 ppm. • FDA□s tolerance levels for PCBs in food range between 0.2 and 3 ppm.			

Progress Check	5. Which of the following statements is **FALSE** regarding U.S. standards for PCBs levels? A. EPA has set an enforceable MCL for PCBs in public drinking water systems. B. EPA considers PCBs a probable human carcinogen and prohibits industrial discharges under the Clean Water Act Effluent Guidelines. C. FDA has banned PCBs in paper food-packaging materials. D. NIOSH recommends that all workplace exposures to PCBs be reduced to the lowest feasible level. *To review relevant content, see "Environmental Standards" in this section.*

What Is the Biologic Fate of PCBs in Humans?

Learning Objective	Upon completion of this section, you will be able to • Describe the characteristics of PCB metabolism in humans.
Introduction	Rates of PCB metabolism vary greatly with the degree of chlorination of the biphenyl rings and the positions of the chlorines on these rings. In the environment, PCBs undergo environmental alterations through • Partitioning, • Chemical transformation, and • Preferential bioaccumulation. As a result, compositions of environmental PCB mixtures differ from commercial PCB mixtures (original Aroclors).

Absorption and Distribution	Humans can absorb PCBs by the: • Inhalation, • Oral, and • Dermal routes of exposure. Although PCBs are readily absorbed into the body, they are slowly metabolized and excreted. PCBs initially distribute preferentially to the liver and muscle tissue. PCBs, especially the highly chlorinated congeners, tend to accumulate in lipid-rich tissues due to their lipophilic nature. Greater relative amounts of PCBs are usually found in • Adipose tissue, • Breast milk, • The liver, and • Skin [ATSDR 2000; Matthews et al. 1984].
Metabolic Pathways	The liver is the primary site of metabolism of PCBs, which occurs via hydroxylation and conjugation with glucuronic acid and sulfates. PCBs are metabolized by the microsomal monooxygenase system catalyzed by cytochrome P-450 to phenols (via arene oxide intermediates), which can be conjugated or further hydroxylated to form a catechol [Safe SH 2007]. Glucuronide and sulfate conjugates are excreted mainly in the urine, whereas hydroxylated metabolites are excreted mainly in the bile. The rate of individual congener metabolism depends on the number and position of chlorine atoms. Steele et al. estimated the half-life in humans for lower chlorinated biphenyls (Aroclor 1242) as 6□7 months and the corresponding half-life for the more highly chlorinated biphenyls as 33□34 months [Steele et al. 1986]. Phillips et al. measured total PCBs in capacitor workers and calculated half lives of 2.6 and 4.6 years for the lower (Aroclor 1242) and higher (Aroclor 1254) chlorinated

	biphenyls, respectively [Phillips et al. 1989]. A more recent study, taking into account of high initial body burden, ongoing environmental exposure, low serum levels, and congeners with very long half-lives, has showed the estimated half-lives during a period of high internal dose were 1.74 years for Aroclor 1242 and 6.01 years for Aroclor 1254. Half-lives during a period of low internal dose were estimated to be 21.83 years and 133.33 years for Aroclor 1242 and Aroclor 1254, respectively [Hopf et al. 2013]. In general, less-chlorinated PCB congeners are more readily metabolized than are highly chlorinated congeners. As a result of this preferential metabolism, highly chlorinated congeners tend to remain in the body longer than do less-chlorinated congeners. Highly chlorinated congeners therefore tend to become more concentrated in adipose tissues, where they are stored in solubilized form.
Excretion	PCBs are primarily excreted after they have been conjugated and transformed into more polar metabolites. The major routes of excretion of PCBs are fecal and urinary.

Environmental Alteration of PCB Mixtures	Environmental PCBs occur as mixtures whose compositions differ from the commercial mixtures. This is because after release into the environment, PCB mixture composition changes over time through chemical transformation and preferential bioaccumulation [Cogliano 1998]. Chemical transformation can occur through biodegradation of PCB mixtures in the environment. PCBs with higher chlorine content are extremely resistant to oxidation and hydrolysis. Preferential bioaccumulation occurs in living organisms. Bioaccumulation through the food chain tends to concentrate congeners of higher chlorine content. In humans, bioaccumulated PCBs also appear to be more persistent in the body [Hovinga et al. 1992]. This is significant because bioaccumulated PCBs appear to be more toxic than original Aroclors in animals [Aulerich et al. 1986; Cogliano 1998].
Key Points	• PCBs are stored in adipose tissues. • The liver is the primary site of metabolism of PCBs. • The slow metabolism and high lipid solubility of PCBs lead to bioaccumulation. • Binding of PCB metabolites to nucleophilic cellular macromolecules may contribute to the toxic effects of PCBs. • After release into the environment, PCBs occur as mixtures whose compositions differ from the commercial mixtures. Bioaccumulated PCBs also appear to be more persistent in the body.
Progress Check	6. Which of the following statements about the biologic fate of PCBs is **NOT CORRECT**? A. The liver is the primary site of PCBs metabolism, which occurs via hydroxylation and conjugation with glucuronic acid and sulfates. B. Less-chlorinated congeners are more readily metabolized than are highly chlorinated congeners. C. Highly chlorinated congeners tend to become more concentrated in muscle.

D. Bioaccumulated PCBs appear to be more persistent in the body.

To identify relevant content, see all topics in this section.

What Are Adverse Health Effects of PCB Exposure?

Learning Objective

Upon completion of this section, you will be able to

- Describe adverse health effects associated with exposure to PCBs.

Introduction

Human exposures to relatively high levels of PCBs have occurred primarily in persons working in plants that extensively manufactured and used PCBs and PCB-containing equipment. Occupational exposure to PCBs can result in a broad spectrum of effects that includes

- Increased levels of some liver enzymes, with possible hepatic damage,
- Chloracne and related dermal lesions, and
- Respiratory problems [Alvares et al. 1977; Chase et al. 1982; Emmett and Emmett 1985; Lawton et al. 1985; Meigs 1954; Ouw et al. 1976; Safe S 1993; Warshaw et al. 1979].

Potential adverse human health effects of low-level environmental exposure to PCBs are complex and still need further validation [Safe SH 2007].

In animal studies, commercial PCBs elicit a broad range of toxic responses including:

- Acute lethality,
- Body weight loss,
- Carcinogenesis,
- Dermal toxicity,
- Fatty liver,
- Genotoxicity,
- Hepatomegaly,
- Immunosuppressive effects,
- Neurotoxicity,

	• Porphyria, • Reproductive and developmental toxicity, • Thymic atrophy, and • Thyroid hormone-level alterations. This adverse health effects section addresses PCBs as a whole.
Mechanism of PCB Toxicity	PCBs are metabolized by the microsomal monooxygenase system catalyzed by cytochrome P-450 to phenols (via arene oxide intermediates), which can be conjugated or further hydroxylated to form a catechol. Arene oxide intermediates are electrophilic in nature. They can covalently bind to nucleophilic cellular macromolecules (e.g., protein, DNA, RNA) and induce DNA strand breaks and DNA repair, which can contribute to the toxic response of PCBs. Additionally, arene oxide intermediates can be conjugated with glutathione and further metabolized to form methylsulfonyl metabolites, which have been identified in human serum and tissue samples and in laboratory animals. Binding of methylsulfonyl metabolites to some proteins may contribute to some of the toxic effects of PCBs. It has also been hypothesized that hydroxylated PCB metabolites could contribute to the toxicity of PCBs [ATSDR 2000; Safe SH 2007].

Dermatologic Effects	Chloracne and related dermal lesions have been reported in workers occupationally exposed to PCBs. Mild to moderate chloracne was observed in 7 of 14 workers exposed to 0.1 mg m□Aroclors for an average duration of 14.3 months [Meigs 1954]. Among 80 workers who manufactured capacitors in Italy, 10 cases of acne or folliculitis, or both, and 5 cases of dermatitis were reported. All of the workers with chloracne were employed in high exposure jobs. Their blood PCB concentrations ranged from 41 to 1319 □g kg [Maroni et al. 1981]. In a person with PCB-induced chloracne, the acne-like lesions arise as a result of inflammatory responses to irritants in the sebaceous glands. Chloracne usually begins with the formation of keratin plugs in the pilosebaceous orifices. The resulting inflammatory folliculitis stimulates keratinization of the sebaceous gland ducts and outer root sheath of the hair, leading to the formation of keratin cysts. The chin, periorbital, and malar areas are most often involved, although lesions might also appear in areas not usually affected by acne vulgaris (e.g., the chest, arms, thighs, genitalia, and buttocks). The most distinctive lesions are cystic and measure 1□10 mm, although comedonal lesions can also be present. The cysts and comedones can become inflamed and secondarily infected, and papules and cysts can be surrounded by edema and erythema [Crow 1970; Letz 1983]. Chloracne generally indicates systemic toxicity and can be caused by not only dermal contact but also ingestion of PCBs. However, the absence of chloracne does not rule out exposure [Kimbrough 1980; Letz 1983]. No reliable dose-response model exists for chloracne in exposed populations, and the dose-response relationship might be dependent on individual predisposition. Chloracne typically develops weeks or months after exposure. The lesions are often refractory to treatment and can last for years or decades.

	In addition to chloracne, other dermal effects noted in some PCB-exposed workers include pigmentation disturbances of skin and nails, erythema and thickening of the skin, and burning sensations [Fischbein et al. 1982; Fischbein et al. 1979; Ouw et al. 1976; Smith et al. 1982]. Skin effects were reported widely among victims of the Yusho (Japan) and Yu-Cheng (Taiwan) poisoning episodes in 1968 and in 1978, respectively. In these episodes, persons were exposed to PCBs and their heat-degradation products, mainly polychlorinated dibenzofurans (PCDFs). Exposure to PCBs occurred by consuming rice oil that had become contaminated by heat-degraded PCBs during processing. Unlike usual PCB mixtures, the Yusho and Yu-Cheng mixtures were heated in thermal heat exchangers during the cooking process, resulting in contamination of the oil by chlorinated dibenzofurans as well as PCBs. This co-contamination created controversy [Anonymous 1997; Kimbrough et al. 2003; Ross 2004] about the extent to which the health effects observed in the Yusho and Yu-Cheng populations can be attributed to PCBs legitimately, as opposed to the dibenzofuran co-contaminants. No adverse dermal effects have been reported in persons who consume large amounts of Great Lakes fish contaminated with PCBs and other environmentally persistent chemicals, or in other cohorts from the general population. However, whether this outcome was systematically studied in these cohorts is unknown [ATSDR 2000]. A skin lesion exactly like chloracne in humans has been observed in several species of animals experimentally exposed to PCBs [Allen 1975]. After monkeys incur long-term oral exposure to commercial PCB mixtures, related dermal effects are well characterized and generally are similar to those observed in humans [ATSDR 2000].
Key Points	• Conclusive evidence that exposure to PCBs induces adverse dermal effects in humans exists.

	• A typical dermal sign of exposure is chloracne.
Reproductive and Developmental Effects	Courval, DeHoog et al. [1999] conducted a study of 626 married couples in Michigan. The relative risk of conception failure (defined as inability to conceive after 12 months) rose in men but not in women with increasing consumption of PCB-contaminated fish. Some evidence shows that increased intake of PCB-contaminated fish can shorten menstrual cycle length, but no adverse association was found between the duration of fish consumption and time-to-pregnancy in the same population. In a study of 1,820 multigravida women, no significant association was found between low-to-moderate PCB intake and clinically recognized spontaneous fetal death [Mendola et al. 1995]. A recent oocupational cohort study examined the data from 2595 live births of female workers from three capacitor plants and found no evidence of altered sex ratio among children born to PCB-exposed female workers [Rocheleau et al. 2011]. The first epidemiologic investigation to demonstrate an association between the amounts of PCB-contaminated fish eaten by pregnant women and behavioral deficits in their newborns was the Michigan Maternal Infant Cohort Study, published in 1984 [Fein et al. 1984; Jacobson SW et al. 1985]. In this study, developmental and cognitive deficits were observed in the children of mothers who had eaten moderate to high amounts of contaminated fish during the six years preceding pregnancy and who continued to do so during pregnancy. Developmental effects in this population included statistically significant decreases in • Gestational age (4.9 days), • Birth weight (160□190 grams), and • Head circumference (0.6 centimeters). In addition, infants born to mothers who had eaten the greatest amount of contaminated fish during pregnancy exhibited weaker reflexes, greater motor immaturity,

and more pronounced startle responses compared with infants born to women who had consumed less fish.

It is essential that women of childbearing age be aware of fish advisories to ensure they not only limit their consumption of fish with elevated PCB levels but also learn how to prepare fish to limit their PCB ingestion.

Follow-up studies of the children from this cohort have demonstrated that the effects of perinatal exposure to PCBs are persistent. At four years of age, these children still had deficits in

- Weight gain,
- Depressed responsiveness, and
- Reduced performance on the visual recognition-memory test.

At 11 years of age, the children of highly exposed mothers were

- Three times more likely than controls to have low full-scale verbal IQ scores,
- Twice as likely to lag behind at least 2 years in reading comprehension, and
- More likely to have difficulty paying attention [Jacobson JL et al. 1990a, 1990b].

Recent studies indicate that maternal consumption of PCB-contaminated fish can cause disturbances in reproductive parameters and neurobehavioral and developmental deficits in newborns and older children. Prenatal exposure to PCBs from the mother□s body burden, rather than exposure through human milk, is believed to account for the developmental effects of these compounds [Jacobson JL et al. 1996; Longnecker et al. 2003; Ribas-Fito et al. 2001; Schantz et al. 2003].

Similar reproductive, developmental, and neurobehavioral deficits have been reported in children born to women who were pregnant during the Yusho and Yu-Cheng incidents [Hsu et al. 2003; Hsu et al. 2005; Yang et al. 2005].

	Developmental delays were seen at all ages and were greater in children who were smaller and had neonatal signs of intoxication or nail deformities, or both. Follow-up testing indicated that effects on cognitive development persisted for several years after exposure [Guo et al. 1995]. In rhesus monkeys, exposure to PCBs is associated with alterations in the menstrual cycle, decreases in fertility, increases in spontaneous abortion, and a reduced number of conceptions [Arnold et al. 1990; Barsotti et al. 1976].
Key Points	• Reproductive function may be disrupted by exposure to PCBs. • Neurobehavioral and developmental deficits have been reported in newborns exposed to PCBs in utero.
Endocrine Effects	Limited but corroborative occupational data indicate a potential for toxic effects in the thyroid system in humans. Studies that have examined relationships between exposure to PCBs and thyroid hormone status have reported a variety of results. Findings include both negative and positive significant correlations between exposure to PCBs and circulating levels of thyroid-stimulating hormone (TSH), T4, or T3. These findings are dependent on the: • Specific type of analysis for exposure to PCBs, • Age of the cohort, and • Specific exposure scenario [Emmett et al. 1988; Koopman-Esseboom et al. 1994; Langer et al. 1998; Longnecker et al. 2003; Nagayama et al. 1998; Osius et al. 1999]. In a Dutch population, elevated levels of PCBs correlated with lower maternal levels of circulating triiodothyronine and total thyroxine and with higher plasma levels of TSH in infants during the second week and third month after birth. Infants exposed to higher levels of PCBs also had lower plasma levels of free

	thyroxine and total thyroxine in the second week after birth [Koopman-Esseboom et al. 1994]. In addition, a significantly elevated odds ratio for goiter was found among the Yu-Cheng cohort [Guo et al. 1999], suggesting the possibility of excessive thyroid disease in a population that experienced relatively high exposures to mixtures of PCBs and PCDFs. Thyroid hormones are essential for normal behavioral, intellectual, and neurologic development. Thus, the deficits in learning, memory, and attention processes among the offspring of women exposed to PCBs are partially or predominantly mediated by alterations in hormonal binding to the thyroid hormone receptor [ATSDR and EPA 1998]. Some PCB congeners are capable of competing with endogenous hormone for binding to this receptor, suggesting a possible mechanism of thyroid toxicity. Hydroxylated PCB metabolites appear to be particularly potent in this regard [ATSDR 2000]. Studies in animals, including rodents and primates, provide evidence of thyroid hormone involvement in PCB toxicity. The most convincing evidence that PCBs can exert toxicity by disrupting thyroid hormone system derives from two studies in rats [Cooke et al. 1996; Goldey et al. 1998]. The contribution of persistent organic pollutants (POPs) exposure to the incidence of diabetes has received little attention until recently. Recent studies in populations exposed to PCBs and chlorinated pesticides found a dose-dependent elevated risk of diabetes [Carpenter 2008].
Key Points	• The epidemiological studies suggest a link between exposure to PCBs and thyroid hormone toxicity in humans. • Studies in animals provide evidence of thyroid hormone involvement in the mechanism of PCB toxicity.
Hepatic Effects	Evidence for liver effects of occupational exposure to PCBs is essentially limited to elevation of serum liver

enzymes that are routinely examined in clinical assays. These serum liver enzymes include aspartate aminotransferase (AST), alanine aminotransferase (ALT), glutamyl transpeptidase (GGT) and other biochemical indices (e.g., bilirubin). No overt hepatotoxicity has been seen in workers exposed to PCBs [ATSDR 2000].

A cross-sectional survey found no significant differences in liver function test results between workers who manufacture capacitors with low-level chronic exposure and non-exposed controls [Fischbein et al. 1979]. However, in another cross-sectional study, liver function tests showed abnormalities that seemed to correlate with serum PCB levels [Maroni et al. 1981].

Increases in urinary excretion of porphyrins appear to be associated with occupational exposure to PCBs, an effect that is believed to be secondary to the induction of hepatic microsomal enzymes. Total bilirubin levels exhibit a positive correlation with serum PCB levels [Colombi et al. 1982; Maroni et al. 1984; Smith et al. 1982].

PCBs are more potent enzyme inducers than phenobarbital, a drug that occasionally causes clinical problems due to its microsomal enzyme-inducing effects. The health implications of enzyme induction include the occurrence of disease secondary to increased metabolism of endogenous or exogenous substances and interference in medical therapy due to increased metabolism of administered drugs. The enzyme-inducing effects of PCBs can persist long after cessation of exposure [Letz 1983].

In the Yu-Cheng population, the incidence of chronic liver disease and cirrhosis was significantly higher than the incidence of these conditions in the general population of Taiwan. Asymptomatic hepatomegaly has been reported in exposed workers, many of whom had concomitant elevated serum PCB levels. Due to the mixed chemical nature of the exposure, the results cannot be attributed solely to PCBs [ATSDR 2000].

	Liver damage is a consistent and prominent finding among animals exposed to PCBs, particularly rats and monkeys, which are the species tested most extensively. Liver effects are similar in nature among species and appear to be reversible when mild. Liver effects characteristically include • Fat deposition, • Fibrosis, • Hepatic microsomal enzyme induction, • Increased serum levels of liver-related enzymes indicative of possible hepatocellular damages, • Liver enlargement, and • Necrosis [ATSDR 2000].
Key Points	• Although liver damage is common in animals exposed to PCBs, overt hepatotoxicity is uncommon in humans. • Exposure to PCBs can increase serum levels of hepatic enzymes and can induce microsomal enzyme function.
Carcinogenic Effects	Epidemiologic studies have raised concerns about the potential carcinogenicity of PCBs. A retrospective analysis of a study of two plants that manufactured electrical capacitors in the United States found a significant increase in the incidence of cancer. The primary target tissues for the cancers were the liver, gallbladder, and biliary tract [Brown 1987]. Likewise, an increased incidence of melanomas associated with exposure to PCBs has also been observed for workers who manufactured capacitors [Bahn et al. 1976; Ruder et al. 2006; Sinks et al. 1992]. Sinks et al. [1992] observed the increased risks for brain cancer among workers exposed to PCBs in an electrical capacitor manufacturing plant in Indiana, and this finding has been further confirmed by a recent study from Ruder et al. [2006]. One study suggests that exposure to electrical insulating fluids, for which the main constituent is PCBs, may

cause malignant melanoma of the skin [Loomis et al. 1997].

The results of a mortality study of workers employed between 1944 and 1977 at an electrical capacitor manufacturing plant were recently reported. The report pointed out that PCBs alone or in combination with other chemicals could be associated with increased risks for

- Liver or biliary,
- Stomach, intestinal, and
- Thyroid cancers [Mallin et al. 2004].

A recent analysis of a cohort of 24,865 capacitor-manufacturing workers exposed to PCBs at three plants showed evidence of associations between cumulative exposure to PCBs and increased total cancer and intestinal cancer mortality among female long-term workers and excess myeloma for male long-term workers [Ruder et al. 2014].

In contrast, increased cancer incidence was not observed in male workers who manufactured capacitors in Sweden exposed to PCBs for an average of 6.5 years [Gustavsson et al. 1986]. The results from the Swedish study, however, cannot rule out the possibility of a carcinogenic risk from PCB exposure because of the small size of the cohort and relatively brief follow-up period.

Different mixtures of PCBs had different potencies and, thus, different toxicity. As noted previously, PCB mixtures found in the environment are different from commercial PCB mixtures. EPA agreed that some mixtures of PCBs are more likely to cause cancer than others, and found that all PCBs mixtures can cause cancer [Cogliano 1998; EPA 1996c].

In environmental case-control studies that compared PCB concentrations in breast tissue in both women with (case patients) and without (case controls) breast cancer, some studies reported higher levels of total PCBs among case patients than control patients [Falck et al. 1992; Guttes et al. 1998; Wassermann et al.

1976]. Other studies found no elevated PCB levels in breast tissue in patients with breast cancer [Aronson et al. 2000; Liljegren et al. 1998; Unger et al. 1984]. A recent occupational cohort study found no overall elevation in breast cancer risk after occupational exposure to PCBs [Silver et al. 2009].

In persons without known occupational exposure to PCBs, elevations of PCB level in the adipose tissue and serum have been associated with an increased risk of non-Hodgkin lymphoma (NHL) [De Roos et al. 2005; Engel et al. 2007; Hardell E et al. 2001; Hardell L et al. 1996; Rothman et al. 1997].

After registering as Yusho victims, 887 male and 874 female patients were observed for an average 11 years. A retrospective study found statistically significant increased liver cancer mortality rates among the males compared to national liver cancer mortality rates [Kuratsune et al. 1987].

A retrospective mortality study of 1940 Yu-Cheng cases found no statistically significant increased mortality from liver and intrahepatic bile duct cancers [Hsieh et al. 1996].

Before the comprehensive study conducted by Mayes et al. [1998], only commercial mixtures 60□ chlorinated had been tested, and controversy existed about whether mixtures with lower chlorine content were carcinogenic. The Mayes et al. study [Mayes et al. 1998] supported the position that all PCB mixtures can cause cancer. Data from animal studies have shown that PCBs cause gastrointestinal tract tumors, hepatocarcinomas, leukemia, lymphomas, and pituitary tumors [ATSDR 2000].

On the basis of these laboratory data, EPA has determined that PCBs are probable human carcinogens and has assigned them the cancer weight-of-evidence classification B2 [IRIS 2012]. DHHS concluded that PCBs are reasonably anticipated to be carcinogenic in humans based on sufficient evidence of carcinogenicity

	in animals [NTP 2011]. In February 2013, 26 experts from 12 countries met at the International Agency for Research on Cancer (IARC), Lyon, France, to reassess the carcinogenicity of PCBs. The Working Group considered more than 70 independent epidemiological studies with informative data for carcinogenicity of PCBs in human beings. On the basis of sufficient evidence of carcinogenicity in humans and experimental animals, the IARC classified PCBs as carcinogenic to humans (Group 1). The classification is based on consistent association between exposure to PCBs and increased risk of melanoma in humans [IARC 2013].
Key Points	• On the basis of data from animal studies, DHHS and EPA consider PCBs a probable human carcinogen. • On the basis of sufficient evidence of carcinogenicity in humans and experimental animals, the IARC classified PCBs as carcinogenic to humans (Group 1).
Other Effects	Occupational and epidemiologic studies have suggested or demonstrated other adverse health effects from exposure to PCBs. These health effects can involve the: • Cardiovascular, • Gastrointestinal, • Immune, • Musculoskeletal, and • Neurological systems. In southwest Quebec, adults who ate fish from PCB-contaminated waters had • Significantly greater motor retardation, • Poorer results on certain memory and attention tests, and • Higher scores on a standardized confusion scale than did control adults.

	These neurological deficits were directly related to the frequency of fish consumption [Mergler et al. 1998]. Immune system effects reported in PCB-exposed populations include alterations in the ratio of helper to killer (CD4 CD8) T-cells, decreases in IgA and IgM antibody levels, decreases in monocyte and granulocyte counts, and decreases in natural killer cell count [Svensson et al. 1994]. In the Yusho and Yu-Cheng populations, the immunosuppressive effects of PCB exposure were associated with an increased incidence of persistent respiratory infection and enhanced responsiveness to mitogens [Guo et al. 1995]. Appetite loss has been reported in transformer and electrical equipment manufacturing workers exposed to various PCB-containing mixtures. Other nonspecific gastrointestinal symptoms experienced by workers exposed to PCBs include nausea, epigastric distress and pain, and intolerance to fatty foods [Emmett et al. 1988; Smith et al. 1982]. A recent study has indicated that several PCB metabolites induce gene mutations, chromosome breaks, chromosome loss and polyploidization in cells in culture and even provided the first evidence that a PCB congener is mutagenic in vivo [Robertson and Ludewig 2011].
Key Points	• Additional adverse effects of PCBs may involve the o Cardiovascular, o Gastrointestinal, o Genetic systems, o Immune, o Musculoskeletal, and o Neurological systems.

Progress Check

7. Adverse dermal effects have been reported in the following subjects **EXCEPT**

   A. Workers occupationally exposed to PCBs.
   B. Those in the general population who consume large amounts of fish contaminated with PCBs and other environmentally persistent chemicals.
   C. Victims of the Yusho (Japan) and Yu-Cheng (Taiwan) poisoning episodes exposed to PCBs and their heat-degradation products.
   D. Monkeys after long-term oral exposure to commercial mixtures of PCBs.

   To identify relevant content, see "Dermal Effects" in this section.

8. Which of the following statements about the potential carcinogenicity of PCBs is considered **INCORRECT**?

   A. Potential human health effects from exposure to mixtures of PCBs do not include cancer.
   B. Some mixtures of PCBs are more likely to cause cancer than others.
   C. Exposure to PCBs has been associated with increased incidence of some cancers.
   D. Data from animal studies have shown clearly that PCBs cause different kinds of tumors.

   To identify relevant content, see "Carcinogenic Effects" in this section.

9. Additional adverse effects of PCBs may include which of the following?

   A. Liver damage.
   B. Neurobehavioral and developmental deficits.
   C. Thyroid hormone anomalies.
   D. All of the above.

   To identify relevant content, see "Other Effects" in this section.

Clinical Assessment

Learning Objective	Upon completion of this section, you will be able to • Describe characteristic findings on clinical assessment of patients exposed to PCBs, and • Describe a rational approach for evaluating a patient with a history of occupational or environmental exposure, or both, to PCBs.
Introduction	Patients who have been exposed to PCBs often are undergoing clinical assessment long after their last exposure occurred (possibly years). The ability to extrapolate peak blood levels is problematic in these cases. PCBs have low acute toxicity but are of public health concern because they persist in the environment, bioaccumulate in human and animal tissues, and potentially can cause chronic or delayed toxicity. Documenting an adequate occupational and environmental exposure history in addition to a physical examination is essential for identifying health effects related to PCBs. Identifying cases of chloracne may be helpful, but the absence of chloracne would not rule out significant exposure.
Patient History and Physical Examination	A detailed history will facilitate the diagnosis of chronic PCB poisoning. Pertinent information includes occupational histories of all household members and history of the patient□s sport and subsistence fish consumption. Because PCBs are hepatotoxins, history of exposure to other potentially hepatotoxic agents, such as ethanol intake and medications with known hepatotoxicity, should be obtained. During the physical examination, physicians should pay particular attention to the skin and hepatic systems. Encountering a patient with PCB toxicity should trigger consideration of whether this is a sentinel event,

	indicating the possibility of other similarly exposed persons such as co-workers or family members.
Signs and Symptoms-Acute Exposure	**Acute exposure** PCBs have very low potential for producing acute toxic effects. The only overt sign of exposure to PCBs is chloracne, which is a specific skin lesion. Although chloracne may resemble typical adolescent acne, it has certain distinct features [Crow 1970; Letz 1983]. • Chloracne's most distinctive feature is cystic, skin colored lesions that measure 1–10 mm. • Chloracne's other prominent feature is comedonal lesions. The comedones and cysts can become inflamed and secondarily infected with large pustules. Unlike adolescent acne, chloracne may occur at any age and may involve the arms, back, face, legs, neck, and trunk. Chloracne can be very persistent and refractory to treatment. Acneiform lesions do not appear in all severely exposed patients, so the absence of chloracne does not rule out exposure. New cases of chloracne should be reported to the local or state health department. Other acute effects that may be seen include eye irritation, nausea, and vomiting [LaDou 2006]. Elevated liver enzymes are the most sensitive indicator of exposure to PCBs in animals, and alterations in • AST (SGOT), • GGT (GGTP), • Bilirubin, and • Albumin levels have been reported in human epidemiologic studies.

	The absence of alterations in these liver function markers does not rule out excessive exposure to PCBs. The presence of specific signs, symptoms, or laboratory abnormalities, with the possible exception of chloracne, is difficult to relate to exposure to PCBs absolutely in any given patient. A practical approach for the routine work-up of individual patients potentially exposed to PCBs would be to do the following: • Take a through occupational and environmental exposure history, • Examine the skin, • Order baseline liver function tests, and • If indicated, perform subsequent testing limited to patients with clinical problems or history of extensive exposure such as an accidental spill or a capacitor rupture that caused heavy skin contamination [Letz 1983]. This clinical approach may be used for monitoring electrical utility workers or other persons with some potential for ongoing occupational exposure. Serum PCB level is a useful indicator of a patient□s exposure. Serum PCB tests are readily available at most commercial reference laboratories. However, serum PCB levels may not be consistent with adverse health effects. [Roseman 2005].
Signs and Symptoms-Chronic Exposure	**Chronic exposure** Many people who are chronically exposed to PCBs exhibit no overt signs or symptoms of toxicity. Among persons with hepatic involvement, signs of exposure to PCBs can include • Abdominal pain, • Anorexia, • Jaundice, • Nausea, • Vomiting, • Weight loss, and

	• Uroporphyria. Headache, dizziness, and edema have also been reported (see earlier section on Adverse Health Effects for more detail).
Differential Diagnosis	Occupational exposure to PCBs may be accompanied by exposure to chlorinated dibenzodioxin and dibenzofuran contaminants, which are much more toxic than PCBs in comparative animal studies. These substances can cause chronic fatigue and elevated liver enzymes. Mild chloracne should not be confused with other rashes (e.g., acne, seborrheic keratitis, keratoma). A skin biopsy of lesions may help establish the diagnosis [LaDou 2006].
Medical Surveillance	Workers intermittently exposed to PCBs should have a baseline skin examination and liver function tests. Follow-up examination can be limited to symptomatic persons and workers exposed as a consequence of accidental contamination. For persons with signs and symptoms consistent with high exposures to PCBs (e.g., chloracne, elevated AST and ALT), a serum PCB level should be obtained to confirm exposure.
Key Points	• Chloracne is the only known overt sign of PCB toxicity; however, the absence of chloracne does not rule out exposure. • Signs of low level, chronic exposure to PCBs are generally subtle, if present at all.
Progress Check	10. Which of the following should be included in the clinical evaluation of a patient with a history of exposure to PCBs? A. A thorough occupational and environmental exposure history. B. A thorough skin examination. C. Liver function tests. D. All of the above. *To review relevant content, see "Patient History and Physical Examinations" in this section.*

11. PCB exposure may manifest clinically as which of the following?

    A. Acne vulgaris.
    B. Chloracne.
    C. Parkinsonism.
    D. Acute tubular necrosis.

    To review relevant content, see "Signs and Symptoms – Acute Exposure" and "Signs and Symptoms – Chronic Exposure" in this section.

Clinical Assessment - Laboratory Tests

Learning Objective	Upon completion of this section, you will be able to • Describe measurements that can help diagnose exposure to PCBs.
Introduction	The lipophilic nature of PCBs causes them to accumulate in fat; consequently, analyzing biopsied adipose tissue has been used to measure long-term exposure. Serum PCB analysis is less invasive than tissue biopsy, and it can be performed by most commercial reference laboratories. Although such tests are useful for gauging exposure, they may not be consistent with adverse health effects. Select laboratories have the capability to perform PCB analyses on human tissue. Testing human tissue for PCB content, however, remains principally a research tool.
Direct Biologic Indicators	PCBs have been detected in the blood, adipose tissue, and breast milk of non-occupationally exposed members of the general population [CDC 2009; EPA 1986b; Greizerstein et al. 1999; Gunderson and Gunderson 1995; Patterson et al. 2008]. Since the United States stopped making PCB compounds, body burdens of PCBs in humans have decreased. This decrease is evidenced by lower PCB levels reported in human adipose tissue, blood serum, and breast milk [Anderson et al. 1998;

Fensterheim 1993; Hanrahan et al. 1999; Lunden et al. 1998; Schade et al. 1998].

PCB compounds generally can be found at the parts per trillion (ppt) levels in the lipid stores of humans, especially persons living in industrialized societies. The general population is exposed to PCB compounds primarily by ingesting high-fat foods, such as

- Dairy products,
- Eggs,
- Animal fats, and
- Some fish and wildlife [CDC 2009; Patterson et al. 2008].

However, no specified PCB values are deemed normal or toxic levels.

Some researchers believe that PCB levels in the serum and tissue provide a reliable measurement of long-term exposure. PCB levels in the serum and tissue can be measured by many laboratories although analyses results may not be consistent with health effects.

A correlation between increasing levels of serum PCBs and dermatologic findings, including chloracne, has not been found consistently in human epidemiologic studies. However, statistically significant associations between dermatologic effects and plasma levels of higher chlorinated PCB congeners have been reported [Fischbein et al. 1982; Fischbein et al. 1979; Smith et al. 1982].

Although PCBs accumulate in breast milk, the American Association of Pediatrics (AAP) has concluded that the risks posed by PCBs in breast milk are outweighed by the benefits of breastfeeding in all but the most unusual circumstances. Therefore, AAP does not recommend that breast milk be tested for PCBs because the test results would not likely change the recommendation to breast feed. Additionally, AAP recommends consulting local health department officials who are aware of the PCB

	problems in unusual circumstances or where high exposures have occurred [AAP 2003].
Indirect Biologic Indicators	Liver function tests are nonspecific. The combination of asymptomatic hepatomegaly and mild, nonspecific elevations of hepatic enzymes suggests a chronic inflammatory liver process or hepatitis. Hepatitis can be • Drug-induced, • Genetic, • Infectious, • Toxic, • Caused by ethanol ingestion, or • Associated with connective tissue disease. The major cause of liver disease in the United States is ethanol ingestion. Less common causes are environmental exposures, resulting in either acute or chronic toxic hepatitis. Infectious hepatitis includes disease caused by viruses such as A, B, C, and other possible agents of non-A, non-B hepatitis. Hepatitis can also occur with Epstein-Barr virus and cytomegalovirus infections. Some connective tissue diseases such as lupus erythematosus are associated with a specific type of hepatitis. Infiltrative diseases such as sarcoidosis or amyloidosis, and rare genetic diseases such as Wilson disease, primary hemochromatosis, and alpha-1-antitrypsin deficiency, must be excluded. Normal liver enzyme values do not rule out significant PCB exposure; body burden still might be elevated. To help arrive at a diagnosis, viral serology and a heterophil antibody test should be considered. If the

	patient has suggestive signs or symptoms, a serum iron and total iron binding capacity, serum copper and ceruloplasmin, and antinuclear antibodies might help with the diagnosis. Assays for suspected hepatotoxins might also be useful. If other tests do not provide sufficient information, further evaluation might include ultrasound and percutaneous liver biopsy.
Key Points	• Serum or adipose tissue PCB levels can indicate exposure, but they are difficult to interpret clinically. • AAP does not recommend testing breast milk for PCBs, and encourages breastfeeding in all but the most unusual circumstances. • Elevated hepatic enzyme levels are of limited value in diagnosing exposure to PCBs.

Progress Check	12. Which of the following statements is true? A. Testing PCB serum level is expensive and not readily available, but correlates well with health risk. B. AAP recommends that breast milk be tested for PCBs because human milk contains a steroid that inhibits PCB metabolism and excretion. C. The toxic serum PCB value is 20 ppb. D. None of the above. *To review relevant content, see "Direct Biologic Indicators" in this section.*

How Should Patients Exposed to PCBs Be Treated and Managed?

Learning Objectives	Upon completion of this section, you will be able to • Describe the principal treatment strategy for managing PCB poisoning and • Describe the measures for preventing occupational and environmental exposure to PCBs.
Introduction	No specific treatment exists for PCB accumulation. Patients should avoid further PCB exposure and also avoid other hepatotoxic substances, including ethanol.

Acute Exposure	Treat acute skin and eye PCB exposure immediately by flushing with copious amounts of water. However, post-contamination washing cannot ensure removal of all contamination [Wester et al. 1983]. Remove contaminated clothing and discard properly. Carefully observe patients with inhalation exposure for any systemic signs or symptoms of toxicity and administer treatment as necessary. No specific measures are available to reduce respiratory tract absorption. In the rare event of ingestion of PCBs, emesis would be contraindicated because of the high risk of aspiration. The value of administering activated charcoal after ingestion is unknown. Unless a patient has an intact or protected airway, administering charcoal is contraindicated [Alaspaa et al. 2005; Chyka et al. 2005]. Exposed persons should have periodic follow-up examinations with particular attention to hepatic function and dermal lesions.
Key Points	• No antidote exists for PCB exposure; therefore, treatment is supportive.
Chronic Exposure	No specific treatment is available for chronic PCB toxicity. Because no known methods exist for reducing the reserves of PCBs in adipose tissues, purging the body of PCBs should not be attempted. Initial treatment of chloracne is based on • Cessation of PCB exposure, • Good skin hygiene, and • Dermatologic measures commonly used for acne vulgaris. Given the difficulty in treating chloracne, the patient should be referred to a dermatologist. If chronic exposure has occurred due to consuming contaminated fish or game, the patient should be

informed that PCBs tend to accumulate in the body with continued exposure, and counseled about the importance of minimizing further exposure.

In areas with a known PCB problem, state and local public health or natural resources departments typically issue advisories. These advisories specify the waters or hunting areas where PCB-contaminated fish and game likely are, and list the species and size of fish or game that are of concern. Such advisories might completely ban consumption, or might recommend limits on the frequency with which certain species are to be consumed. To minimize the risk for further exposure, sport and subsistence fishers are encouraged to familiarize themselves with and observe advisory recommendations [ATSDR 2000].

Patients should be monitored for increased hepatic enzymes. Because PCBs are hepatotoxins, history of exposure to other potentially hepatotoxic agents should be obtained. To minimize the risk of hepatic damage, patients should be encouraged to avoid exposure to other hepatotoxins, including medications with known hepatotoxicity, ethanol, and chlorinated solvents.

The carcinogenic potential and other risks from exposure to PCBs should be carefully reviewed with the patient.

AAP encourages breastfeeding in all but the most unusual circumstances [AAP 2003].

Prevention in the Workplace	**Work Practices** The following measures [LaDou 2006] may be adopted at work to avoid exposure to PCBs. • Eliminate PCBs from the workplace or implement engineering changes to isolate the PCBs. If neither of these approaches is feasible, then use special PCB-resistant gloves and protective clothing. • Maintain adequate ventilation during spill cleanup or maintenance of vessels containing PCBs. If this is not possible, provide approved respirators. • Make provisions for proper decontamination or disposal of contaminated clothing or equipment. • Post clearly the locations where PCBs are stored as required by law. • Conduct environmental sampling as necessary to ensure adequate worker protection or safety for public reentry to contaminated areas. • Establish reentry or cleanup levels for dioxins and PCBs to protect workers who reoccupy buildings after a PCB fire. • Record health complaints of any type. **Medical Surveillance** Workers intermittently exposed to PCBs should have a baseline skin examination and liver function tests. For workers with signs and symptoms consistent with large exposures to PCBs (e.g., chloracne, elevated AST and ALT), obtain confirmation of exposure to determine serum PCB level.
Prevention at Home	**Home Practices** • Open all windows and use fans in your workspace when maintaining or repairing any products containing PCBs. • Wear a respirator or protective gloves, or both. • You and your children may be exposed to PCBs by eating fish or wildlife caught from contaminated locations. Certain states, Native American tribes, and U.S. territories have issued advisories to warn people about PCB-contaminated fish and fish-eating

	wildlife. You can reduce your family's exposure to PCBs by following these advisories.
Key Points	• The goal of treating chronically exposed patients is preventing any additional exposure to PCBs. • Exposure to PCBs at work or home is avoidable if the proper preventive measures are adopted.
Progress Check	13. Which of the following statements regarding treatment for chronic PCB toxicity is **NOT CORRECT**? A. The goal in treating chronically exposed patients is preventing any additional exposure to PCBs. B. No specific treatment is available for chronic PCB toxicity. C. Breastfeeding should be avoided. D. No known methods exist for reducing the burdens of PCBs in human tissues. *To review relevant content, see "Chronic Exposure" in this section.* 14. All of the following preventive measures to avoid PCB exposure at work or home are true **EXCEPT** A. Use special PCB-resistant gloves and protective clothing. B. Maintain adequate ventilation during spill cleanup or maintenance of vessels containing PCBs. If this is not possible, provide masks. C. Certain states, Native American tribes, and U.S. territories have issued advisories to warn people about PCB-contaminated fish and fish-eating wildlife. You can reduce your family's exposure to PCBs by obeying these advisories. D. Locations where PCBs are stored should be clearly posted as required by law. *To review relevant content, see "Prevention at Home" and "Prevention in the Workplace" in this section.*

What Instructions Should Be Given to Patients Exposed to PCBs?

Learning Objective	Upon completion of this section, you will be able to • Describe appropriate instructions for patients exposed to PCBs.
Introduction	All patients exposed to PCBs need basic guidance on • Self-care, so they can minimize further risks and avoid complications to the extent possible, and • Clinical follow-up, so they understand when and why to return for further medical attention. ATSDR has developed a patient education sheet on PCBs that you might find useful. It can be found at http: www.atsdr.cdc.gov csem pcb docs pcb patient education.pdf
Self-care Guidance for Patients	Patients should be advised to avoid exposures and conditions that might further increase their risk of disease or worsen their existing condition. **At Work** • Eliminate PCBs from the workplace, or implement engineering changes to isolate the PCBs. If neither of these approaches is feasible, use special PCB-resistant gloves and protective clothing. • Maintain adequate ventilation during spill cleanup or maintenance of vessels containing PCBs. If this is not possible, provide approved respirators. • Make provisions for proper decontamination or disposal of contaminated clothing or equipment. • Dispose of existing PCBs through appropriate toxic waste facilities. • Conduct environmental sampling as necessary to ensure adequate worker protection or safety for public reentry to contaminated areas. • Establish reentry or cleanup levels for dioxins and PCBs to protect workers who reoccupy buildings after a PCB fire.

	• Report persistent health effects (e.g., unexplained weight loss, muscle pain, frequent coughing, and sleep problems). These symptoms may be due to stress or recall bias and may not be specifically linked to the toxic effects of PCBs. **At Home** • Open all windows and use fans in your workspace when conducting maintenance or repairing any products containing PCBs. • If ventilation is poor, wear a respirator and protective gloves. • Seek medical attention immediately if an acute exposure occurs. • Lower exposure to PCBs by looking for and following health advisories issued by states, Native American tribes, or U.S. territories when eating fish or wildlife caught from locations contaminated with PCBs.
Clinical Follow- up Guidance for Patients	PCBs have been implicated as a potential cause of cancer in humans. Screening tests are available for breast cancer and melanoma. If patients believe that they are being exposed to PCBs, advise them how to stop the exposure. Also tell them how to contact worksite or environmental regulatory agencies that will assess exposure risks and prescribe protective actions. Advise patients with suspected or confirmed historic exposure to PCBs to be seen by you or their primary care provider periodically and monitored for signs of disease and changes in health status. Advise patients to consult their physicians if they develop signs or symptoms of PCB exposure such as • Appetite loss, • Joint pain, • Nausea, • Skin disorders, changes, or discoloration, • Breast changes or lumps, and or • Stomach distress and pain.

	ATSDR patient education sheet on PCBs includes a more detailed checklist that you can use to indicate which types of follow up are relevant for a given patient.
Key Points	• Advise patients to avoid PCB exposures and conditions that might further increase their risk of disease or worsen their existing condition. • Advise patients to contact their physicians if they develop skin problems or other health changes.
Progress Check	15. Patients who have been exposed to PCBs should be advised to A. Speak to their employers about reducing workplace exposures (if exposures are occupational). B. Learn how to avoid further exposure. C. Know when to call their doctors. D. All of the above. *To review relevant content, see all topics in this section.*

Sources of Additional Information

Polychlorinated Biphenyls (PCBs) Specific Information	Please refer to the following resources for more information on the adverse effects of PCBs, the treatment of PCB-associated diseases, and management of persons exposed to PCBs. • ATSDR http: www.atsdr.cdc.gov o For chemical emergency situations, contact ▪ CDC Emergency Response at 770-488-7100 and request the ATSDR Duty Officer o For chemical non-emergency situations, contact ▪ CDC-INFO at http: www.cdc.gov cdc-info ▪ 800-CDC-INFO at (800-232-4636) TTY 888-232-6348 - 24 Hours Day ▪ E-mail at: cdcinfo cdc.gov

	PLEASE NOTE ATSDR cannot respond to public inquiry and questions about individual medical cases, provide second opinions, or make specific recommendations regarding therapy. Such guidance requires clinical examination by a health care provider. o Toxicological Profile for Polychlorinated Biphenyls (PCBs) http: www.atsdr.cdc.gov toxprofiles tp .asp?id 142 tid 26 o Addendum to the Toxicological Profile for Polychlorinated Biphenyls (PCBs) http: www.atsdr.cdc.gov toxprofiles p cbs addendum.pdf o TOXFAQs for Polychlorinated Biphenyls (PCBs) (English) http: www.atsdr.cdc.gov toxfaqs tf. asp?id 140 tid 26 o TOXFAQs for Polychlorinated Biphenyls (PCBs) (Spanish) http: www.atsdr.cdc.gov es toxfaqs es tfacts17.html o ATSDR Minimal Response Levels http: www.atsdr.cdc.gov mrls index.ht ml • Centers for Disease Control and Prevention http: www.cdc.gov • EPA Polychlorinated Biphenyls (PCBs) https: www.epa.gov hw
General Environmental Health Information	Please refer to the following Web resources for general information on environmental health. • ATSDR http: www.atsdr.cdc.gov o Taking an Exposure History CSEM http: www.atsdr.cdc.gov csem csem.as p?csem 33 po 0 o View the complete library of CSEMs http: www.atsdr.cdc.gov csem csem.ht ml

- Exposure History Worksheet http://www.atsdr.cdc.gov/csem/exphistory/docs/CSEMExposHist-26-29.pdf
- ATSDR Regional Operations.
  - ATSDR regional staff are able to maintain current and historic knowledge of the sites and issues in their regions through the working relationships they have established with EPA, other federal and state agencies, individual citizens, and community groups.
  - A list of ATSDR's regional staff, the states and territories that they cover, and contact information can be found at http://www.atsdr.cdc.gov/DRO/dro_contact.html
- ATSDR State Cooperative Agreement Program http://www.atsdr.cdc.gov/states/index.html
  - The Cooperative Agreement Program provides essential support to communities nationwide to fulfill the mission of ATSDR.
  - The program funds 30 states and one tribal government to help develop and strengthen their abilities to evaluate and respond to environmental public health issues.

- CDC http://www.cdc.gov
  - CDC works to protect public health and safety by providing information to enhance health decisions, and promotes health through partnerships with state health departments and other organizations.
  - CDC focuses national attention on developing and applying activities surrounding disease prevention and control (especially infectious diseases), environmental health, occupational safety and health, health promotion, and education designed to improve the health of the people of the United States.

- National Center for Environmental Health (NCEH) http: www.cdc.gov nceh

  - NCEH works to prevent illness, disability, and death caused by interactions between people and the environment. NCEH is especially committed to safeguarding the health of populations that are particularly vulnerable to certain environmental hazards□children, the elderly, and people with disabilities.
  - NCEH seeks to achieve its mission through science, service, and leadership.

- National Institute of Health (NIH) http: www.nih.gov

  - A part of the U.S. Department of Health and Human Services, NIH is the primary federal agency for conducting and supporting medical research.

- National Institute for Occupational Safety and Health (NIOSH) http: www.cdc.gov niosh

  - NIOSH is part of the U.S. Department of Health and Human Services and is an agency established to help ensure safe and healthful working conditions for working men and women by providing research, information, education, and training in the field of occupational safety and health.

- American College of Occupational and Environmental Medicine (ACOEM) http: www.acoem.org

  - ACOEM is the nation's largest medical society dedicated to promoting the health of workers through preventive medicine, clinical care, research, and education.
  - ACOEM members are a dynamic group of physicians including specialists in a variety of medical practices. ACOEM is united to develop positions and policies on vital issues relevant to

preventive medicine both within and outside of the workplace.

- American College of Medical Toxicologists (ACMT) http: www.acmt.net

  - ACMT is a professional, nonprofit association of physicians with recognized expertise in medical toxicology.
  - ACMT is dedicated to advancing the science and practice of medical toxicology through a variety of activities.

- American College of Preventive Medicine (ACPM) http: www.acpm.org

  - ACPM is the national professional society for physicians committed to disease prevention and health promotion.
  - ACPM's 2,000 members are engaged in preventive medicine practice, teaching, and research.

- Association of Occupational and Environmental Clinics (AOEC) http: aoec.org

  - AOEC is a network of more than 60 clinics and more than 250 individuals committed to improving the practice of occupational and environmental medicine through information sharing and collaborative research.

- Pediatric Environmental Health Specialty Units (PEHSUs) http: www.pehsu.net

  - The PEHSUs are developed to provide education and consultation for health professionals, public health professionals and others about the topic of children's environmental health.
  - The PEHSU staff is available for consultation about potential pediatric environmental health concerns affecting both the child and the family.

Health care professionals may contact their regional PEHSU site for clinical advice.

- Poison Control Center
  - The American Association of Poison Control Centers (AAPC) may be contacted for questions about poisons and poisonings. Their Web site provides information about poison centers and poison prevention. AAPC does not provide information about treatment or diagnosis of poisoning, or research information for student papers.
  - American Association of Poison Control Centers may be contacted at 1-800-222-1222 or http: www.aapcc.org

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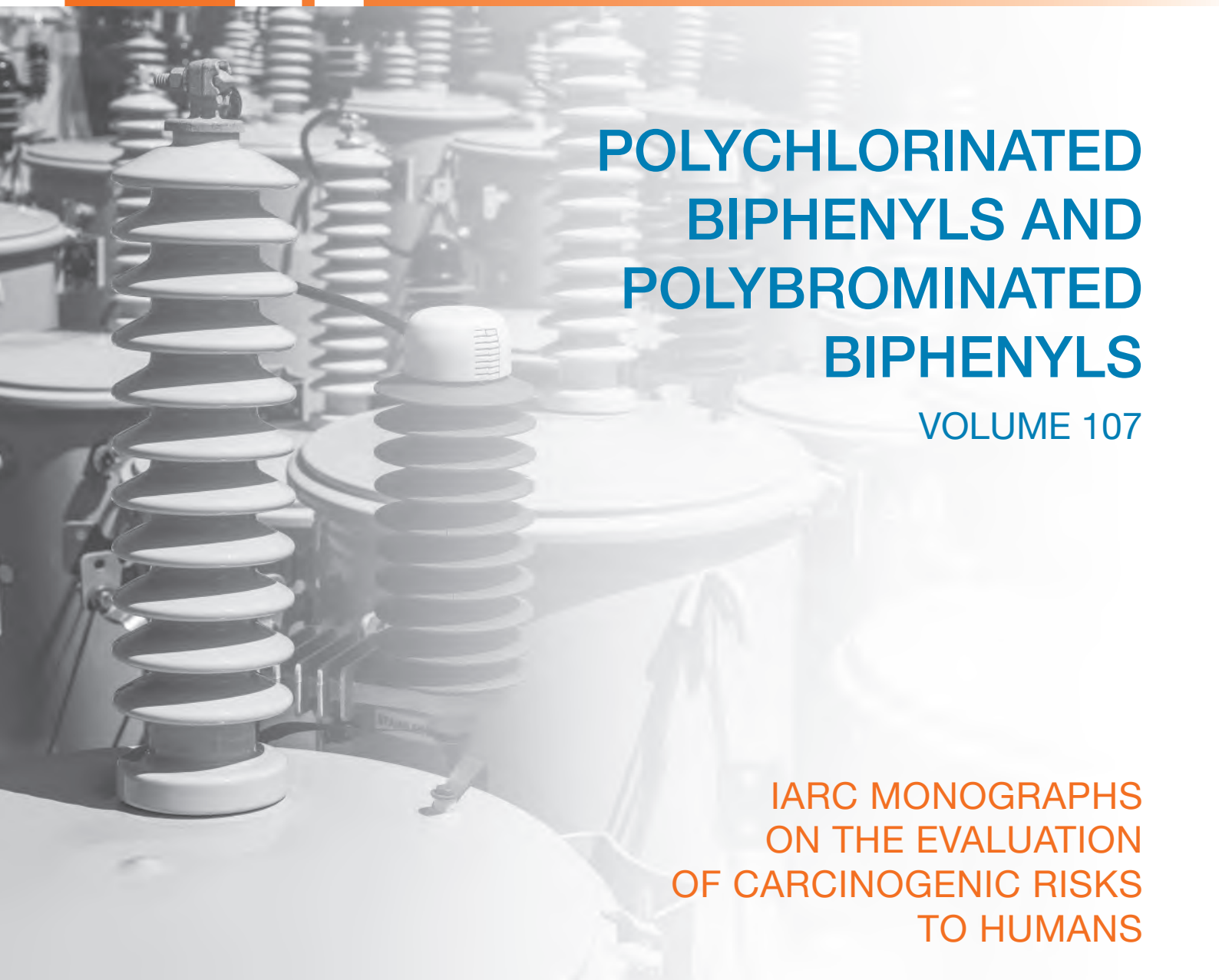
Attachment D

Polychlorinated Biphenyls and Polybrominated Biphenyls, World Health Organization International Agency for Research on Cancer

IARC MONOGRAPHS



POLYCHLORINATED BIPHENYLS AND POLYBROMINATED BIPHENYLS

VOLUME 107

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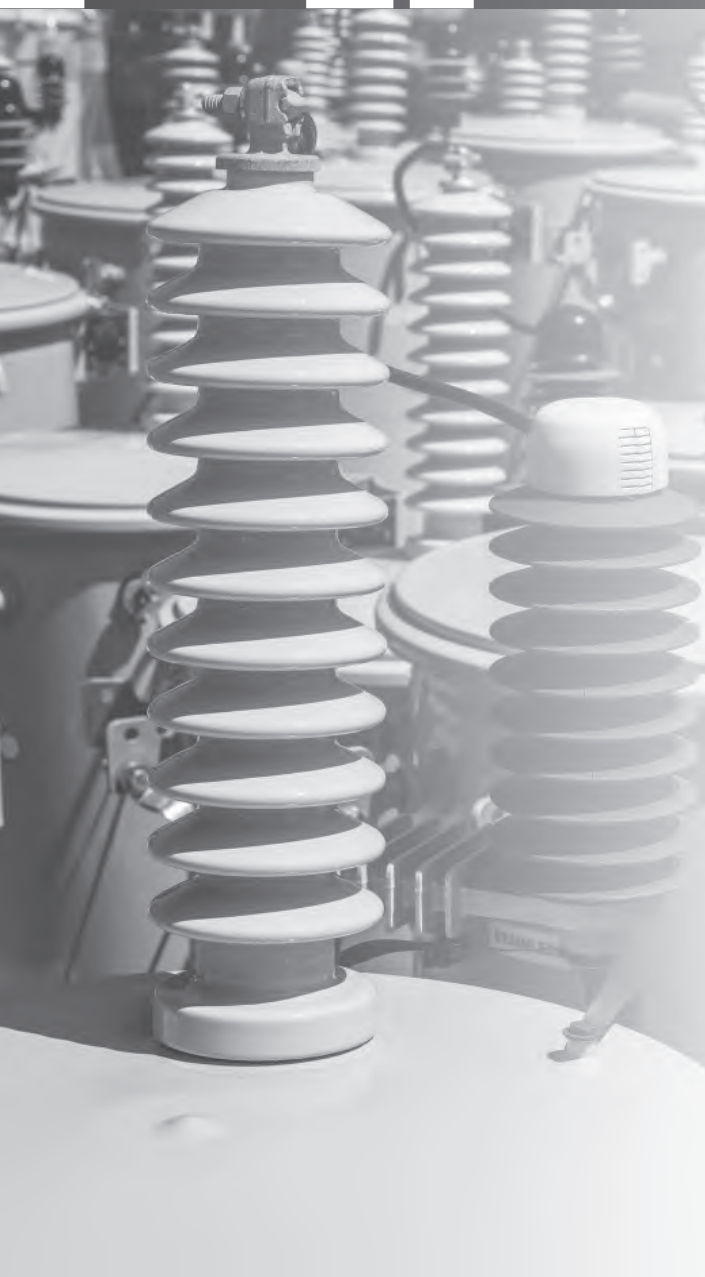
International Agency for Research on Cancer

World Health Organization

IARC MONOGRAPHS



POLYCHLORINATED BIPHENYLS AND POLYBROMINATED BIPHENYLS

VOLUME 107

This publication represents the views and expert opinions of an IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, which met in Lyon, 12–19 February 2013

Lyon, France - 2016

IARC MONOGRAPHS ON THE EVALUATION OF CARCINOGENIC RISKS TO HUMANS

International Agency for Research on Cancer

World Health Organization

IARC MONOGRAPHS

In 1969, the International Agency for Research on Cancer (IARC) initiated a programme on the evaluation of the carcinogenic risk of chemicals to humans involving the production of critically evaluated monographs on individual chemicals. The programme was subsequently expanded to include evaluations of carcinogenic risks associated with exposures to complex mixtures, lifestyle factors and biological and physical agents, as well as those in specific occupations. The objective of the programme is to elaborate and publish in the form of monographs critical reviews of data on carcinogenicity for agents to which humans are known to be exposed and on specific exposure situations; to evaluate these data in terms of human risk with the help of international working groups of experts in carcinogenesis and related fields; and to indicate where additional research efforts are needed. The lists of IARC evaluations are regularly updated and are available on the Internet at http://monographs.iarc.fr/.

This programme has been supported since 1982 by Cooperative Agreement U01 CA33193 with the United States National Cancer Institute, Department of Health and Human Services. Additional support has been provided since 1986 by the European Commission Directorate-General for Employment, Social Affairs, and Inclusion, initially by the Unit of Health, Safety and Hygiene at Work, and since 2014 by the European Union Programme for Employment and Social Innovation "EaSI" (2014–2020) (for further information please consult: http://ec.europa.eu/social/easi). Support has also been provided since 1992 by the United States National Institute of Environmental Health Sciences, Department of Health and Human Services. The contents of this volume are solely the responsibility of the Working Group and do not necessarily represent the official views of the United States National Cancer Institute, the United States National Institute of Environmental Health Sciences, the United States Department of Health and Human Services, or the European Commission.

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CONTENTS

NOTE TO THE READER

The term 'carcinogenic risk' in the *IARC Monographs* series is taken to mean that an agent is capable of causing cancer. The *Monographs* evaluate cancer hazards, despite the historical presence of the word 'risks' in the title.

Inclusion of an agent in the *Monographs* does not imply that it is a carcinogen, only that the published data have been examined. Equally, the fact that an agent has not yet been evaluated in a *Monograph* does not mean that it is not carcinogenic. Similarly, identification of cancer sites with *sufficient evidence* or *limited evidence* in humans should not be viewed as precluding the possibility that an agent may cause cancer at other sites.

The evaluations of carcinogenic risk are made by international working groups of independent scientists and are qualitative in nature. No recommendation is given for regulation or legislation.

Anyone who is aware of published data that may alter the evaluation of the carcinogenic risk of an agent to humans is encouraged to make this information available to the Section of IARC Monographs, International Agency for Research on Cancer, 150 cours Albert Thomas, 69372 Lyon Cedex 08, France, in order that the agent may be considered for re-evaluation by a future Working Group.

Although every effort is made to prepare the *Monographs* as accurately as possible, mistakes may occur. Readers are requested to communicate any errors to the Section of IARC Monographs, so that corrections can be reported in future volumes.

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Each participant was asked to disclose pertinent research, employment, and financial interests. Current financial interests and research and employment interests during the past 4 years or anticipated in the future are identified here. Minor pertinent interests are not listed and include stock valued at no more than US $1000 overall, grants that provide no more than 5% of the research budget of the expert's organization and that do not support the expert's research or position, and consulting or speaking on matters not before a court or government agency that does not exceed 2% of total professional time or compensation. All grants that support the expert's research or position and all consulting or speaking on behalf of an interested party on matters before a court or government agency are listed as significant pertinent interests.

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[3] Bernard W. Stewart attended as a Representative of Cancer Australia.
[4] Each Observer agreed to respect the Guidelines for Observers at IARC Monographs meetings. Observers did not serve as Meeting Chair or Subgroup Chair, draft any part of a Monograph, or participate in the evaluations. They also agreed not to contact participants before the meeting, not to lobby them at any time, not to send them written materials, and not to offer them meals or other favours. IARC asked and reminded Working Group Members to report any contact or attempt to influence that they may have encountered, either before or during the meeting.
[5] Erik Carlson is employed by General Electric Company as a toxicologist and works on PCB-related issues. He has provided expert opinion and testimony in the context of regulatory and legislative process and he holds stocks and bonds from stakeholders.
[6] John Schell has consulted for several companies on PCB-related issues and provided expert opinions on regulatory issues related to PCBs.

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PREAMBLE

The Preamble to the *IARC Monographs* describes the objective and scope of the programme, the scientific principles and procedures used in developing a *Monograph*, the types of evidence considered and the scientific criteria that guide the evaluations. The Preamble should be consulted when reading a *Monograph* or list of evaluations.

A. GENERAL PRINCIPLES AND PROCEDURES

1. Background

Soon after IARC was established in 1965, it received frequent requests for advice on the carcinogenic risk of chemicals, including requests for lists of known and suspected human carcinogens. It was clear that it would not be a simple task to summarize adequately the complexity of the information that was available, and IARC began to consider means of obtaining international expert opinion on this topic. In 1970, the IARC Advisory Committee on Environmental Carcinogenesis recommended '...that a compendium on carcinogenic chemicals be prepared by experts. The biological activity and evaluation of practical importance to public health should be referenced and documented.' The IARC Governing Council adopted a resolution concerning the role of IARC in providing government authorities with expert, independent, scientific opinion on environmental carcinogenesis. As one means to that end, the Governing Council recommended that IARC should prepare monographs on the evaluation of carcinogenic risk of chemicals to man, which became the initial title of the series.

In the succeeding years, the scope of the programme broadened as *Monographs* were developed for groups of related chemicals, complex mixtures, occupational exposures, physical and biological agents and lifestyle factors. In 1988, the phrase 'of chemicals' was dropped from the title, which assumed its present form, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*.

Through the *Monographs* programme, IARC seeks to identify the causes of human cancer. This is the first step in cancer prevention, which is needed as much today as when IARC was established. The global burden of cancer is high and continues to increase: the annual number of new cases was estimated at 10.1 million in 2000 and is expected to reach 15 million by 2020 (Stewart & Kleihues, 2003). With current trends in demographics and exposure, the cancer burden has been shifting from high-resource countries to low- and medium-resource countries. As a result of *Monographs* evaluations, national health agencies have been able, on scientific grounds, to take measures to reduce human exposure to carcinogens in the workplace and in the environment.

The criteria established in 1971 to evaluate carcinogenic risks to humans were adopted by the Working Groups whose deliberations resulted in the first 16 volumes of the *Monographs* series. Those criteria were subsequently updated by further ad hoc Advisory Groups (IARC, 1977, 1978, 1979, 1982, 1983, 1987, 1988, 1991; Vainio *et al.*, 1992; IARC, 2005, 2006).

The Preamble is primarily a statement of scientific principles, rather than a specification of working procedures. The procedures through which a Working Group implements these principles are not specified in detail. They usually involve operations that have been established as being effective during previous *Monograph* meetings but remain, predominantly, the prerogative of each individual Working Group.

2. Objective and scope

The objective of the programme is to prepare, with the help of international Working Groups of experts, and to publish in the form of *Monographs*, critical reviews and evaluations of evidence on the carcinogenicity of a wide range of human exposures. The *Monographs* represent the first step in carcinogen risk assessment, which involves examination of all relevant information to assess the strength of the available evidence that an agent could alter the age-specific incidence of cancer in humans. The *Monographs* may also indicate where additional research efforts are needed, specifically when data immediately relevant to an evaluation are not available.

In this Preamble, the term 'agent' refers to any entity or circumstance that is subject to evaluation in a *Monograph*. As the scope of the programme has broadened, categories of agents now include specific chemicals, groups of related chemicals, complex mixtures, occupational or environmental exposures, cultural or behavioural practices, biological organisms and physical agents. This list of categories may expand as causation of, and susceptibility to, malignant disease become more fully understood.

A cancer 'hazard' is an agent that is capable of causing cancer under some circumstances, while a cancer 'risk' is an estimate of the carcinogenic effects expected from exposure to a cancer hazard. The *Monographs* are an exercise in evaluating cancer hazards, despite the historical presence of the word 'risks' in the title. The distinction between hazard and risk is important, and the *Monographs* identify cancer hazards even when risks are very low at current exposure levels, because new uses or unforeseen exposures could engender risks that are significantly higher.

In the *Monographs*, an agent is termed 'carcinogenic' if it is capable of increasing the incidence of malignant neoplasms, reducing their latency, or increasing their severity or multiplicity. The induction of benign neoplasms may in some circumstances (see Part B, Section 3a) contribute to the judgement that the agent is carcinogenic. The terms 'neoplasm' and 'tumour' are used interchangeably.

The Preamble continues the previous usage of the phrase 'strength of evidence' as a matter of historical continuity, although it should be understood that *Monographs* evaluations consider studies that support a finding of a cancer hazard as well as studies that do not.

Some epidemiological and experimental studies indicate that different agents may act at different stages in the carcinogenic process, and several different mechanisms may be involved. The aim of the *Monographs* has been, from their inception, to evaluate evidence of carcinogenicity at any stage in the carcinogenesis process, independently of the underlying mechanisms. Information on mechanisms may, however, be used in making the overall evaluation (IARC, 1991; Vainio *et al.*, 1992; IARC, 2005, 2006; see also Part B, Sections 4 and 6). As mechanisms of carcinogenesis are elucidated, IARC convenes international scientific conferences to determine whether a broad-based consensus has emerged

on how specific mechanistic data can be used in an evaluation of human carcinogenicity. The results of such conferences are reported in IARC Scientific Publications, which, as long as they still reflect the current state of scientific knowledge, may guide subsequent Working Groups.

Although the *Monographs* have emphasized hazard identification, important issues may also involve dose–response assessment. In many cases, the same epidemiological and experimental studies used to evaluate a cancer hazard can also be used to estimate a dose–response relationship. A *Monograph* may undertake to estimate dose–response relationships within the range of the available epidemiological data, or it may compare the dose–response information from experimental and epidemiological studies. In some cases, a subsequent publication may be prepared by a separate Working Group with expertise in quantitative dose–response assessment.

The *Monographs* are used by national and international authorities to make risk assessments, formulate decisions concerning preventive measures, provide effective cancer control programmes and decide among alternative options for public health decisions. The evaluations of IARC Working Groups are scientific, qualitative judgements on the evidence for or against carcinogenicity provided by the available data. These evaluations represent only one part of the body of information on which public health decisions may be based. Public health options vary from one situation to another and from country to country and relate to many factors, including different socioeconomic and national priorities. Therefore, no recommendation is given with regard to regulation or legislation, which are the responsibility of individual governments or other international organizations.

3. Selection of agents for review

Agents are selected for review on the basis of two main criteria: (a) there is evidence of human exposure and (b) there is some evidence or suspicion of carcinogenicity. Mixed exposures may occur in occupational and environmental settings and as a result of individual and cultural habits (such as tobacco smoking and dietary practices). Chemical analogues and compounds with biological or physical characteristics similar to those of suspected carcinogens may also be considered, even in the absence of data on a possible carcinogenic effect in humans or experimental animals.

The scientific literature is surveyed for published data relevant to an assessment of carcinogenicity. Ad hoc Advisory Groups convened by IARC in 1984, 1989, 1991, 1993, 1998 and 2003 made recommendations as to which agents should be evaluated in the *Monographs* series. Recent recommendations are available on the *Monographs* programme web site (http://monographs.iarc.fr). IARC may schedule other agents for review as it becomes aware of new scientific information or as national health agencies identify an urgent public health need related to cancer.

As significant new data become available on an agent for which a *Monograph* exists, a re-evaluation may be made at a subsequent meeting, and a new *Monograph* published. In some cases it may be appropriate to review only the data published since a prior evaluation. This can be useful for updating a database, reviewing new data to resolve a previously open question or identifying new tumour sites associated with a carcinogenic agent. Major changes in an evaluation (e.g. a new classification in Group 1 or a determination that a mechanism does not operate in humans, see Part B, Section 6) are more appropriately addressed by a full review.

4. Data for the *Monographs*

Each *Monograph* reviews all pertinent epidemiological studies and cancer bioassays in experimental animals. Those judged inadequate

or irrelevant to the evaluation may be cited but not summarized. If a group of similar studies is not reviewed, the reasons are indicated.

Mechanistic and other relevant data are also reviewed. A *Monograph* does not necessarily cite all the mechanistic literature concerning the agent being evaluated (see Part B, Section 4). Only those data considered by the Working Group to be relevant to making the evaluation are included.

With regard to epidemiological studies, cancer bioassays, and mechanistic and other relevant data, only reports that have been published or accepted for publication in the openly available scientific literature are reviewed. The same publication requirement applies to studies originating from IARC, including meta-analyses or pooled analyses commissioned by IARC in advance of a meeting (see Part B, Section 2c). Data from government agency reports that are publicly available are also considered. Exceptionally, doctoral theses and other material that are in their final form and publicly available may be reviewed.

Exposure data and other information on an agent under consideration are also reviewed. In the sections on chemical and physical properties, on analysis, on production and use and on occurrence, published and unpublished sources of information may be considered.

Inclusion of a study does not imply acceptance of the adequacy of the study design or of the analysis and interpretation of the results, and limitations are clearly outlined in square brackets at the end of each study description (see Part B). The reasons for not giving further consideration to an individual study also are indicated in the square brackets.

5. Meeting participants

Five categories of participant can be present at *Monograph* meetings.

(a) The Working Group

The Working Group is responsible for the critical reviews and evaluations that are developed during the meeting. The tasks of Working Group Members are: (i) to ascertain that all appropriate data have been collected; (ii) to select the data relevant for the evaluation on the basis of scientific merit; (iii) to prepare accurate summaries of the data to enable the reader to follow the reasoning of the Working Group; (iv) to evaluate the results of epidemiological and experimental studies on cancer; (v) to evaluate data relevant to the understanding of mechanisms of carcinogenesis; and (vi) to make an overall evaluation of the carcinogenicity of the exposure to humans. Working Group Members generally have published significant research related to the carcinogenicity of the agents being reviewed, and IARC uses literature searches to identify most experts. Working Group Members are selected on the basis of (a) knowledge and experience and (b) absence of real or apparent conflicts of interests. Consideration is also given to demographic diversity and balance of scientific findings and views.

(b) Invited Specialists

Invited Specialists are experts who also have critical knowledge and experience but have a real or apparent conflict of interests. These experts are invited when necessary to assist in the Working Group by contributing their unique knowledge and experience during subgroup and plenary discussions. They may also contribute text on non-influential issues in the section on exposure, such as a general description of data on production and use (see Part B, Section 1). Invited Specialists do not serve as meeting chair or subgroup chair, draft text that pertains to the description or interpretation of cancer data, or participate in the evaluations.

(c) Representatives of national and international health agencies

Representatives of national and international health agencies often attend meetings because their agencies sponsor the programme or are interested in the subject of a meeting. Representatives do not serve as meeting chair or subgroup chair, draft any part of a *Monograph*, or participate in the evaluations.

(d) Observers with relevant scientific credentials

Observers with relevant scientific credentials may be admitted to a meeting by IARC in limited numbers. Attention will be given to achieving a balance of Observers from constituencies with differing perspectives. They are invited to observe the meeting and should not attempt to influence it. Observers do not serve as meeting chair or subgroup chair, draft any part of a *Monograph*, or participate in the evaluations. At the meeting, the meeting chair and subgroup chairs may grant Observers an opportunity to speak, generally after they have observed a discussion. Observers agree to respect the Guidelines for Observers at *IARC Monographs* meetings (available at http://monographs.iarc.fr).

(e) The IARC Secretariat

The IARC Secretariat consists of scientists who are designated by IARC and who have relevant expertise. They serve as rapporteurs and participate in all discussions. When requested by the meeting chair or subgroup chair, they may also draft text or prepare tables and analyses.

Before an invitation is extended, each potential participant, including the IARC Secretariat, completes the WHO Declaration of Interests to report financial interests, employment and consulting, and individual and institutional research support related to the subject of the meeting. IARC assesses these interests to determine whether there is a conflict that warrants some limitation on participation. The declarations are updated and reviewed again at the opening of the meeting. Interests related to the subject of the meeting are disclosed to the meeting participants and in the published volume (Cogliano *et al.*, 2004).

The names and principal affiliations of participants are available on the *Monographs* programme web site (http://monographs.iarc.fr) approximately two months before each meeting. It is not acceptable for Observers or third parties to contact other participants before a meeting or to lobby them at any time. Meeting participants are asked to report all such contacts to IARC (Cogliano *et al.*, 2005).

All participants are listed, with their principal affiliations, at the beginning of each volume. Each participant who is a Member of a Working Group serves as an individual scientist and not as a representative of any organization, government or industry.

6. Working procedures

A separate Working Group is responsible for developing each volume of *Monographs*. A volume contains one or more *Monographs*, which can cover either a single agent or several related agents. Approximately one year in advance of the meeting of a Working Group, the agents to be reviewed are announced on the *Monographs* programme web site (http://monographs.iarc.fr) and participants are selected by IARC staff in consultation with other experts. Subsequently, relevant biological and epidemiological data are collected by IARC from recognized sources of information on carcinogenesis, including data storage and retrieval systems such as PubMed. Meeting participants who are asked to prepare preliminary working papers for specific sections are expected to supplement the IARC literature searches with their own searches.

Industrial associations, labour unions and other knowledgeable organizations may be asked to provide input to the sections on production and use, although this involvement is not required as a general rule. Information on production and trade is obtained from governmental, trade and market research publications and, in some cases, by direct contact with industries. Separate production data on some agents may not be available for a variety of reasons (e.g. not collected or made public in all producing countries, production is small). Information on uses may be obtained from published sources but is often complemented by direct contact with manufacturers. Efforts are made to supplement this information with data from other national and international sources.

Six months before the meeting, the material obtained is sent to meeting participants to prepare preliminary working papers. The working papers are compiled by IARC staff and sent, before the meeting, to Working Group Members and Invited Specialists for review.

The Working Group meets at IARC for seven to eight days to discuss and finalize the texts and to formulate the evaluations. The objectives of the meeting are peer review and consensus. During the first few days, four subgroups (covering exposure data, cancer in humans, cancer in experimental animals, and mechanistic and other relevant data) review the working papers, develop a joint subgroup draft and write summaries. Care is taken to ensure that each study summary is written or reviewed by someone not associated with the study being considered. During the last few days, the Working Group meets in plenary session to review the subgroup drafts and develop the evaluations. As a result, the entire volume is the joint product of the Working Group, and there are no individually authored sections.

IARC Working Groups strive to achieve a consensus evaluation. Consensus reflects broad agreement among Working Group Members, but not necessarily unanimity. The chair may elect to poll Working Group Members to determine the diversity of scientific opinion on issues where consensus is not readily apparent.

After the meeting, the master copy is verified by consulting the original literature, edited and prepared for publication. The aim is to publish the volume within six months of the Working Group meeting. A summary of the outcome is available on the *Monographs* programme web site soon after the meeting.

B. SCIENTIFIC REVIEW AND EVALUATION

The available studies are summarized by the Working Group, with particular regard to the qualitative aspects discussed below. In general, numerical findings are indicated as they appear in the original report; units are converted when necessary for easier comparison. The Working Group may conduct additional analyses of the published data and use them in their assessment of the evidence; the results of such supplementary analyses are given in square brackets. When an important aspect of a study that directly impinges on its interpretation should be brought to the attention of the reader, a Working Group comment is given in square brackets.

The scope of the *IARC Monographs* programme has expanded beyond chemicals to include complex mixtures, occupational exposures, physical and biological agents, lifestyle factors and other potentially carcinogenic exposures. Over time, the structure of a *Monograph* has evolved to include the following sections:

Exposure data
Studies of cancer in humans
Studies of cancer in experimental animals
Mechanistic and other relevant data
Summary
Evaluation and rationale

In addition, a section of General Remarks at the front of the volume discusses the reasons the agents were scheduled for evaluation and some key issues the Working Group encountered during the meeting.

This part of the Preamble discusses the types of evidence considered and summarized in each section of a *Monograph*, followed by the scientific criteria that guide the evaluations.

1. Exposure data

Each *Monograph* includes general information on the agent: this information may vary substantially between agents and must be adapted accordingly. Also included is information on production and use (when appropriate), methods of analysis and detection, occurrence, and sources and routes of human occupational and environmental exposures. Depending on the agent, regulations and guidelines for use may be presented.

(a) General information on the agent

For chemical agents, sections on chemical and physical data are included: the Chemical Abstracts Service Registry Number, the latest primary name and the IUPAC systematic name are recorded; other synonyms are given, but the list is not necessarily comprehensive. Information on chemical and physical properties that are relevant to identification, occurrence and biological activity is included. A description of technical products of chemicals includes trade names, relevant specifications and available information on composition and impurities. Some of the trade names given may be those of mixtures in which the agent being evaluated is only one of the ingredients.

For biological agents, taxonomy, structure and biology are described, and the degree of variability is indicated. Mode of replication, life cycle, target cells, persistence, latency, host response and clinical disease other than cancer are also presented.

For physical agents that are forms of radiation, energy and range of the radiation are included. For foreign bodies, fibres and respirable particles, size range and relative dimensions are indicated.

For agents such as mixtures, drugs or lifestyle factors, a description of the agent, including its composition, is given.

Whenever appropriate, other information, such as historical perspectives or the description of an industry or habit, may be included.

(b) Analysis and detection

An overview of methods of analysis and detection of the agent is presented, including their sensitivity, specificity and reproducibility. Methods widely used for regulatory purposes are emphasized. Methods for monitoring human exposure are also given. No critical evaluation or recommendation of any method is meant or implied.

(c) Production and use

The dates of first synthesis and of first commercial production of a chemical, mixture or other agent are provided when available; for agents that do not occur naturally, this information may allow a reasonable estimate to be made of the date before which no human exposure to the agent could have occurred. The dates of first reported occurrence of an exposure are also provided when available. In addition, methods of synthesis used in past and present commercial production and different methods of production, which may give rise to different impurities, are described.

The countries where companies report production of the agent, and the number of companies in each country, are identified. Available data on production, international trade and uses are

obtained for representative regions. It should not, however, be inferred that those areas or nations are necessarily the sole or major sources or users of the agent. Some identified uses may not be current or major applications, and the coverage is not necessarily comprehensive. In the case of drugs, mention of their therapeutic uses does not necessarily represent current practice nor does it imply judgement as to their therapeutic efficacy.

(d) Occurrence and exposure

Information on the occurrence of an agent in the environment is obtained from data derived from the monitoring and surveillance of levels in occupational environments, air, water, soil, plants, foods and animal and human tissues. When available, data on the generation, persistence and bioaccumulation of the agent are also included. Such data may be available from national databases.

Data that indicate the extent of past and present human exposure, the sources of exposure, the people most likely to be exposed and the factors that contribute to the exposure are reported. Information is presented on the range of human exposure, including occupational and environmental exposures. This includes relevant findings from both developed and developing countries. Some of these data are not distributed widely and may be available from government reports and other sources. In the case of mixtures, industries, occupations or processes, information is given about all agents known to be present. For processes, industries and occupations, a historical description is also given, noting variations in chemical composition, physical properties and levels of occupational exposure with date and place. For biological agents, the epidemiology of infection is described.

(e) Regulations and guidelines

Statements concerning regulations and guidelines (e.g. occupational exposure limits, maximal levels permitted in foods and water, pesticide registrations) are included, but they may not reflect the most recent situation, since such limits are continuously reviewed and modified. The absence of information on regulatory status for a country should not be taken to imply that that country does not have regulations with regard to the exposure. For biological agents, legislation and control, including vaccination and therapy, are described.

2. Studies of cancer in humans

This section includes all pertinent epidemiological studies (see Part A, Section 4). Studies of biomarkers are included when they are relevant to an evaluation of carcinogenicity to humans.

(a) Types of study considered

Several types of epidemiological study contribute to the assessment of carcinogenicity in humans — cohort studies, case–control studies, correlation (or ecological) studies and intervention studies. Rarely, results from randomized trials may be available. Case reports and case series of cancer in humans may also be reviewed.

Cohort and case–control studies relate individual exposures under study to the occurrence of cancer in individuals and provide an estimate of effect (such as relative risk) as the main measure of association. Intervention studies may provide strong evidence for making causal inferences, as exemplified by cessation of smoking and the subsequent decrease in risk for lung cancer.

In correlation studies, the units of investigation are usually whole populations (e.g. in particular geographical areas or at particular times), and cancer frequency is related to a summary measure of the exposure of the population

to the agent under study. In correlation studies, individual exposure is not documented, which renders this kind of study more prone to confounding. In some circumstances, however, correlation studies may be more informative than analytical study designs (see, for example, the *Monograph* on arsenic in drinking-water; IARC, 2004).

In some instances, case reports and case series have provided important information about the carcinogenicity of an agent. These types of study generally arise from a suspicion, based on clinical experience, that the concurrence of two events — that is, a particular exposure and occurrence of a cancer — has happened rather more frequently than would be expected by chance. Case reports and case series usually lack complete ascertainment of cases in any population, definition or enumeration of the population at risk and estimation of the expected number of cases in the absence of exposure.

The uncertainties that surround the interpretation of case reports, case series and correlation studies make them inadequate, except in rare instances, to form the sole basis for inferring a causal relationship. When taken together with case–control and cohort studies, however, these types of study may add materially to the judgement that a causal relationship exists.

Epidemiological studies of benign neoplasms, presumed preneoplastic lesions and other end-points thought to be relevant to cancer are also reviewed. They may, in some instances, strengthen inferences drawn from studies of cancer itself.

(b) Quality of studies considered

It is necessary to take into account the possible roles of bias, confounding and chance in the interpretation of epidemiological studies. Bias is the effect of factors in study design or execution that lead erroneously to a stronger or weaker association than in fact exists between an agent and disease. Confounding is a form of bias that occurs when the relationship with disease is made to appear stronger or weaker than it truly is as a result of an association between the apparent causal factor and another factor that is associated with either an increase or decrease in the incidence of the disease. The role of chance is related to biological variability and the influence of sample size on the precision of estimates of effect.

In evaluating the extent to which these factors have been minimized in an individual study, consideration is given to several aspects of design and analysis as described in the report of the study. For example, when suspicion of carcinogenicity arises largely from a single small study, careful consideration is given when interpreting subsequent studies that included these data in an enlarged population. Most of these considerations apply equally to case–control, cohort and correlation studies. Lack of clarity of any of these aspects in the reporting of a study can decrease its credibility and the weight given to it in the final evaluation of the exposure.

First, the study population, disease (or diseases) and exposure should have been well defined by the authors. Cases of disease in the study population should have been identified in a way that was independent of the exposure of interest, and exposure should have been assessed in a way that was not related to disease status.

Second, the authors should have taken into account — in the study design and analysis — other variables that can influence the risk of disease and may have been related to the exposure of interest. Potential confounding by such variables should have been dealt with either in the design of the study, such as by matching, or in the analysis, by statistical adjustment. In cohort studies, comparisons with local rates of disease may or may not be more appropriate than those with national rates. Internal comparisons of frequency of disease among individuals at different levels of exposure are also desirable in cohort studies, since they minimize the potential for

confounding related to the difference in risk factors between an external reference group and the study population.

Third, the authors should have reported the basic data on which the conclusions are founded, even if sophisticated statistical analyses were employed. At the very least, they should have given the numbers of exposed and unexposed cases and controls in a case–control study and the numbers of cases observed and expected in a cohort study. Further tabulations by time since exposure began and other temporal factors are also important. In a cohort study, data on all cancer sites and all causes of death should have been given, to reveal the possibility of reporting bias. In a case–control study, the effects of investigated factors other than the exposure of interest should have been reported.

Finally, the statistical methods used to obtain estimates of relative risk, absolute rates of cancer, confidence intervals and significance tests, and to adjust for confounding should have been clearly stated by the authors. These methods have been reviewed for case–control studies (Breslow & Day, 1980) and for cohort studies (Breslow & Day, 1987).

(c) Meta-analyses and pooled analyses

Independent epidemiological studies of the same agent may lead to results that are difficult to interpret. Combined analyses of data from multiple studies are a means of resolving this ambiguity, and well conducted analyses can be considered. There are two types of combined analysis. The first involves combining summary statistics such as relative risks from individual studies (meta-analysis) and the second involves a pooled analysis of the raw data from the individual studies (pooled analysis) (Greenland, 1998).

The advantages of combined analyses are increased precision due to increased sample size and the opportunity to explore potential confounders, interactions and modifying effects that may explain heterogeneity among studies in more detail. A disadvantage of combined analyses is the possible lack of compatibility of data from various studies due to differences in subject recruitment, procedures of data collection, methods of measurement and effects of unmeasured co-variates that may differ among studies. Despite these limitations, well conducted combined analyses may provide a firmer basis than individual studies for drawing conclusions about the potential carcinogenicity of agents.

IARC may commission a meta-analysis or pooled analysis that is pertinent to a particular *Monograph* (see Part A, Section 4). Additionally, as a means of gaining insight from the results of multiple individual studies, ad hoc calculations that combine data from different studies may be conducted by the Working Group during the course of a *Monograph* meeting. The results of such original calculations, which would be specified in the text by presentation in square brackets, might involve updates of previously conducted analyses that incorporate the results of more recent studies or de-novo analyses. Irrespective of the source of data for the meta-analyses and pooled analyses, it is important that the same criteria for data quality be applied as those that would be applied to individual studies and to ensure also that sources of heterogeneity between studies be taken into account.

(d) Temporal effects

Detailed analyses of both relative and absolute risks in relation to temporal variables, such as age at first exposure, time since first exposure, duration of exposure, cumulative exposure, peak exposure (when appropriate) and time since cessation of exposure, are reviewed and summarized when available. Analyses of temporal relationships may be useful in making causal inferences. In addition, such analyses may suggest whether a carcinogen acts early or late in the process of carcinogenesis, although, at best, they

allow only indirect inferences about mechanisms of carcinogenesis.

(e) Use of biomarkers in epidemiological studies

Biomarkers indicate molecular, cellular or other biological changes and are increasingly used in epidemiological studies for various purposes (IARC, 1991; Vainio *et al.*, 1992; Toniolo *et al.*, 1997; Vineis *et al.*, 1999; Buffler *et al.*, 2004). These may include evidence of exposure, of early effects, of cellular, tissue or organism responses, of individual susceptibility or host responses, and inference of a mechanism (see Part B, Section 4b). This is a rapidly evolving field that encompasses developments in genomics, epigenomics and other emerging technologies.

Molecular epidemiological data that identify associations between genetic polymorphisms and interindividual differences in susceptibility to the agent(s) being evaluated may contribute to the identification of carcinogenic hazards to humans. If the polymorphism has been demonstrated experimentally to modify the functional activity of the gene product in a manner that is consistent with increased susceptibility, these data may be useful in making causal inferences. Similarly, molecular epidemiological studies that measure cell functions, enzymes or metabolites that are thought to be the basis of susceptibility may provide evidence that reinforces biological plausibility. It should be noted, however, that when data on genetic susceptibility originate from multiple comparisons that arise from subgroup analyses, this can generate false-positive results and inconsistencies across studies, and such data therefore require careful evaluation. If the known phenotype of a genetic polymorphism can explain the carcinogenic mechanism of the agent being evaluated, data on this phenotype may be useful in making causal inferences.

(f) Criteria for causality

After the quality of individual epidemiological studies of cancer has been summarized and assessed, a judgement is made concerning the strength of evidence that the agent in question is carcinogenic to humans. In making its judgement, the Working Group considers several criteria for causality (Hill, 1965). A strong association (e.g. a large relative risk) is more likely to indicate causality than a weak association, although it is recognized that estimates of effect of small magnitude do not imply lack of causality and may be important if the disease or exposure is common. Associations that are replicated in several studies of the same design or that use different epidemiological approaches or under different circumstances of exposure are more likely to represent a causal relationship than isolated observations from single studies. If there are inconsistent results among investigations, possible reasons are sought (such as differences in exposure), and results of studies that are judged to be of high quality are given more weight than those of studies that are judged to be methodologically less sound.

If the risk increases with the exposure, this is considered to be a strong indication of causality, although the absence of a graded response is not necessarily evidence against a causal relationship. The demonstration of a decline in risk after cessation of or reduction in exposure in individuals or in whole populations also supports a causal interpretation of the findings.

Several scenarios may increase confidence in a causal relationship. On the one hand, an agent may be specific in causing tumours at one site or of one morphological type. On the other, carcinogenicity may be evident through the causation of multiple tumour types. Temporality, precision of estimates of effect, biological plausibility and coherence of the overall database are considered. Data on biomarkers may be employed in

an assessment of the biological plausibility of epidemiological observations.

Although rarely available, results from randomized trials that show different rates of cancer among exposed and unexposed individuals provide particularly strong evidence for causality.

When several epidemiological studies show little or no indication of an association between an exposure and cancer, a judgement may be made that, in the aggregate, they show evidence of lack of carcinogenicity. Such a judgement requires first that the studies meet, to a sufficient degree, the standards of design and analysis described above. Specifically, the possibility that bias, confounding or misclassification of exposure or outcome could explain the observed results should be considered and excluded with reasonable certainty. In addition, all studies that are judged to be methodologically sound should (a) be consistent with an estimate of effect of unity for any observed level of exposure, (b) when considered together, provide a pooled estimate of relative risk that is at or near to unity, and (c) have a narrow confidence interval, due to sufficient population size. Moreover, no individual study nor the pooled results of all the studies should show any consistent tendency that the relative risk of cancer increases with increasing level of exposure. It is important to note that evidence of lack of carcinogenicity obtained from several epidemiological studies can apply only to the type(s) of cancer studied, to the dose levels reported, and to the intervals between first exposure and disease onset observed in these studies. Experience with human cancer indicates that the period from first exposure to the development of clinical cancer is sometimes longer than 20 years; latent periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity.

3. Studies of cancer in experimental animals

All known human carcinogens that have been studied adequately for carcinogenicity in experimental animals have produced positive results in one or more animal species (Wilbourn *et al.*, 1986; Tomatis *et al.*, 1989). For several agents (e.g. aflatoxins, diethylstilbestrol, solar radiation, vinyl chloride), carcinogenicity in experimental animals was established or highly suspected before epidemiological studies confirmed their carcinogenicity in humans (Vainio *et al.*, 1995). Although this association cannot establish that all agents that cause cancer in experimental animals also cause cancer in humans, it is biologically plausible that agents for which there is *sufficient evidence of carcinogenicity* in experimental animals (see Part B, Section 6b) also present a carcinogenic hazard to humans. Accordingly, in the absence of additional scientific information, these agents are considered to pose a carcinogenic hazard to humans. Examples of additional scientific information are data that demonstrate that a given agent causes cancer in animals through a species-specific mechanism that does not operate in humans or data that demonstrate that the mechanism in experimental animals also operates in humans (see Part B, Section 6).

Consideration is given to all available long-term studies of cancer in experimental animals with the agent under review (see Part A, Section 4). In all experimental settings, the nature and extent of impurities or contaminants present in the agent being evaluated are given when available. Animal species, strain (including genetic background where applicable), sex, numbers per group, age at start of treatment, route of exposure, dose levels, duration of exposure, survival and information on tumours (incidence, latency, severity or multiplicity of neoplasms or preneoplastic lesions) are reported. Those studies in experimental animals that are judged to be irrelevant to the evaluation or judged to be inadequate

(e.g. too short a duration, too few animals, poor survival; see below) may be omitted. Guidelines for conducting long-term carcinogenicity experiments have been published (e.g. OECD, 2002).

Other studies considered may include: experiments in which the agent was administered in the presence of factors that modify carcinogenic effects (e.g. initiation–promotion studies, co-carcinogenicity studies and studies in genetically modified animals); studies in which the end-point was not cancer but a defined precancerous lesion; experiments on the carcinogenicity of known metabolites and derivatives; and studies of cancer in non-laboratory animals (e.g. livestock and companion animals) exposed to the agent.

For studies of mixtures, consideration is given to the possibility that changes in the physicochemical properties of the individual substances may occur during collection, storage, extraction, concentration and delivery. Another consideration is that chemical and toxicological interactions of components in a mixture may alter dose–response relationships. The relevance to human exposure of the test mixture administered in the animal experiment is also assessed. This may involve consideration of the following aspects of the mixture tested: (i) physical and chemical characteristics, (ii) identified constituents that may indicate the presence of a class of substances and (iii) the results of genetic toxicity and related tests.

The relevance of results obtained with an agent that is analogous (e.g. similar in structure or of a similar virus genus) to that being evaluated is also considered. Such results may provide biological and mechanistic information that is relevant to the understanding of the process of carcinogenesis in humans and may strengthen the biological plausibility that the agent being evaluated is carcinogenic to humans (see Part B, Section 2f).

(a) Qualitative aspects

An assessment of carcinogenicity involves several considerations of qualitative importance, including (i) the experimental conditions under which the test was performed, including route, schedule and duration of exposure, species, strain (including genetic background where applicable), sex, age and duration of follow-up; (ii) the consistency of the results, for example, across species and target organ(s); (iii) the spectrum of neoplastic response, from preneoplastic lesions and benign tumours to malignant neoplasms; and (iv) the possible role of modifying factors.

Considerations of importance in the interpretation and evaluation of a particular study include: (i) how clearly the agent was defined and, in the case of mixtures, how adequately the sample characterization was reported; (ii) whether the dose was monitored adequately, particularly in inhalation experiments; (iii) whether the doses, duration of treatment and route of exposure were appropriate; (iv) whether the survival of treated animals was similar to that of controls; (v) whether there were adequate numbers of animals per group; (vi) whether both male and female animals were used; (vii) whether animals were allocated randomly to groups; (viii) whether the duration of observation was adequate; and (ix) whether the data were reported and analysed adequately.

When benign tumours (a) occur together with and originate from the same cell type as malignant tumours in an organ or tissue in a particular study and (b) appear to represent a stage in the progression to malignancy, they are usually combined in the assessment of tumour incidence (Huff *et al.*, 1989). The occurrence of lesions presumed to be preneoplastic may in certain instances aid in assessing the biological plausibility of any neoplastic response observed. If an agent induces only benign neoplasms that appear to be end-points that do not readily undergo

transition to malignancy, the agent should nevertheless be suspected of being carcinogenic and requires further investigation.

(b) Quantitative aspects

The probability that tumours will occur may depend on the species, sex, strain, genetic background and age of the animal, and on the dose, route, timing and duration of the exposure. Evidence of an increased incidence of neoplasms with increasing levels of exposure strengthens the inference of a causal association between the exposure and the development of neoplasms.

The form of the dose–response relationship can vary widely, depending on the particular agent under study and the target organ. Mechanisms such as induction of DNA damage or inhibition of repair, altered cell division and cell death rates and changes in intercellular communication are important determinants of dose–response relationships for some carcinogens. Since many chemicals require metabolic activation before being converted to their reactive intermediates, both metabolic and toxicokinetic aspects are important in determining the dose–response pattern. Saturation of steps such as absorption, activation, inactivation and elimination may produce nonlinearity in the dose–response relationship (Hoel et al., 1983; Gart *et al.*, 1986), as could saturation of processes such as DNA repair. The dose–response relationship can also be affected by differences in survival among the treatment groups.

(c) Statistical analyses

Factors considered include the adequacy of the information given for each treatment group: (i) number of animals studied and number examined histologically, (ii) number of animals with a given tumour type and (iii) length of survival. The statistical methods used should be clearly stated and should be the generally accepted techniques refined for this purpose (Peto *et al.*, 1980; Gart *et al.*, 1986; Portier & Bailer, 1989; Bieler & Williams, 1993). The choice of the most appropriate statistical method requires consideration of whether or not there are differences in survival among the treatment groups; for example, reduced survival because of non-tumour-related mortality can preclude the occurrence of tumours later in life. When detailed information on survival is not available, comparisons of the proportions of tumour-bearing animals among the effective number of animals (alive at the time the first tumour was discovered) can be useful when significant differences in survival occur before tumours appear. The lethality of the tumour also requires consideration: for rapidly fatal tumours, the time of death provides an indication of the time of tumour onset and can be assessed using life-table methods; non-fatal or incidental tumours that do not affect survival can be assessed using methods such as the Mantel-Haenzel test for changes in tumour prevalence. Because tumour lethality is often difficult to determine, methods such as the Poly-K test that do not require such information can also be used. When results are available on the number and size of tumours seen in experimental animals (e.g. papillomas on mouse skin, liver tumours observed through nuclear magnetic resonance tomography), other more complicated statistical procedures may be needed (Sherman *et al.*, 1994; Dunson *et al.*, 2003).

Formal statistical methods have been developed to incorporate historical control data into the analysis of data from a given experiment. These methods assign an appropriate weight to historical and concurrent controls on the basis of the extent of between-study and within-study variability: less weight is given to historical controls when they show a high degree of variability, and greater weight when they show little variability. It is generally not appropriate to discount a tumour response that is significantly increased compared with concurrent controls by arguing that it falls within the range of historical controls,

particularly when historical controls show high between-study variability and are, thus, of little relevance to the current experiment. In analysing results for uncommon tumours, however, the analysis may be improved by considering historical control data, particularly when between-study variability is low. Historical controls should be selected to resemble the concurrent controls as closely as possible with respect to species, gender and strain, as well as other factors such as basal diet and general laboratory environment, which may affect tumour-response rates in control animals (Haseman *et al.*, 1984; Fung *et al.*, 1996; Greim *et al.*, 2003).

Although meta-analyses and combined analyses are conducted less frequently for animal experiments than for epidemiological studies due to differences in animal strains, they can be useful aids in interpreting animal data when the experimental protocols are sufficiently similar.

4. Mechanistic and other relevant data

Mechanistic and other relevant data may provide evidence of carcinogenicity and also help in assessing the relevance and importance of findings of cancer in animals and in humans. The nature of the mechanistic and other relevant data depends on the biological activity of the agent being considered. The Working Group considers representative studies to give a concise description of the relevant data and issues that they consider to be important; thus, not every available study is cited. Relevant topics may include toxicokinetics, mechanisms of carcinogenesis, susceptible individuals, populations and life-stages, other relevant data and other adverse effects. When data on biomarkers are informative about the mechanisms of carcinogenesis, they are included in this section.

These topics are not mutually exclusive; thus, the same studies may be discussed in more than one subsection. For example, a mutation in a gene that codes for an enzyme that metabolizes the agent under study could be discussed in the subsections on toxicokinetics, mechanisms and individual susceptibility if it also exists as an inherited polymorphism.

(a) Toxicokinetic data

Toxicokinetics refers to the absorption, distribution, metabolism and elimination of agents in humans, experimental animals and, where relevant, cellular systems. Examples of kinetic factors that may affect dose–response relationships include uptake, deposition, biopersistence and half-life in tissues, protein binding, metabolic activation and detoxification. Studies that indicate the metabolic fate of the agent in humans and in experimental animals are summarized briefly, and comparisons of data from humans and animals are made when possible. Comparative information on the relationship between exposure and the dose that reaches the target site may be important for the extrapolation of hazards between species and in clarifying the role of in-vitro findings.

(b) Data on mechanisms of carcinogenesis

To provide focus, the Working Group attempts to identify the possible mechanisms by which the agent may increase the risk of cancer. For each possible mechanism, a representative selection of key data from humans and experimental systems is summarized. Attention is given to gaps in the data and to data that suggests that more than one mechanism may be operating. The relevance of the mechanism to humans is discussed, in particular, when mechanistic data are derived from experimental model systems. Changes in the affected organs, tissues or cells can be divided into three non-exclusive levels as described below.

(i) Changes in physiology

Physiological changes refer to exposure-related modifications to the physiology and/or response of cells, tissues and organs. Examples of potentially adverse physiological changes include mitogenesis, compensatory cell division, escape from apoptosis and/or senescence, presence of inflammation, hyperplasia, metaplasia and/or preneoplasia, angiogenesis, alterations in cellular adhesion, changes in steroidal hormones and changes in immune surveillance.

(ii) Functional changes at the cellular level

Functional changes refer to exposure-related alterations in the signalling pathways used by cells to manage critical processes that are related to increased risk for cancer. Examples of functional changes include modified activities of enzymes involved in the metabolism of xenobiotics, alterations in the expression of key genes that regulate DNA repair, alterations in cyclin-dependent kinases that govern cell cycle progression, changes in the patterns of post-translational modifications of proteins, changes in regulatory factors that alter apoptotic rates, changes in the secretion of factors related to the stimulation of DNA replication and transcription and changes in gap–junction-mediated intercellular communication.

(iii) Changes at the molecular level

Molecular changes refer to exposure-related changes in key cellular structures at the molecular level, including, in particular, genotoxicity. Examples of molecular changes include formation of DNA adducts and DNA strand breaks, mutations in genes, chromosomal aberrations, aneuploidy and changes in DNA methylation patterns. Greater emphasis is given to irreversible effects.

The use of mechanistic data in the identification of a carcinogenic hazard is specific to the mechanism being addressed and is not readily described for every possible level and mechanism discussed above.

Genotoxicity data are discussed here to illustrate the key issues involved in the evaluation of mechanistic data.

Tests for genetic and related effects are described in view of the relevance of gene mutation and chromosomal aberration/aneuploidy to carcinogenesis (Vainio *et al.*, 1992; McGregor *et al.*, 1999). The adequacy of the reporting of sample characterization is considered and, when necessary, commented upon; with regard to complex mixtures, such comments are similar to those described for animal carcinogenicity tests. The available data are interpreted critically according to the end-points detected, which may include DNA damage, gene mutation, sister chromatid exchange, micronucleus formation, chromosomal aberrations and aneuploidy. The concentrations employed are given, and mention is made of whether the use of an exogenous metabolic system in vitro affected the test result. These data are listed in tabular form by phylogenetic classification.

Positive results in tests using prokaryotes, lower eukaryotes, insects, plants and cultured mammalian cells suggest that genetic and related effects could occur in mammals. Results from such tests may also give information on the types of genetic effect produced and on the involvement of metabolic activation. Some end-points described are clearly genetic in nature (e.g. gene mutations), while others are associated with genetic effects (e.g. unscheduled DNA synthesis). In-vitro tests for tumour promotion, cell transformation and gap–junction intercellular communication may be sensitive to changes that are not necessarily the result of genetic alterations but that may have specific relevance to the process of carcinogenesis. Critical appraisals of these tests have been published (Montesano *et al.*, 1986; McGregor *et al.*, 1999).

Genetic or other activity manifest in humans and experimental mammals is regarded to be of

greater relevance than that in other organisms. The demonstration that an agent can induce gene and chromosomal mutations in mammals in vivo indicates that it may have carcinogenic activity. Negative results in tests for mutagenicity in selected tissues from animals treated in vivo provide less weight, partly because they do not exclude the possibility of an effect in tissues other than those examined. Moreover, negative results in short-term tests with genetic end-points cannot be considered to provide evidence that rules out the carcinogenicity of agents that act through other mechanisms (e.g. receptor-mediated effects, cellular toxicity with regenerative cell division, peroxisome proliferation) (Vainio *et al.*, 1992). Factors that may give misleading results in short-term tests have been discussed in detail elsewhere (Montesano *et al.*, 1986; McGregor *et al.*, 1999).

When there is evidence that an agent acts by a specific mechanism that does not involve genotoxicity (e.g. hormonal dysregulation, immune suppression, and formation of calculi and other deposits that cause chronic irritation), that evidence is presented and reviewed critically in the context of rigorous criteria for the operation of that mechanism in carcinogenesis (e.g. Capen *et al.*, 1999).

For biological agents such as viruses, bacteria and parasites, other data relevant to carcinogenicity may include descriptions of the pathology of infection, integration and expression of viruses, and genetic alterations seen in human tumours. Other observations that might comprise cellular and tissue responses to infection, immune response and the presence of tumour markers are also considered.

For physical agents that are forms of radiation, other data relevant to carcinogenicity may include descriptions of damaging effects at the physiological, cellular and molecular level, as for chemical agents, and descriptions of how these effects occur. 'Physical agents' may also be considered to comprise foreign bodies, such as surgical implants of various kinds, and poorly soluble fibres, dusts and particles of various sizes, the pathogenic effects of which are a result of their physical presence in tissues or body cavities. Other relevant data for such materials may include characterization of cellular, tissue and physiological reactions to these materials and descriptions of pathological conditions other than neoplasia with which they may be associated.

(c) Other data relevant to mechanisms

A description is provided of any structure–activity relationships that may be relevant to an evaluation of the carcinogenicity of an agent, the toxicological implications of the physical and chemical properties, and any other data relevant to the evaluation that are not included elsewhere.

High-output data, such as those derived from gene expression microarrays, and high-throughput data, such as those that result from testing hundreds of agents for a single end-point, pose a unique problem for the use of mechanistic data in the evaluation of a carcinogenic hazard. In the case of high-output data, there is the possibility to overinterpret changes in individual end-points (e.g. changes in expression in one gene) without considering the consistency of that finding in the broader context of the other end-points (e.g. other genes with linked transcriptional control). High-output data can be used in assessing mechanisms, but all end-points measured in a single experiment need to be considered in the proper context. For high-throughput data, where the number of observations far exceeds the number of end-points measured, their utility for identifying common mechanisms across multiple agents is enhanced. These data can be used to identify mechanisms that not only seem plausible, but also have a consistent pattern of carcinogenic response across entire classes of related compounds.

(d) Susceptibility data

Individuals, populations and life-stages may have greater or lesser susceptibility to an agent, based on toxicokinetics, mechanisms of carcinogenesis and other factors. Examples of host and genetic factors that affect individual susceptibility include sex, genetic polymorphisms of genes involved in the metabolism of the agent under evaluation, differences in metabolic capacity due to life-stage or the presence of disease, differences in DNA repair capacity, competition for or alteration of metabolic capacity by medications or other chemical exposures, pre-existing hormonal imbalance that is exacerbated by a chemical exposure, a suppressed immune system, periods of higher-than-usual tissue growth or regeneration and genetic polymorphisms that lead to differences in behaviour (e.g. addiction). Such data can substantially increase the strength of the evidence from epidemiological data and enhance the linkage of in-vivo and in-vitro laboratory studies to humans.

(e) Data on other adverse effects

Data on acute, subchronic and chronic adverse effects relevant to the cancer evaluation are summarized. Adverse effects that confirm distribution and biological effects at the sites of tumour development, or alterations in physiology that could lead to tumour development, are emphasized. Effects on reproduction, embryonic and fetal survival and development are summarized briefly. The adequacy of epidemiological studies of reproductive outcome and genetic and related effects in humans is judged by the same criteria as those applied to epidemiological studies of cancer, but fewer details are given.

5. Summary

This section is a summary of data presented in the preceding sections. Summaries can be found on the *Monographs* programme web site (http://monographs.iarc.fr).

(a) Exposure data

Data are summarized, as appropriate, on the basis of elements such as production, use, occurrence and exposure levels in the workplace and environment and measurements in human tissues and body fluids. Quantitative data and time trends are given to compare exposures in different occupations and environmental settings. Exposure to biological agents is described in terms of transmission, prevalence and persistence of infection.

(b) Cancer in humans

Results of epidemiological studies pertinent to an assessment of human carcinogenicity are summarized. When relevant, case reports and correlation studies are also summarized. The target organ(s) or tissue(s) in which an increase in cancer was observed is identified. Dose–response and other quantitative data may be summarized when available.

(c) Cancer in experimental animals

Data relevant to an evaluation of carcinogenicity in animals are summarized. For each animal species, study design and route of administration, it is stated whether an increased incidence, reduced latency, or increased severity or multiplicity of neoplasms or preneoplastic lesions were observed, and the tumour sites are indicated. If the agent produced tumours after prenatal exposure or in single-dose experiments, this is also mentioned. Negative findings, inverse relationships, dose–response and other quantitative data are also summarized.

(d) Mechanistic and other relevant data

Data relevant to the toxicokinetics (absorption, distribution, metabolism, elimination) and

the possible mechanism(s) of carcinogenesis (e.g. genetic toxicity, epigenetic effects) are summarized. In addition, information on susceptible individuals, populations and life-stages is summarized. This section also reports on other toxic effects, including reproductive and developmental effects, as well as additional relevant data that are considered to be important.

6. Evaluation and rationale

Evaluations of the strength of the evidence for carcinogenicity arising from human and experimental animal data are made, using standard terms. The strength of the mechanistic evidence is also characterized.

It is recognized that the criteria for these evaluations, described below, cannot encompass all of the factors that may be relevant to an evaluation of carcinogenicity. In considering all of the relevant scientific data, the Working Group may assign the agent to a higher or lower category than a strict interpretation of these criteria would indicate.

These categories refer only to the strength of the evidence that an exposure is carcinogenic and not to the extent of its carcinogenic activity (potency). A classification may change as new information becomes available.

An evaluation of the degree of evidence is limited to the materials tested, as defined physically, chemically or biologically. When the agents evaluated are considered by the Working Group to be sufficiently closely related, they may be grouped together for the purpose of a single evaluation of the degree of evidence.

(a) Carcinogenicity in humans

The evidence relevant to carcinogenicity from studies in humans is classified into one of the following categories:

***Sufficient evidence of carcinogenicity*:** The Working Group considers that a causal relationship has been established between exposure to the agent and human cancer. That is, a positive relationship has been observed between the exposure and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence. A statement that there is *sufficient evidence* is followed by a separate sentence that identifies the target organ(s) or tissue(s) where an increased risk of cancer was observed in humans. Identification of a specific target organ or tissue does not preclude the possibility that the agent may cause cancer at other sites.

***Limited evidence of carcinogenicity*:** A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

***Inadequate evidence of carcinogenicity*:** The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer, or no data on cancer in humans are available.

***Evidence suggesting lack of carcinogenicity*:** There are several adequate studies covering the full range of levels of exposure that humans are known to encounter, which are mutually consistent in not showing a positive association between exposure to the agent and any studied cancer at any observed level of exposure. The results from these studies alone or combined should have narrow confidence intervals with an upper limit close to the null value (e.g. a relative risk of 1.0). Bias and confounding should be ruled out with reasonable confidence, and the studies should have an adequate length of follow-up. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the cancer sites, conditions and levels of exposure, and length of observation covered by the available studies. In

addition, the possibility of a very small risk at the levels of exposure studied can never be excluded.

In some instances, the above categories may be used to classify the degree of evidence related to carcinogenicity in specific organs or tissues.

When the available epidemiological studies pertain to a mixture, process, occupation or industry, the Working Group seeks to identify the specific agent considered most likely to be responsible for any excess risk. The evaluation is focused as narrowly as the available data on exposure and other aspects permit.

(b) Carcinogenicity in experimental animals

Carcinogenicity in experimental animals can be evaluated using conventional bioassays, bioassays that employ genetically modified animals, and other in-vivo bioassays that focus on one or more of the critical stages of carcinogenesis. In the absence of data from conventional long-term bioassays or from assays with neoplasia as the end-point, consistently positive results in several models that address several stages in the multistage process of carcinogenesis should be considered in evaluating the degree of evidence of carcinogenicity in experimental animals.

The evidence relevant to carcinogenicity in experimental animals is classified into one of the following categories:

***Sufficient evidence of carcinogenicity*:** The Working Group considers that a causal relationship has been established between the agent and an increased incidence of malignant neoplasms or of an appropriate combination of benign and malignant neoplasms in (a) two or more species of animals or (b) two or more independent studies in one species carried out at different times or in different laboratories or under different protocols. An increased incidence of tumours in both sexes of a single species in a well conducted study, ideally conducted under Good Laboratory Practices, can also provide *sufficient evidence*.

A single study in one species and sex might be considered to provide *sufficient evidence of carcinogenicity* when malignant neoplasms occur to an unusual degree with regard to incidence, site, type of tumour or age at onset, or when there are strong findings of tumours at multiple sites.

***Limited evidence of carcinogenicity*:** The data suggest a carcinogenic effect but are limited for making a definitive evaluation because, e.g. (a) the evidence of carcinogenicity is restricted to a single experiment; (b) there are unresolved questions regarding the adequacy of the design, conduct or interpretation of the studies; (c) the agent increases the incidence only of benign neoplasms or lesions of uncertain neoplastic potential; or (d) the evidence of carcinogenicity is restricted to studies that demonstrate only promoting activity in a narrow range of tissues or organs.

***Inadequate evidence of carcinogenicity*:** The studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect because of major qualitative or quantitative limitations, or no data on cancer in experimental animals are available.

***Evidence suggesting lack of carcinogenicity*:** Adequate studies involving at least two species are available which show that, within the limits of the tests used, the agent is not carcinogenic. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the species, tumour sites, age at exposure, and conditions and levels of exposure studied.

(c) Mechanistic and other relevant data

Mechanistic and other evidence judged to be relevant to an evaluation of carcinogenicity and of sufficient importance to affect the overall evaluation is highlighted. This may include data on preneoplastic lesions, tumour pathology, genetic and related effects, structure–activity relationships, metabolism and toxicokinetics,

physicochemical parameters and analogous biological agents.

The strength of the evidence that any carcinogenic effect observed is due to a particular mechanism is evaluated, using terms such as 'weak', 'moderate' or 'strong'. The Working Group then assesses whether that particular mechanism is likely to be operative in humans. The strongest indications that a particular mechanism operates in humans derive from data on humans or biological specimens obtained from exposed humans. The data may be considered to be especially relevant if they show that the agent in question has caused changes in exposed humans that are on the causal pathway to carcinogenesis. Such data may, however, never become available, because it is at least conceivable that certain compounds may be kept from human use solely on the basis of evidence of their toxicity and/or carcinogenicity in experimental systems.

The conclusion that a mechanism operates in experimental animals is strengthened by findings of consistent results in different experimental systems, by the demonstration of biological plausibility and by coherence of the overall database. Strong support can be obtained from studies that challenge the hypothesized mechanism experimentally, by demonstrating that the suppression of key mechanistic processes leads to the suppression of tumour development. The Working Group considers whether multiple mechanisms might contribute to tumour development, whether different mechanisms might operate in different dose ranges, whether separate mechanisms might operate in humans and experimental animals and whether a unique mechanism might operate in a susceptible group. The possible contribution of alternative mechanisms must be considered before concluding that tumours observed in experimental animals are not relevant to humans. An uneven level of experimental support for different mechanisms may reflect that disproportionate resources have been focused on investigating a favoured mechanism.

For complex exposures, including occupational and industrial exposures, the chemical composition and the potential contribution of carcinogens known to be present are considered by the Working Group in its overall evaluation of human carcinogenicity. The Working Group also determines the extent to which the materials tested in experimental systems are related to those to which humans are exposed.

(d) Overall evaluation

Finally, the body of evidence is considered as a whole, to reach an overall evaluation of the carcinogenicity of the agent to humans.

An evaluation may be made for a group of agents that have been evaluated by the Working Group. In addition, when supporting data indicate that other related agents, for which there is no direct evidence of their capacity to induce cancer in humans or in animals, may also be carcinogenic, a statement describing the rationale for this conclusion is added to the evaluation narrative; an additional evaluation may be made for this broader group of agents if the strength of the evidence warrants it.

The agent is described according to the wording of one of the following categories, and the designated group is given. The categorization of an agent is a matter of scientific judgement that reflects the strength of the evidence derived from studies in humans and in experimental animals and from mechanistic and other relevant data.

Group 1: The agent is carcinogenic to humans.

This category is used when there is *sufficient evidence of carcinogenicity* in humans. Exceptionally, an agent may be placed in this category when evidence of carcinogenicity in humans is less than *sufficient* but there is *sufficient evidence of carcinogenicity* in experimental

animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

Group 2.

This category includes agents for which, at one extreme, the degree of evidence of carcinogenicity in humans is almost *sufficient*, as well as those for which, at the other extreme, there are no human data but for which there is evidence of carcinogenicity in experimental animals. Agents are assigned to either Group 2A (*probably carcinogenic to humans*) or Group 2B (*possibly carcinogenic to humans*) on the basis of epidemiological and experimental evidence of carcinogenicity and mechanistic and other relevant data. The terms *probably carcinogenic* and *possibly carcinogenic* have no quantitative significance and are used simply as descriptors of different levels of evidence of human carcinogenicity, with *probably carcinogenic* signifying a higher level of evidence than *possibly carcinogenic*.

Group 2A: The agent is probably carcinogenic to humans.

This category is used when there is *limited evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals. In some cases, an agent may be classified in this category when there is *inadequate evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals and strong evidence that the carcinogenesis is mediated by a mechanism that also operates in humans. Exceptionally, an agent may be classified in this category solely on the basis of *limited evidence of carcinogenicity* in humans. An agent may be assigned to this category if it clearly belongs, based on mechanistic considerations, to a class of agents for which one or more members have been classified in Group 1 or Group 2A.

Group 2B: The agent is possibly carcinogenic to humans.

This category is used for agents for which there is *limited evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals. It may also be used when there is *inadequate evidence of carcinogenicity* in humans but there is *sufficient evidence of carcinogenicity* in experimental animals. In some instances, an agent for which there is *inadequate evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data.

Group 3: The agent is not classifiable as to its carcinogenicity to humans.

This category is used most commonly for agents for which the evidence of carcinogenicity is *inadequate* in humans and *inadequate* or *limited* in experimental animals.

Exceptionally, agents for which the evidence of carcinogenicity is *inadequate* in humans but *sufficient* in experimental animals may be placed in this category when there is strong evidence that the mechanism of carcinogenicity in experimental animals does not operate in humans.

Agents that do not fall into any other group are also placed in this category.

An evaluation in Group 3 is not a determination of non-carcinogenicity or overall safety. It often means that further research is needed, especially when exposures are widespread or the cancer data are consistent with differing interpretations.

Group 4: The agent is probably not carcinogenic to humans.

This category is used for agents for which there is *evidence suggesting lack of carcinogenicity*

in humans and in experimental animals. In some instances, agents for which there is *inadequate evidence of carcinogenicity* in humans but *evidence suggesting lack of carcinogenicity* in experimental animals, consistently and strongly supported by a broad range of mechanistic and other relevant data, may be classified in this group.

(e) Rationale

The reasoning that the Working Group used to reach its evaluation is presented and discussed. This section integrates the major findings from studies of cancer in humans, studies of cancer in experimental animals, and mechanistic and other relevant data. It includes concise statements of the principal line(s) of argument that emerged, the conclusions of the Working Group on the strength of the evidence for each group of studies, citations to indicate which studies were pivotal to these conclusions, and an explanation of the reasoning of the Working Group in weighing data and making evaluations. When there are significant differences of scientific interpretation among Working Group Members, a brief summary of the alternative interpretations is provided, together with their scientific rationale and an indication of the relative degree of support for each alternative.

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GENERAL REMARKS

This one-hundred-and-seventh volume of the *IARC Monographs* contains evaluations of the carcinogenic hazard to humans of exposure to polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs). The *IARC Monographs* programme has conducted several evaluations of the carcinogenicity of these agents (IARC, 1978, 1979, 1987; Table 1). At the meeting of the Advisory Group to Recommend Priorities for the IARC Monographs in 2008, PCBs were identified as an agent with high priority for re-evaluation (IARC, 2009). In the framework of the re-evaluation of 2,3,7,8-tetrachlorodibenzo-*para*-dioxin (TCDD) in October 2009 (IARC, 2012), the congener PCB-126 was upgraded to Group 1, and the Working Group recommended that there be an in-depth re-evaluation of agents with properties similar to TCDD (IARC, 2012). In February 2013, the *IARC Monographs* Working Group undertook a re-evaluation of PCBs and PBBs. A summary of the findings of this meeting appears in *The Lancet Oncology* (Lauby-Secretan *et al.*, 2013).

1. Considerations for definitions and nomenclature of PCBs and PBBs

Four decades after national governments began to ban their production and use, PCBs and PBBs remain a major concern to human health and the natural environment. Epidemiological studies in occupational settings generally studied workers exposed to the "fresh" product, by inhalation or dermal contact, while studies in the general population assessed individuals exposed primarily through intake of contaminated food, for which the exposure profile is difficult to assess. In contrast, experimental studies assessed individual congeners, combinations of a few congeners, or "fresh" commercial PCB products; however, none of these are identical to the PCB or PBB profiles to which people are exposed today. Indeed, most human exposure today is to complex mixtures originating from commercial products that have been altered by environmental processes (i.e. weathering, transport, and bioaccumulation).

The reason that PCB and PBB mixtures in the environment today differ from the original commercial products is that after release into the environment, the congener composition changes through partitioning, chemical transformation, and bioaccumulation. Partitioning refers to processes by which different congeners separate into air, water, sediment, and soil. Some congeners tend to volatilize or disperse as aerosols, providing an effective vehicle for long-range transport. Congeners with low chlorine or bromine content tend to be more volatile, and also somewhat soluble in water. Many congeners adsorb to organic materials in sediments and

Table 1 Historical overview of the *IARC Monographs* evaluations of PCBs and PBBs

Agent	Volume	Reference	Evidence in humans	Evidence in experimental animals	Mechanistic considerations	Group
PCBs	7	IARC (1974)	No formal evaluation	No formal evaluation	–	–
	18	IARC (1978)	No formal evaluation	No formal evaluation	–	–
	Suppl. 1	IARC (1979)	Inadequate	Sufficient	–	2B[a]
	Suppl. 4	IARC (1982)	Inadequate	Sufficient	–	2B
	Suppl. 7	IARC (1987)	Limited	Sufficient	–	2A
PCB-126	100F	IARC (2012)	–	Sufficient	Mechanistic upgrade	1
PBBs	18	IARC (1978)	No formal evaluation	No formal evaluation	–	
	41	IARC (1986)	Inadequate	Sufficient	No evidence for genotoxicity	–
	Suppl. 7	IARC (1987)	Inadequate	Sufficient	No evidence for genotoxicity	2B

[a] Possible target organs in humans identified as "skin (melanoma)" and "all sites"
PBB, polybrominated biphenyl; PCB, polychlorinated biphenyl

soils; adsorption tends to increase with chlorine or bromine content of the congener and with the organic content of the other material. Chemical transformation refers to the dechlorination or debromination of congeners. This can occur through photolysis, especially for some PBB congeners, or through interactions with bacteria. Chemical transformation is not synonymous with detoxication, as congeners having carcinogenic activity can be formed through dechlorination. Bioaccumulation occurs because PCBs and PBBs are absorbed by fish and other animals, and are highly soluble in lipids, while metabolism and elimination are relatively slower than absorption. Bioaccumulation through the food-chain tends to concentrate congeners of higher chlorine and bromine content.

The nomenclature of PCBs is complex. Publications often attempt to find dichotomies in these mixtures, or refer to PCBs in loose terms, such as:

- Higher and lower chlorinated
- Non-*ortho*, di-*ortho*, and similar terms
- Planar and non-planar
- Dioxin-like and non-dioxin-like
- Aryl hydrocarbon receptor-activating and non-activating
- High and low toxic equivalency (TEQ)
- Estrogenic and non-estrogenic
- Immunotoxic and non-immunotoxic.

The Working Group considered how to characterize the agents to be evaluated. The possibilities included:

- Specific congeners (e.g. PCB-126, PBB-153);
- Groupings of a small number of congeners (e.g. PCB-126 plus PCB-153);
- Commercial products (e.g. Aroclor 1242, Firemaster FF1);
- Large subsets of congeners (e.g. dioxin-like PCB congeners);
- PCBs or PBBs as a class.

Since human exposure always occurs to mixtures, the Working Group considered that it was appropriate to evaluate PCBs and PBBs each as a group.

2. Analysis of PCBs and PBBs

There are some difficulties in assessing and comparing PCB or PBB concentrations in any medium because of differences in analytical methods between laboratories, and differences in the numbers and types of congeners reported. Since there are 209 congeners, values reported

are rarely for true total PCB or PBB concentrations, but rather for a few selected congeners, or a "total" PCB or PBB concentration reported on the basis of analysis of a certain number of congeners only. Thus both the number and the specific congeners analysed must be considered when comparing results among studies. Another complication is that some authors present results for concentrations in total serum (usually called "wet weight"), while others report concentration in the lipid fraction of serum or other media (called "lipid adjusted"). The rationale for lipid adjustment is that these compounds are lipophilic, although there is some evidence that lipid adjustment poses risk of bias. Some investigators now report results as wet weight concentrations with serum lipids considered as a covariate. A further complication is that concentrations are reported in different units in different studies, and cannot always be directly compared.

Several biomarkers of exposure have been used as indicators of the internal dose or the body burden of PCBs or PBBs. These include measurement in blood (serum or plasma), adipose tissue, maternal or cord blood, breast milk and hair. In principle, blood lipid concentrations reflect recent exposures and the full spectrum of congeners to which a person is exposed, while the profile in adipose tissue reflects long-term intakes. However, recent exposure to less chlorinated congeners could result in higher non-equilibrium levels in the circulation. Levels in breast milk largely reflect the concentrations in adipose tissue.

A common theme with PCBs and PBBs is that major industrial accidents have resulted in unforeseeable human dietary exposure. In the 1968 Yusho incident in Japan, leaking Kanechlor 400 contaminated rice oil destined for human consumption. The 1979 Yucheng incident in Taiwan, China, also involved contamination of rice oil, this time by Kanechlor 500. And during 1973–1974, PBBs were unintentionally shipped as an animal feed supplement, contaminating milk, eggs, other dairy products, beef, pork, sheep, and chickens in Michigan, USA. Each incident involved relatively small amounts of PCBs or PBBs, but soon affected thousands of people. It should be noted that the effects of these incidents are not limited to cancer. The Yusho and Yucheng incidents also involved major effects on skin, such as severe chloracne, and recent studies have linked PBB exposure in Michigan to increased risks of spontaneous abortion, genitourinary conditions in male offspring, and suggestions of altered ovarian function.

3. Assessment of exposure to PCBs in epidemiological studies

Epidemiological studies investigating the potential carcinogenic effects of PCBs are basically of three types: occupational cohorts, environmental cohorts, and case–control studies. Most cohort studies were unable to quantify PCB exposures, although in some studies potential PCB exposure was estimated, or a qualitative scale was used. Within some cohort studies, more detailed analyses were achieved through nested case–control studies that collected additional information, sometimes including biomarkers, for specific subgroups of cancer cases and controls. Studies (nested case–control, and case–control) with biomarkers of exposure allow quantification of PCBs in serum or adipose tissue.

In this last group of studies, PCB exposure has been evaluated in a variety of ways: as to a group of congeners; as more or less specific commercial products; as specific PCB functional groupings; as specific combinations, such as PCB-118 + PCB-126; or as specific congeners.

There are several challenges in the interpretation and evaluation of the evidence for PCBs and cancer:

- In studies of workers and consumers of food items allegedly or known to have been "contaminated" with PCBs, it is usually not possible to determine the actual level of exposure.
- PCB exposure usually occurs to mixtures, and while these are often analysed as individual congeners in studies using biomarkers, many congeners are highly correlated and disentangling results for specific congeners is difficult.
- Several specific congeners are rarely or never included in epidemiological studies, primarily because they are excluded from "batch" gas chromatography analyses in many laboratories. Different studies focus on different PCBs; sometimes congeners are grouped and these groupings may differ across studies. Analytical results for specific congeners are best interpreted as markers for exposure to PCBs in general.
- In the occupational cohorts, the exposure route is usually dermal and inhalation, while in the environmental cohorts and case–control studies, the exposure route is usually ingestion (PCB exposure through diet).
- A few environmental studies refer to acute exposures (accidents), while most studies refer to long-term exposures (occupational exposure, and contamination of diet) and long-term consequences of accidents.
- Latency considerations are usually not possible when using biomarker samples collected long after exposure. This may be a cause for concern in interpreting findings on less persistent lower-chlorinated PCBs, but it would be less so for the persistent highly chlorinated PCBs.
- In principle, the use of biomarkers should reduce exposure-measurement error; studies evaluating biomarkers for many PCB congeners tend to generate multiple comparisons, potentially increasing the number of false-positive associations.
- Sampling may be problematic when adjusting plasma or serum measurements by lipid content, because of lipid degradation in samples. Most cohort studies could not take into account relevant confounders, while some of these were considered in the nested case–control and case–control studies.
- Very few studies have addressed interaction or effect modification with other environmental exposures such as tobacco smoke and other chemicals.

4. Genotoxicity of PCBs

Many early tests for genotoxicity with PCBs, performed 10 years ago or more, reported negative results. However, almost all of these studies are not suited for hazard assessment, primarily due to the low doses tested and, in case of studies in vitro, the lack of an exogenous metabolic system. If retested with metabolic activation, many PCB congeners would show genotoxicity. Most PCB mixtures and the few congeners that were tested gave negative results in the Ames test with and without metabolic activation [reviewed in (Silberhorn *et al.*, 1990; Ludewig, 2001)]. A negative result in the Ames test is not uncommon for compounds with complicated and multistep activation pathways such as that proposed for less chlorinated PCBs, i.e. metabolic activation to quinones. Thus a bacterial test for mutagenicity is probably not an appropriate assay for evaluating the genotoxicity of PCBs.

5. The pleiotropic carcinogenicity of PCBs

In experimental animals, commercial PCB mixtures and some individual congeners are complete carcinogens, producing neoplastic

lesions primarily in the liver (hepatocytes and biliary tract); however, benign and malignant tumours have also been observed in many other organs of the treated animals (lung, oral mucosa, thyroid gland, uterus, skin, and the mammary gland in the offspring of treated mothers).

Accidental release of PCBs into food in Taiwan, China, and in Japan, has led to acute and chronic PCB toxicity in thousands of people. Examination of the mortality rate of the Yusho victims in Japan 40 years after the event revealed an increased risk of all types of cancer combined, cancers of the liver and lung in men, and cancer of the liver in women (Onozuka *et al.*, 2009). A similar 24-year follow-up study of Yucheng victims in Taiwan, China, found increased mortality from liver disease, but no increase in risk of cancer of the liver (Tsai *et al.*, 2007). After reviewing all epidemiological studies on occupational and environmental exposure to PCBs, the Working Group concluded that there was *sufficient evidence* of carcinogenicity in humans, on the basis of an increased risk of malignant melanoma; one study found a significant association with uveal melanoma in exposed workers. In addition, increased risks were seen in some studies between exposure to PCBs and non-Hodgkin lymphoma, and for cancer of the breast in some subgroups of women. Positive findings were observed in individual studies for cancers of the brain, prostate, stomach, and pancreas.

PCBs bioaccumulate in fatty tissue, so higher marine mammals are particularly exposed. Reports of cancers in marine wildlife living in areas with high measured PCB concentrations provide another source of cancer data. For example, a large cell immunoblastic lymphoma in a bottlenose dolphin (*Tursiops truncates*) with high blood PCB concentrations (Jaber *et al.*, 2005); uterine leiomyomas in 257 female Baltic grey seals (*Halichoerus grypus*) (Bredhult *et al.*, 2008); and undefined carcinomas in 38 stranded wild California sea lions (*Zalophus californianus*), which were reported to be strongly associated with high PCB concentrations measured in the animals (Ylitalo *et al.*, 2005).

6. Toxicity and carcinogenicity of PBBs

The Working Group also considered the evidence on carcinogenicity of PBBs. The chemical structure of PBBs resembles that of PCBs, with substitution by bromine rather than chlorine atoms. PBBs were used primarily as flame retardants in the 1970s, but production has been discontinued in most countries for many years. Following the accidental release of PBBs in Michigan, USA, the one study that investigated cancer reported adjusted odds ratios of up to 23-fold for cancer of the digestive system and up to 33-fold for lymphoma, with an exposure–response trend across exposure groups. This study included cancer results until 1993; the study has not yet been updated to include cancers that have occurred during the subsequent 20 years. Concerning experimental and mechanistic studies, while there is an extensive body of literature on the carcinogenicity of PCBs, their brominated analogues have received much less attention and study. PBBs will likely be found to exhibit their toxicity and disease potential via many of the same pathways as their chlorinated counterparts, with equivalent or greater toxicity.

The information contained in this volume has contributed to the report "Health risks of PCB in the indoor climate in Denmark," published by the Sundhedsstyrelsen (Danish Health and Medicines Authority, 2013) and was considered during the evaluation of non-dioxin like PCBs by the Joint FAO/WHO Expert Committee on Food Additives (June 2015).

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POLYCHLORINATED BIPHENYLS

1. EXPOSURE DATA

1.1 Identification of the agent

1.1.1 Nomenclature

Polychlorinated biphenyls (PCBs) are a class of aromatic chemical compounds in which some or all hydrogen atoms attached to the biphenyl ring are substituted by chlorine atoms (m + n = 1–10) (Fig. 1.1). Synonyms for PCBs include chlorinated biphenyls, chlorinated diphenyls, chlorobiphenyls, or polychlorobiphenyls.

The general chemical formula is $C_{12}H_{(10-m-n)}Cl_{(m+n)}$, where (m + n) is the number of chlorine atoms on the two rings. Depending on the position and number of the chlorine atoms, there are theoretically 209 individual PCB compounds (congeners). The carbon positions are numbered 1 to 6 on one ring, and 1′ to 6′ on the other. While positions 2,2′,6, and 6′ are called "*ortho*," positions 3,3′,5 and 5′ are named "*meta*" and positions 4 and 4′ are called "*para*."

Two different but correlated nomenclature systems are currently used. According to the International Union of Pure and Applied Chemistry (IUPAC) and in particular rule A-52.3 related to hydrocarbon systems, an unprimed number is considered lower (higher priority) than the same number when primed. Assemblies of unprimed and primed numbers are arranged in ascending numerical order. For a given PCB congener, the name lists the numbers sequentially [e.g. the PCB congener with chlorines on carbons 2,4,5, and 3′,4′ is identified as 2,3′,4,4′,5 (and not 2′,3,4,4′,5′)]. A deviation in that system lists the unprimed and primed chlorinated ring positions separately, sometimes eliminating the prime symbols and the commas for clarity and ease of typing (e.g. 245–3′4′5′ or 245–345).

In an additional strategy proposed by Ballschmiter & Zell (1980), a number (called "BZ number") is attributed to each individual congener. This number correlates the structural arrangement of the PCB congener and ascending order of number of chlorine substitutions within each sequential homologue (Ballschmiter & Zell, 1980). This results in the congeners being numbered from PCB-1 to PCB-209. This shorthand nomenclature has become quite popular and is convenient for many uses, although it is important to note that it obscures the chemical identity of the congener and does not strictly follow the IUPAC rules.

Slight changes in the original BZ congener-numbering system were later recommended to correct some errors (Schulte & Malisch, 1983; Ballschmiter *et al.*, 1992), and this resulted in the renumbering of BZ numbers 199–201. Guitart *et al.* (1993) used a computer program to systematically renumber the PCBs according to the strict IUPAC rules. As a result, they recommended that the congeners previously numbered 107, 108, 109, 199, 200, and 201 be renumbered 109, 107, 108, 200, 201, and 199, respectively (reviewed in Mills *et al.*, 2007). The nomenclature for PCB congeners based on this report is shown in Table 1.1 and will be preferred in this *Monograph*. However, in the scientific literature,

Table 1.1 Correspondance between BZ number[a] and position of chlorine atoms on each phenyl ring of the PCBs[b]

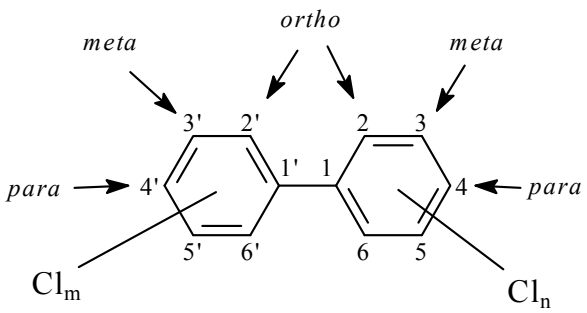
Position of chlorine atom on each ring	2	3	4	2,3	2,4	2,5	2,6	3,4	3,5	2,3,4	2,3,5	2,3,6	2,4,5	2,4,6	3,4,5	2,3,4,5	2,3,4,6	2,3,5,6	2,3,4,5,6
None	1	2	3	5	7	9	10	12	14	21	23	24	29	30	38	61	62	65	116
2′	4	6	8	16	17	18	19	33	34	41	43	45	48	50	76	86	88	93	142
3′		11	13	20	25	26	27	35	36	55	57	59	67	69	78	106	108	112	160
4′			15	22	28	31	32	37	39	60	63	64	74	75	**81**	**114**	115	117	166
2′,3′				40	42	44	46	56	58	82	83	84	97	98	122	129	131	134	173
2′,4′					47	49	51	66	68	85	90	91	99	100	**123**	137	139	147	181
2′,5′						52	53	70	72	87	92	95	101	103	124	141	144	151	185
2′,6′							54	71	73	89	94	96	102	104	125	143	145	152	186
3′,4′								**77**	79	**105**	109	110	**118**	119	**126**	**156**	158	163	190
3′,5′									80	107	111	113	120	121	127	159	161	165	192
2′,3′,4′										128	130	132	138	140	**157**	170	171	177	195
2′,3′,5′											133	135	146	148	162	172	175	178	198
2′,3′,6′												136	149	150	164	174	176	179	200
2′,4′,5′													153	154	**167**	180	183	187	203
2′,4′,6′														155	168	182	184	188	204
3′,4′,5′															**169**	**189**	191	193	205
2′,3′,4′,5′																194	196	199	206
2′,3′,4′,6′																	197	201	207
2′,3′,5′,6′																		202	208
2′,3′,4′,5′,6′																			209

[a] Revised PCB numbering system, including the revised numbering of congeners 107–109 and 199–201. For several PCB congeners, the indicated (truncated) structural names do not strictly adhere to the IUPAC rules (primed and unprimed numbers are interchanged). A comprehensive review of PCB nomenclature, including IUPAC names, is given in Mills *et al.* (2007).

[b] Dioxin-like PCBs are indicated in bold type

BZ, Ballschmiter and Zell; IUPAC, International Union of Pure and Applied Chemistry; PCB, polychlorinated biphenyl

Fig. 1.1 Chemical structure of PCBs and the IUPAC numbering system



Hydrogen atoms in positions 2,2′,6,6′ (*ortho*), 3,3′,5,5′ (*meta*) and/or 4,4′ (*para*) may be substituted by chlorine atoms; (m + n) is the number of chlorine atoms on the two rings
IUPAC, International Union of Pure and Applied Chemistry; PCB, polychlorinated biphenyl

the revised numbering of congeners 107–109 has not been adopted systematically; the numbering system commonly used has been that proposed by Ballschmiter *et al.* (1992) where only the original BZ numbers 199–201 are changed.

PCBs can be categorized by degree of chlorination (number of chlorine atoms) in 10 homologue groups (Table 1.2) from monochlorobiphenyls to decachlorobiphenyls. More than 60% of the PCBs are tetra- to hexachlorophenyls.

In the biphenyl molecule, the two aromatic rings can rotate about the connecting single 1,1′-bond (Fig. 1.1). As with all molecules, there is a low-energy preferred conformation. With PCBs, this conformation is dependent on the degree of chlorine substitution, since chlorine is larger than hydrogen and creates more steric hindrance to the rotation (Erickson, 2001). The two extreme theoretical configurations are "planar" or "coplanar," in which the two benzene rings are in the same plane, and "non-planar" in which the benzene rings are at a 90° angle to each other (Faroon *et al.*, 2000). The probability of attaining a planar configuration is essentially determined by the number of substitutions in the *ortho* positions (2,2′,6,6′): the benzene rings of non-*ortho* substituted PCBs as well as mono-*ortho* substituted PCBs can assume a planar configuration and are referred to as "planar" or "coplanar" congeners (Erickson, 1997). The replacement of hydrogen atoms in the *ortho* positions with larger chlorine atoms forces the aromatic rings to rotate out of the planar configuration (Fig. 1.2); such structures are referred to as "non-planar" or "non-coplanar" congeners. [The Working Group does not recommend the use of this terminology, which is not technically appropriate since these PCBs do not easily assume a planar conformation.]

The relationship between PCB congener number and the Chemical Abstracts Service (CAS) registry number is given in Table 1.3. The congener numbering presented in this table follows that in Table 1.1, with the revised numbering of congeners 107–109. The congener lipophilicity is given in the same table, and was expressed against capacity to partition in octanol and water (K_{ow}) (see Section 1.1.2). Congeners can also be characterized by descriptors (CP0, CP1, 4Cl, PP, 2M) that give rapid access to geometry and substituent positions. The first descriptor, CP0, characterizes 20 congeners that are referred to as non-*ortho* congeners, consisting of those with chlorine substitution at none of the *ortho* positions on the biphenyl backbone. The second descriptor, CP1, comprises 48 congeners that are referred to as mono-*ortho* congeners and include those with chlorine substitution at only one of the *ortho* positions; CP0 and CP1 congeners can adopt a planar configuration. The 4Cl descriptor designates 169 congeners that have a total of four or more chlorine substituents, regardless of position. There are 54 PP congeners that have both *para* positions chlorinated. The 2M group contains 140 congeners that have two or more of the *meta* positions chlorinated. A total of 11 congeners have no descriptor.

The twelve congeners that display all descriptors are referred to as "dioxin-like" (Table 1.4). These twelve PCBs, namely PCB-77, PCB-81, PCB-105, PCB-114, PCB-118, PCB-123, PCB-126, PCB-156, PCB-157, PCB-167, PCB-169, and

Table 1.2 Physical and chemical properties of PCBs according to homologue group

Homologue group	CAS No.	Formula	No. of isomers	BZ No.	Relative molecular mass	Chlorine (% w/w)	Vapour pressure (Pa at 25 °C)[a]	Melting point (°C)[b]	Boiling point (°C)[c]
Monochlorobiphenyl	27323-18-8	$C_{12}H_9Cl$	3	1–3	188.66	18.79	1.1	25–77.9	285
Dichlorobiphenyl	25512-42-9	$C_{12}H_8Cl_2$	12	4–15	223.10	31.77	0.24	24.4–149	312
Trichlorobiphenyl	25323-68-6	$C_{12}H_7Cl_3$	24	16–39	257.55	41.30	0.054	28–87	337
Tetrachlorobiphenyl	26914-33-0	$C_{12}H_6Cl_4$	42	40–81	291.99	48.65	0.012	47–180	360
Pentachlorobiphenyl	25429-29-2	$C_{12}H_5Cl_5$	46	82–127	326.44	54.30	$2.6.10^{-3}$	76.5–124	381
Hexachlorobiphenyl	26601-64-9	$C_{12}H_4Cl_6$	42	128–169	360.88	58.93	$5.8.10^{-4}$	77–200	400
Heptachlorobiphenyl	28655-71-2	$C_{12}H_3Cl_7$	24	170–193	395.33	62.77	$1.3.10^{-4}$	83–149	417
Octachlorobiphenyl	55722-26-4	$C_{12}H_2Cl_8$	12	194–205	429.77	65.98	$2.8.10^{-5}$	159–162	432
Nonachlorobiphenyl	53742-07-7	$C_{12}HCl_9$	3	206–208	464.22	68.73	$6.3.10^{-6}$	182.8–206	445
Decachlorobiphenyl	2051-24-3	$C_{12}Cl_{10}$	1	209	498.66	71.10	$1.4.10^{-6}$	305.9	456

[a] Mean value for liquid.

[b] Values are approximations of the range across the isomers.

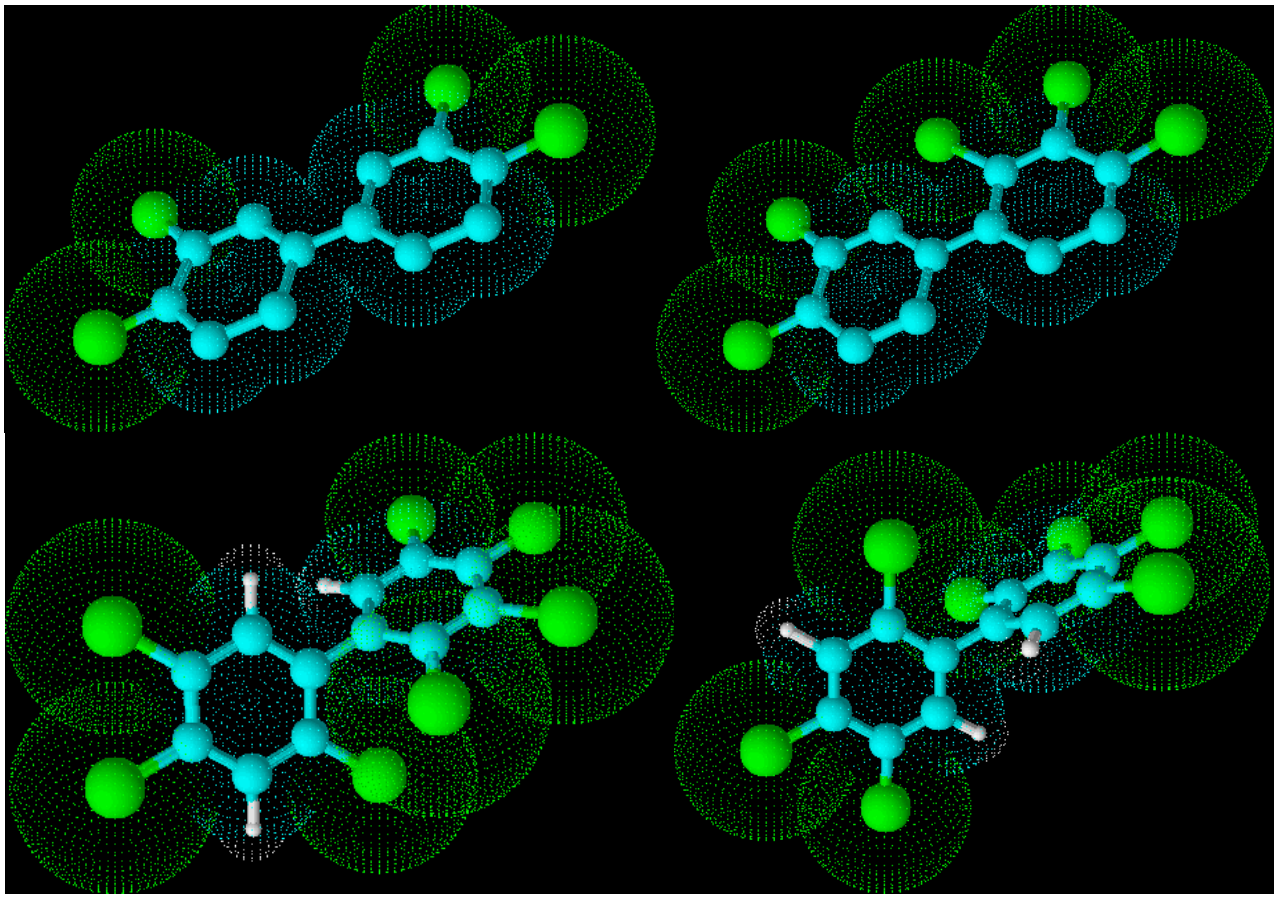
[c] Average value of all isomers in the group.

[The Working Group noted that the CAS No. for octachlorobiphenyl homologue group differs between ATSDR (2000) and Lindell (2012).]

BZ, Ballschmiter and Zell; CAS, Chemical Abstracts Service

From Shiu & Mackay (1986), ATSDR (2000), Erickson (2001), and Lindell (2012)

Fig. 1.2 Tridimensional chemical structures of selected PCBs



Upper panel: Spatial configuration of two dioxin-like PCBs: PCB-77 (3,3′,4,4′-tetrachlorobiphenyl), a non-*ortho* congener (left), and PCB-105 (2,3,3′,4,4′-pentachlorobiphenyl), a mono-*ortho* congener (right)
Lower panel: Spatial configuration of two di-*ortho* PCBs: PCB-153 (2,2′,4,4′,5,5′-hexachlorobiphenyl; left) and PCB-180 (2,2′,3,4,4′,5,5′-heptachlorobiphenyl; right)
Courtesy of Professor B. LeBizec

PCB-189, have been assigned toxicity equivalency factors (TEFs, assigned by WHO in 1998 and revised in 2005) (Van den Berg *et al.*, 2006). [The Working Group stressed that the activities of these PCB congeners are not solely dioxin-like.]

Depending on the context of the study or investigation, specific congeners may be monitored. For instance, the Stockholm Convention on Persistent Organic Pollutants (POPS) recommends measurement of six indicator PCBs (PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, and PCB-180) to characterize contamination by PCBs. These congeners were chosen because they are found at higher concentrations in the environment, in food, or in human fluids/tissues. Depending on country and context, different lists of varying numbers of congeners may be used, e.g. 36 congeners for the Centers for Disease Control and Prevention, USA, or only PCB-138, PCB-153, and PCB-180 most frequently in epidemiological studies with human blood (see Section 2).

Of the 209 PCB congeners, 78 display axial chirality. Only 19 of these congeners, those with three or more chlorine atoms in the *ortho* position, exist as two mirror-image atropisomers, i.e. two chiral atropisomers (Lehmler & Robertson,

Table 1.3 Relationship between BZ number, CAS number, IUPAC name,[a] congener descriptor, and log K_{ow} for individual PCBs

BZ No.	IUPAC name	CAS No.	Descriptor[b]	Log K_{ow}	Vapour pressure (atm at 25 °C)[c]
1	2-CB	2051-60-7	CP1	4.46	
2	3-CB	2051-61-8	CP0	4.69	
3	4-CB	2051-62-9	CP0	4.69	
4	2,2′-DiCB	13029-08-8		4.65	1.5 to 4.2×10^{-6}
5	2,3-DiCB	16605-91-7	CP1	4.97	
6	2,3′-DiCB	25569-80-6	CP1	5.06	
7	2,4-DiCB	33284-50-3	CP1	5.07	9.9×10^{-7} to 2.1×10^{-6}
8	2,4′-DiCB	34883-43-7	CP1	5.07	
9	2,5-DiCB	34883-39-1	CP1	5.06	2.0 to 2.3×10^{-6}
10	2,6-DiCB	33146-45-1		4.84	
11	3,3′-DiCB	2050-67-1	CP0, 2M	5.28	4.1 to 9.1×10^{-7}
12	3,4-DiCB	2974-92-7	CP0	5.22	1.3×10^{-8} to 7.8×10^{-7}
13	3,4′-DiCB	2974-90-5	CP0	5.29	
14	3,5-DiCB	34883-41-5	CP0, 2M	5.28	
15	4,4′-DiCB	2050-68-2	CP0, PP	5.30	5.0 to 7.4×10^{-7}
16	2,2′,3-TriCB	38444-78-9		5.16	
17	2,2′,4-TriCB	37680-66-3		5.25	
18	2,2′,5-TriCB	37680-65-2		5.24	3.5×10^{-7} to 1.2×10^{-6}
19	2,2′,6-TriCB	38444-73-4		5.02	
20	2,3,3′-TriCB	38444-84-7	CP1, 2M	5.57	
21	2,3,4-TriCB	55702-46-0	CP1	5.51	
22	2,3,4′-TriCB	38444-85-8	CP1	5.58	
23	2,3,5-TriCB	55720-44-0	CP1, 2M	5.57	
24	2,3,6-TriCB	55702-45-9		5.35	
25	2,3′,4-TriCB	55712-37-3	CP1	5.67	
26	2,3′,5-TriCB	38444-81-4	CP1, 2M	5.66	1.8 to 4.5×10^{-7}
27	2,3′,6-TriCB	38444-76-7		5.44	
28	2,4,4′-TriCB	7012-37-5	CP1, PP	5.67	1.5 to 3.3×10^{-7}
29	2,4,5-TriCB	15862-07-4	CP1	5.60	
30	2,4,6-TriCB	35693-92-6		5.44	9.3×10^{-7} to 1.5×10^{-6}
31	2,4′,5-TriCB	16606-02-3	CP1	5.67	
32	2,4′,6-TriCB	38444-77-8		5.44	
33	2,3′,4′-TriCB	38444-86-9	CP1	5.60	
34	2,3′,5′-TriCB	37680-68-5	CP1, 2M	5.66	
35	3,3′,4-TriCB	37680-69-6	CP0, 2M	5.82	
36	3,3′,5-TriCB	38444-87-0	CP0, 2M	5.88	
37	3,4,4′-TriCB	38444-90-5	CP0, PP	5.83	
38	3,4,5-TriCB	53555-66-1	CP0, 2M	5.76	
39	3,4′,5-TriCB	38444-88-1	CP0, 2M	5.89	
40	2,2′,3,3′-TetraCB	38444-93-8	4CL, 2M	5.66	4.5×10^{-8} to 1.1×10^{-7}
41	2,2′,3,4-TetraCB	52663-59-9	4CL	5.69	
42	2,2′,3,4′-TetraCB	36559-22-5	4CL	5.76	
43	2,2′,3,5-TetraCB	70362-46-8	4CL, 2M	5.75	
44	2,2′,3,5′-TetraCB	41464-39-5	4CL, 2M	5.75	

Table 1.3 (continued)

BZ No.	IUPAC name	CAS No.	Descriptor[b]	Log K_{ow}	Vapour pressure (atm at 25 °C)[c]
45	2,2′,3,6-TetraCB	70362-45-7	4CL	5.53	
46	2,2′,3,6′-TetraCB	41464-47-5	4CL	5.53	
47	2,2′,4,4′-TetraCB	2437-79-8	4CL, PP	5.85	
48	2,2′,4,5-TetraCB	70362-47-9	4CL	5.78	
49	2,2′,4,5′-TetraCB	41464-40-8	4CL	5.85	
50	2,2′,4,6-TetraCB	62796-65-0	4CL	5.63	
51	2,2′,4,6′-TetraCB	68194-04-7	4CL	5.63	
52	2,2′,5,5′-TetraCB	35693-99-3	4CL, 2M	5.84	$1.8 \text{ to } 8.9 \times 10^{-7}$
53	2,2′,5,6′-TetraCB	41464-41-9	4CL	5.62	$1.1 \text{ to } 4.0 \times 10^{-7}$
54	2,2′,6,6′-TetraCB	15968-05-5	4CL	5.21	$1.2 \times 10^{-6} \text{ to } 6.5 \times 10^{-7}$
55	2,3,3′,4-TetraCB	74338-24-2	CP1, 4CL, 2M	6.11	
56	2,3,3′,4′-TetraCB	41464-43-1	CP1, 4CL, 2M	6.11	
57	2,3,3′,5-TetraCB	70424-67-8	CP1, 4CL, 2M	6.17	
58	2,3,3′,5′-TetraCB	41464-49-7	CP1, 4CL, 2M	6.17	
59	2,3,3′,6-TetraCB	74472-33-6	4CL, 2M	5.95	
60	2,3,4,4′-TetraCB	33025-41-1	CP1, 4CL, PP	6.11	
61	2,3,4,5-TetraCB	33284-53-6	CP1, 4CL, 2M	6.04	
62	2,3,4,6-TetraCB	54230-22-7	4CL	5.89	
63	2,3,4′,5-TetraCB	74472-34-7	CP1, 4CL, 2M	6.17	
64	2,3,4′,6-TetraCB	52663-58-8	4CL	5.95	
65	2,3,5,6-TetraCB	33284-54-7	4CL, 2M	5.86	
66	2,3′,4,4′-TetraCB	32598-10-0	CP1, 4CL, PP	6.20	
67	2,3′,4,5-TetraCB	73575-53-8	CP1, 4CL, 2M	6.20	
68	2,3′,4,5′-TetraCB	73575-52-7	CP1, 4CL, 2M	6.26	
69	2,3′,4,6-TetraCB	60233-24-1	4CL	6.04	
70	2,3′,4′,5-TetraCB	32598-11-1	CP1, 4CL, 2M	6.20	
71	2,3′,4′,6-TetraCB	41464-46-4	4CL	5.98	
72	2,3′,5,5′-TetraCB	41464-42-0	CP1, 4CL, 2M	6.26	
73	2,3′,5′,6-TetraCB	74338-23-1	4CL, 2M	6.04	
74	2,4,4′,5-TetraCB	32690-93-0	CP1, 4CL, PP	6.20	
75	2,4,4′,6-TetraCB	32598-12-2	4CL, PP	6.05	
76	2,3′,4′,5′-TetraCB	70362-48-0	CP1, 4CL, 2M	6.13	
77	3,3′,4,4′-TetraCB	32598-13-3	**CP0, 4CL, PP, 2M**	6.36	$5.2 \times 10^{-9} \text{ to } 2.1 \times 10^{-8}$
78	3,3′,4,5-TetraCB	70362-49-1	CP0, 4CL, 2M	6.35	
79	3,3′,4,5′-TetraCB	41464-48-6	CP0, 4CL, 2M	6.42	
80	3,3′,5,5′-TetraCB	33284-52-5	CP0, 4CL, 2M	6.48	
81	3,4,4′,5-TetraCB	70362-50-4	**CP0, 4CL, PP, 2M**	6.36	
82	2,2′,3,3′,4-PentaCB	52663-62-4	4CL, 2M	6.20	
83	2,2′,3,3′,5-PentaCB	60145-20-2	4CL, 2M	6.26	
84	2,2′,3,3′,6-PentaCB	52663-60-2	4CL, 2M	6.04	
85	2,2′,3,4,4′-PentaCB	65510-45-4	4CL, PP	6.30	
86	2,2′,3,4,5-PentaCB	55312-69-1	4CL, 2M	6.23	
87	2,2′,3,4,5′-PentaCB	38380-02-8	4CL, 2M	6.29	
88	2,2′,3,4,6-PentaCB	55215-17-3	4CL	6.07	
89	2,2′,3,4,6′-PentaCB	73575-57-2	4CL	6.07	

Table 1.3 (continued)

BZ No.	IUPAC name	CAS No.	Descriptor[b]	Log K_{ow}	Vapour pressure (atm at 25 °C)[c]
90	2,2′,3,4′,5-PentaCB	68194-07-0	4CL, 2M	6.36	
91	2,2′,3,4′,6-PentaCB	68194-05-8	4CL	6.13	
92	2,2′,3,5,5′-PentaCB	52663-61-3	4CL, 2M	6.35	
93	2,2′,3,5,6-PentaCB	73575-56-1	4CL, 2M	6.04	
94	2,2′,3,5,6′-PentaCB	73575-55-0	4CL, 2M	6.13	
95	2,2′,3,5′,6-PentaCB	38379-99-6	4CL, 2M	6.13	
96	2,2′,3,6,6′-PentaCB	73575-54-9	4CL	5.71	
97	2,2′,3,4′,5′-PentaCB	41464-51-1	4CL, 2M	6.29	
98	2,2′,3,4′,6′-PentaCB	60233-25-2	4CL	6.13	
99	2,2′,4,4′,5-PentaCB	38380-01-7	4CL, PP	6.39	
100	2,2′,4,4′,6-PentaCB	39485-83-1	4CL, PP	6.23	
101	2,2′,4,5,5′-PentaCB	37680-73-2	4CL, 2M	6.38	$1.4 \text{ to } 3.5 \times 10^{-8}$
102	2,2′,4,5,6′-PentaCB	68194-06-9	4CL	6.16	
103	2,2′,4,5′,6-PentaCB	60145-21-3	4CL	6.22	
104	2,2′,4,6,6′-PentaCB	56558-16-8	4CL	5.81	$4.3 \times 10^{-8} \text{ to } 1.7 \times 10^{-7}$
105	2,3,3′,4,4′-PentaCB	32598-14-4	**CP1, 4CL, PP, 2M**	6.65	8.6×10^{-9}
106	2,3,3′,4,5-PentaCB	70424-69-0	CP1, 4CL, 2M	6.64	
107	2,3,3′,4,5′-PentaCB	70424-68-9	CP1, 4CL, 2M	6.71	
108	2,3,3′,4,6-PentaCB	70362-41-3	4CL, 2M	6.72	
109	2,3,3′,4′,5-PentaCB	74472-35-8	CP1, 4CL, 2M	6.48	
110	2,3,3′,4′,6-PentaCB	38380-03-9	4CL, 2M	6.48	
111	2,3,3′,5,5′-PentaCB	39635-32-0	CP1, 4CL, 2M	6.76	
112	2,3,3′,5,6-PentaCB	74472-36-9	4CL, 2M	6.45	
113	2,3,3′,5′,6-PentaCB	68194-10-5	4CL, 2M	6.54	
114	2,3,4,4′,5-PentaCB	74472-37-0	**CP1, 4CL, PP, 2M**	6.65	
115	2,3,4,4′,6-PentaCB	74472-38-1	4CL, PP	6.49	
116	2,3,4,5,6-PentaCB	18259-05-7	4CL, 2M	6.33	
117	2,3,4′,5,6-PentaCB	68194-11-6	4CL, 2M	6.46	
118	2,3′,4,4′,5-PentaCB	31508-00-6	**CP1, 4CL, PP, 2M**	6.74	1.2×10^{-8}
119	2,3′,4,4′,6-PentaCB	56558-17-9	4CL, PP	6.58	
120	2,3′,4,5,5′-PentaCB	68194-12-7	CP1, 4CL, 2M	6.79	
121	2,3′,4,5′,6-PentaCB	56558-18-0	4CL, 2M	6.64	
122	2,3,3′,4′,5′-PentaCB	76842-07-4	CP1, 4CL, 2M	6.64	
123	2,3′,4,4′,5′-PentaCB	65510-44-3	**CP1, 4CL, PP, 2M**	6.74	
124	2,3′,4′,5,5′-PentaCB	70424-70-3	CP1, 4CL, 2M	6.73	
125	2,3′,4′,5′,6-PentaCB	74472-39-2	4CL, 2M	6.51	
126	3,3′,4,4′,5-PentaCB	57465-28-8	**CP0, 4CL, PP, 2M**	6.89	
127	3,3′,4,5,5′-PentaCB	39635-33-1	CP0, 4CL, 2M	6.95	
128	2,2′,3,3′,4,4′-HexaCB	38380-07-3	4CL, PP, 2M	6.74	$1.0 \text{ to } 3.6 \times 10^{-9}$
129	2,2′,3,3′,4,5-HexaCB	55215-18-4	4CL, 2M	6.73	
130	2,2′,3,3′,4,5′-HexaCB	52663-66-8	4CL, 2M	6.80	
131	2,2′,3,3′,4,6-HexaCB	61798-70-7	4CL, 2M	6.58	
132	2,2′,3,3′,4,6′-HexaCB	38380-05-1	4CL, 2M	6.58	
133	2,2′,3,3′,5,5′-HexaCB	35694-04-3	4CL, 2M	6.86	
134	2,2′,3,3′,5,6-HexaCB	52704-70-8	4CL, 2M	6.55	

Table 1.3 (continued)

BZ No.	IUPAC name	CAS No.	Descriptor[b]	Log K_{ow}	Vapour pressure (atm at 25 °C)[c]
135	2,2′,3,3′,5,6′-HexaCB	52744-13-5	4CL, 2M	6.64	
136	2,2′,3,3′,6,6′-HexaCB	38411-22-2	4CL, 2M	6.22	
137	2,2′3,4,4′,5-HexaCB	35694-06-5	4CL, PP, 2M	6.83	
138	2,2′,3,4,4′,5′-HexaCB	35065-28-2	4CL, PP, 2M	6.83	5.2×10^{-9}
139	2,2′,3,4,4′,6-HexaCB	56030-56-9	4CL, PP	6.67	
140	2,2′,3,4,4′,6′-HexaCB	59291-64-4	4CL, PP	6.67	
141	2,2′,3,4,5,5′-HexaCB	52712-04-6	4CL, 2M	6.82	
142	2,2′,3,4,5,6-HexaCB	41411-61-4	4CL, 2M	6.51	
143	2,2′,3,4,5,6′-HexaCB	68194-15-0	4CL, 2M	6.60	
144	2,2′,3,4,5′,6-HexaCB	68194-14-9	4CL, 2M	6.67	
145	2,2′,3,4,6,6′-HexaCB	74472-40-5	4CL	6.25	
146	2,2′,3,4′,5,5′-HexaCB	51908-16-8	4CL, 2M	6.89	
147	2,2′,3,4′,5,6-HexaCB	68194-13-8	4CL, 2M	6.64	
148	2,2′,3,4′,5,6′-HexaCB	74472-41-6	4CL, 2M	6.73	
149	2,2′,3,4′,5′,6-HexaCB	38380-04-0	4CL, 2M	6.67	
150	2,2′,3,4′,6,6′-HexaCB	68194-08-1	4CL	6.32	
151	2,2′,3,5,5′,6-HexaCB	52663-63-5	4CL, 2M	6.64	
152	2,2′,3,5,6,6′-HexaCB	68194-09-2	4CL, 2M	6.22	
153	2,2′,4,4′,5,5′-HexaCB	35065-27-1	4CL, PP, 2M	6.92	1.9×10^{-9} to 6.9×10^{-8}
154	2,2′,4,4′,5,6′-HexaCB	60145-22-4	4CL, PP	6.76	
155	2,2′,4,4′,6,6′-HexaCB	33979-03-2	4CL, PP	6.41	3.5×10^{-9} to 4.4×10^{-8}
156	2,3,3′,4,4′,5-HexaCB	38380-08-4	**CP1, 4CL, PP, 2M**	7.18	2.1×10^{-9}
157	2,3,3′,4,4′,5′-HexaCB	69782-90-7	**CP1, 4CL, PP, 2M**	7.18	
158	2,3,3′,4,4′,6-HexaCB	74472-42-7	4CL, PP, 2M	7.02	
159	2,3,3′,4,5,5′-HexaCB	39635-35-3	CP1, 4CL, 2M	7.24	
160	2,3,3′,4,5,6-HexaCB	41411-62-5	4CL, 2M	6.93	
161	2,3,3′,4,5′,6-HexaCB	74472-43-8	4CL, 2M	7.08	
162	2,3,3′,4′,5,5′-HexaCB	39635-34-2	CP1, 4CL, 2M	7.24	
163	2,3,3′,4′,5,6-HexaCB	74472-44-9	4CL, 2M	6.99	7.9×10^{-10}
164	2,3,3′,4′,5′,6-HexaCB	74472-45-0	4CL, 2M	7.02	
165	2,3,3′,5,5′,6-HexaCB	74472-46-1	4CL, 2M	7.05	
166	2,3,4,4′,5,6-HexaCB	41411-63-6	4CL, PP, 2M	6.93	
167	2,3′,4,4′,5,5′-HexaCB	52663-72-6	**CP1, 4CL, PP, 2M**	7.27	
168	2,3′,4,4′,5′,6-HexaCB	59291-65-5	4CL, PP, 2M	7.11	
169	3,3′,4,4′,5,5′-HexaCB	32774-16-6	**CP0, 4CL, PP, 2M**	7.42	7.9×10^{-10}
170	2,2′,3,3′,4,4′,5-HeptaCB	35065-30-6	4CL, PP, 2M	7.27	
171	2,2′,3,3′,4,4′,6-HeptaCB	52663-71-5	4CL, PP, 2M	7.11	
172	2,2′,3,3′,4,5,5′-HeptaCB	52663-74-8	4CL, 2M	7.33	
173	2,2′,3,3′,4,5,6-HeptaCB	68194-16-1	4CL, 2M	7.02	
174	2,2′,3,3′,4,5,6′-HeptaCB	38411-25-5	4CL, 2M	7.11	
175	2,2′,3,3′,4,5′,6-HeptaCB	40186-70-7	4CL, 2M	7.17	
176	2,2′,3,3′,4,6,6′-HeptaCB	52663-65-7	4CL, 2M	6.76	
177	2,2′,3,3′,4,5′,6′-HeptaCB	52663-70-4	4CL, 2M	7.08	
178	2,2′,3,3′,5,5′,6-HeptaCB	52663-67-9	4CL, 2M	7.14	
179	2,2′,3,3′,5,6,6′-HeptaCB	52663-64-6	4CL, 2M	6.73	

Table 1.3 (continued)

BZ No.	IUPAC name	CAS No.	Descriptor[b]	Log K_{ow}	Vapour pressure (atm at 25 °C)[c]
180	2,2′,3,4,4′,5,5′-HeptaCB	35065-29-3	4CL, PP, 2M	7.36	1.3×10^{-9}
181	2,2′,3,4,4′,5,6-HeptaCB	74472-47-2	4CL, PP, 2M	7.11	
182	2,2′,3,4,4′,5,6′-HeptaCB	60145-23-5	4CL, PP, 2M	7.20	
183	2,2′,3,4,4′,5′,6-HeptaCB	52663-69-1	4CL, PP, 2M	7.20	
184	2,2′,3,4,4′,6,6′-HeptaCB	74472-48-3	4CL, PP	6.85	
185	2,2′,3,4,5,5′,6-HeptaCB	52712-05-7	4CL, 2M	7.11	
186	2,2′,3,4,5,6,6′-HeptaCB	74472-49-4	4CL, 2M	6.69	
187	2,2′,3,4′,5,5′,6-HeptaCB	52663-68-0	4CL, 2M	7.17	
188	2,2′,3,4′,5,6,6′-HeptaCB	74487-85-7	4CL, 2M	6.82	
189	2,3,3′,4,4′,5,5′-HeptaCB	39635-31-9	**CP1, 4CL, PP, 2M**	7.71	
190	2,3,3′,4,4′,5,6-HeptaCB	41411-64-7	4CL, PP, 2M	7.46	
191	2,3,3′,4,4′,5′,6-HeptaCB	74472-50-7	4CL, PP, 2M	7.55	
192	2,3,3′,4,5,5′,6-HeptaCB	74472-51-8	4CL, 2M	7.52	
193	2,3,3′,4′,5,5′,6-HeptaCB	69782-91-8	4CL, 2M	7.52	
194	2,2′,3,3′,4,4′,5,5′-OctaCB	35694-08-7	4CL, PP, 2M	7.80	
195	2,2′,3,3′,4,4′,5,6-OctaCB	52663-78-2	4CL, PP, 2M	7.56	
196	2,2′,3,3′,4,4′,5,6′-OctaCB	42740-50-1	4CL, PP, 2M	7.65	
197	2,2′,3,3′,4,4′,6,6′-OctaCB	33091-17-7	4CL, PP, 2M	7.30	
198	2,2′,3,3′,4,5,5′,6-OctaCB	68194-17-2	4CL, 2M	7.62	
199	2,2′,3,3′,4,5,5′,6′-OctaCB	52663-75-9	4CL, 2M	7.62	
200	2,2′,3,3′,4,5,6,6′-OctaCB	52663-73-7	4CL, 2M	7.20	
201	2,2′,3,3′,4,5′,6,6′-OctaCB	40186-71-8	4CL, 2M	7.27	
202	2,2′,3,3′,5,5′,6,6′-OctaCB	2136-99-4	4CL, 2M	7.24	
203	2,2′,3,4,4′,5,5′,6-OctaCB	52663-76-0	4CL, PP, 2M	7.65	
204	2,2′,3,4,4′,5,6,6′-OctaCB	74472-52-9	4CL, PP, 2M	7.30	
205	2,3,3′,4,4′,5,5′,6-OctaCB	74472-53-0	4CL, PP, 2M	8.00	
206	2,2′,3,3′,4,4′,5,5′,6-NonaCB	40186-72-9	4CL, PP, 2M	8.09	
207	2,2′,3,3′,4,4′,5,6,6′-NonaCB	52663-79-3	4CL, PP, 2M	7.74	
208	2,2′,3,3′,4,5,5′,6,6′-NonaCB	52663-77-1	4CL, 2M	7.71	
209	2,2′,3,3′,4,4′,5,5′,6,6′-DecaCB	2051-24-3	4CL, PP, 2M	8.18	

[a] The nomenclature in this table adheres to the IUPAC rules and thus primed and unprimed numbers may be interchanged compared with Table 1.1. Please see text for more details.

[b] Congener descriptors (CP0, CP1, 4Cl, PP, 2M) have been given where relevant; they give rapid access to geometry and substituent positions. 68 coplanar congeners fall into one of two groups CP0 or CP1.

The first group of 20 congeners consists of those without chlorine substitution at any of the "*ortho*" positions on the biphenyl backbone and are referred to as CP0 or non-"*ortho*" congeners. The second group of 48 congeners includes those with chlorine substitution at only one of the "*ortho*" positions and are referred to as CP1 or mono-"*ortho*" congeners. 175 congeners have a total of four or more chlorine substituents, regardless of position (4Cl). 54 congeners have both "*para*" positions chlorinated (PP). 146 congeners have two or more of the "*meta*" positions chlorinated (2M). The twelve congeners that have all four of the congener descriptors are referred to as being "dioxin-like," and are indicated in bold type.

In ATSDR (2000), PCB-63 was mistakenly attributed the CAS number of a pentachlorobiphenyl; for Henry's law constants, vapour pressure and solubility of most individual congeners, the reader is referred to Dunnivant & Elzerman (1988) and references within.

[c] Vapour pressures have been indicated for a selection of individual congeners.

BZ, Ballschmiter and Zell; CAS, Chemical Abstracts Service; CB, chlorinated biphenyl; IUPAC, International Union of Pure and Applied Chemistry

From Dunnivant & Elzerman (1988), ATSDR (2000), Mills *et al.* (2007), and Lindell (2012)

Table 1.4 The 12 dioxin-like PCBs, with corresponding CAS number, IUPAC name, and individual WHO_{1998}-TEF and WHO_{2005}-TEF values

PCB	IUPAC name	CAS No.	WHO_{1998}-TEF	WHO_{2005}-TEF
PCB-77	3,3′,4,4′-TetraCB	32598-13-3	0.0001	0.0001
PCB-81	3,4,4′,5-TetraCB	70362-50-4	0.0001	0.0003
PCB-105	2,3,3′,4,4′-PentaCB	32598-14-4	0.0001	0.00003
PCB-114	2,3,4,4′,5-PentaCB	74472-37-0	0.0005	0.00003
PCB-118	2,3′,4,4′,5-PentaCB	31508-00-6	0.0001	0.00003
PCB-123	2,3′,4,4′,5-PentaCB	65510-44-3	0.0001	0.00003
PCB-126	3,3′,4,4′,5-PentaCB	57465-28-8	0.1	0.1
PCB-156	2,3,3′,4,4′,5-HexaCB	38380-08-4	0.0005	0.00003
PCB-157	2,3,3′,4,4′,5′-HexaCB	68782-90-7	0.0005	0.00003
PCB-167	2,3′,4,4′,5,5′-HexaCB	52663-72-6	0.00001	0.00003
PCB-169	3,3′,4,4′,5,5′-HexaCB	32774-16-6	0.01	0.03
PCB-189	2,3,3′,4,4′,5,5′-HeptaCB	39635-31-9	0.0001	0.00003

CAS, Chemical Abstracts Service; CB, chlorinated biphenyl; IUPAC, International Union of Pure and Applied Chemistry; PCB, polychlorinated biphenyl; TEF, toxicity equivalency factor
From Van den Berg *et al.* (1998, 2006)

2001; Kania-Korwel & Lehmler, 2013). The IUPAC nomenclature and BZ number for the 19 atropisomeric PCBs are listed in Table 1.5. They are stereoisomers resulting from hindered rotation around single bonds where the steric-strain barrier to rotation is high enough to allow for the isolation of the enantiomers (Haglund & Wiberg, 1996; Harju & Haglund, 1999). Both atropisomers have the same chemical and physical behaviour, except for optical rotation (Lehmler *et al.*, 2010). They are stable at 25 °C, but at elevated temperatures it is necessary to separate the enantiomers via high-resolution chiral gas chromatography (GC) (Schurig & Reich, 1998; Harju & Haglund, 1999).

1.1.2 Chemical and physical properties of PCBs

Pure single PCB congeners are mostly colourless or slightly yellowish, often odourless, crystalline compounds. Commercial products, however, are viscous liquid mixtures of these compounds, with viscosity increasing with degree of chlorination, and colour ranging from light yellow to a dark colour. For example, Aroclor 1242 is a "mobile liquid" and Aroclor 1260 is a "sticky resin" (Erickson, 2001). These products do not crystallize at low temperatures, but turn into solid resins. An important property of PCBs is their general inertness; they resist acids, alkalis and oxidants and are fire-resistant because of their high flash-points (IPCS, 2003). However, under certain conditions, they may be destroyed by chemical, thermal and biochemical processes. PCBs show excellent dielectric (insulating) properties. This has made them useful in a wide variety of applications, including as dielectric fluids in transformers and capacitors, heat-transfer fluids, and lubricants.

The physical properties of PCBs are important in understanding their analytical, physiological, and environmental properties. However, the interactions of the various physical properties can be extremely complex (Erickson, 2001). Chemical and physical properties such as solubility, vapour pressure, and Henry's law constant have been reported for individual congeners (Shiu & Mackay, 1986; Murphy *et al.*, 1987; Sabljić & Güsten, 1989; Dunnivant *et al.*, 1992; Falconer & Bidleman, 1994). Data for homologue groups and for a selection of PCBs are presented in Table 1.2,

Table 1.5 PCB congeners that exist as chiral atropisomers

PCB	IUPAC name
PCB-45	2,2′,3,6-TetraCB
PCB-84	2,2′,3,3′,6-PentaCB
PCB-88	2,2′,3,4,6-PentaCB
PCB-91	2,2′,3,4′,6-PentaCB
PCB-95	2,2′,3,5′,6-PentaCB
PCB-131	2,2′,3,3′,4,6-HexaCB
PCB-132	2,2′,3,3′,4,6′-HexaCB
PCB-135	2,2′,3,3′,5,6′-HexaCB
PCB-136	2,2′,3,3′,6,6′-HexaCB
PCB-139	2,2′,3,4,4′,6-HexaCB
PCB-144	2,2′,3,4,5′,6-HexaCB
PCB-149	2,2′,3,4′,5′,6-HexaCB
PCB-171	2,2′,3,3′,4,4′,6-HeptaCB
PCB-174	2,2′,3,3′,4,5,6′-HeptaCB
PCB-175	2,2′,3,3′,4,5′,6-HeptaCB
PCB-176	2,2′,3,3′,4,6,6′-HeptaCB
PCB-183	2,2′,3,4,4′,5′,6-HeptaCB
PCB-196	2,2′,3,3′,4,4′,5,6′-OctaCB
PCB-197	2,2′,3,3′,4,4′,6,6′-OctaCB

CB, chlorinated biphenyl; IUPAC, International Union of Pure and Applied Chemistry; PCB, polychlorinated biphenyl

Table 1.3, and Table 1.6. Melting points range from 25 °C (PCB-2, PCB-7 and PCB-9) to 306 °C (PCB-209). Boiling points increase from low (monochlorobiphenyl, 285 °C) to highly (decachlorobiphenyl, 456 °C) chlorinated congeners (Hutzinger *et al.*, 1974; Shiu & Mackay, 1986).

The solubility of PCBs in water is extremely low, ranging from an average of 0.0012 to 4830 µg/L for the chlorobiphenyl congeners that occur commonly. The high solubility of the *ortho*-chlorinated congeners (4.8 mg/L for PCB-1) may be due to hydrogen bonding associated with the more polar character of these molecules. Solubility decreases rapidly in *ortho*-vacant congeners, especially as the *para* positions are filled, which may result in greater and more uniform perimeter electronegativity and interference with hydrogen bonding. PCBs are freely soluble in non-polar organic solvents, oils and biological lipids, and the shift from water to lipid solubility is linked to the degree of chlorination (Hutzinger *et al.*, 1974; Shiu & Mackay, 1986; ATSDR, 2000; IPCS, 2003).

The octanol/water partition coefficient (K_{ow}) is defined as the ratio of a chemical's concentration in the octanol phase to its concentration in the aqueous phase of a two-phase octanol/water system; values of K_{ow} are thus unitless (Table 1.3 and Table 1.6). The reported log K_{ow} values have been reviewed by Shiu & Mackay (1986). Fig. 1.3 shows the remarkable correlation between log K_{ow} (lipophilicity) and number of chlorine atoms (BZ numbers); log K_{ow} values ranged from 4.5 to 8.3. This partitioning plays a key role in environmental fate and transport. PCBs tend to favour the non-polar phase and will partition away from water to most solids, the organic portion being the preferred site (Erickson, 2001).

PCBs are characterized by Henry's law constants [a measure of the equilibrium distribution coefficient between air and water] that tend to decrease with a higher degree of chlorination. Less chlorinated PCB congeners have a considerably higher vapour pressure (1–2 Pa at 25 °C for monochlorobiphenyls) than the more highly chlorinated congeners (1.4×10^{-6} Pa for decachlorobiphenyl) (Shiu & Mackay, 1986). Therefore, the composition in air is dominated by the less chlorinated congeners and atropoisomers.

At high temperatures, PCBs are combustible, and the products of combustion include polychlorinated dibenzofurans (PCDFs) and hydrogen chloride, and polychlorinated dibenzodioxins (PCDDs) (IPCS, 1993; ATSDR, 2000).

Photochemical degradation may be one route for the breakdown of PCBs in the environment: photochemical experiments conducted under simulated natural conditions on several pure chlorobiphenyls and on commercial PCB products have indicated several degradative reactions, such as dechlorination, polymerization and solvolysis.

Table 1.6 Physical and chemical data for a selection of PCB congeners

PCB	No. of chlorine atoms	Melting point (°C)	Boiling point (°C)	Vapour pressure (10^{-6} kPa at 25 °C)	Log K_{ow}	Water solubility (µg/L)
PCB-1[a]	1	34	274	184	4.5	4830 (25 °C)
PCB-105	5	–	–	0.87	7.0	3.4 (25 °C)
PCB-118	5	–	–	1.20	7.1	13.4 (20 °C)
PCB-138	6	78.5–80	400[b]	0.53	6.5–7.4[b]	15.9[b]
PCB-153	6	103–104	–	0.05	6.7	0.9 (25 °C)
PCB-156	6	–	–	0.21	7.6	5.3 (20 °C)
PCB-163	6	–	–	0.08	7.2	1.2 (25 °C)
PCB-169	6	201–202	–	0.05	7.4	0.04–12.3[b]
PCB-180	7	109–110	240–280 (at 2.66 kPa)	0.13	6.7–7.2[b]	0.2 (25 °C)
PCB-183	7	83	–	–	8.3	4.9 (20 °C)

[a] Included based on its significantly different solubility and vapour pressure
[b] Calculated
K_{ow}, octanol/water partition coefficient; PCB, polychlorinated biphenyl
From Lindell (2012)

1.1.3 *Trade names and composition of commercial products*

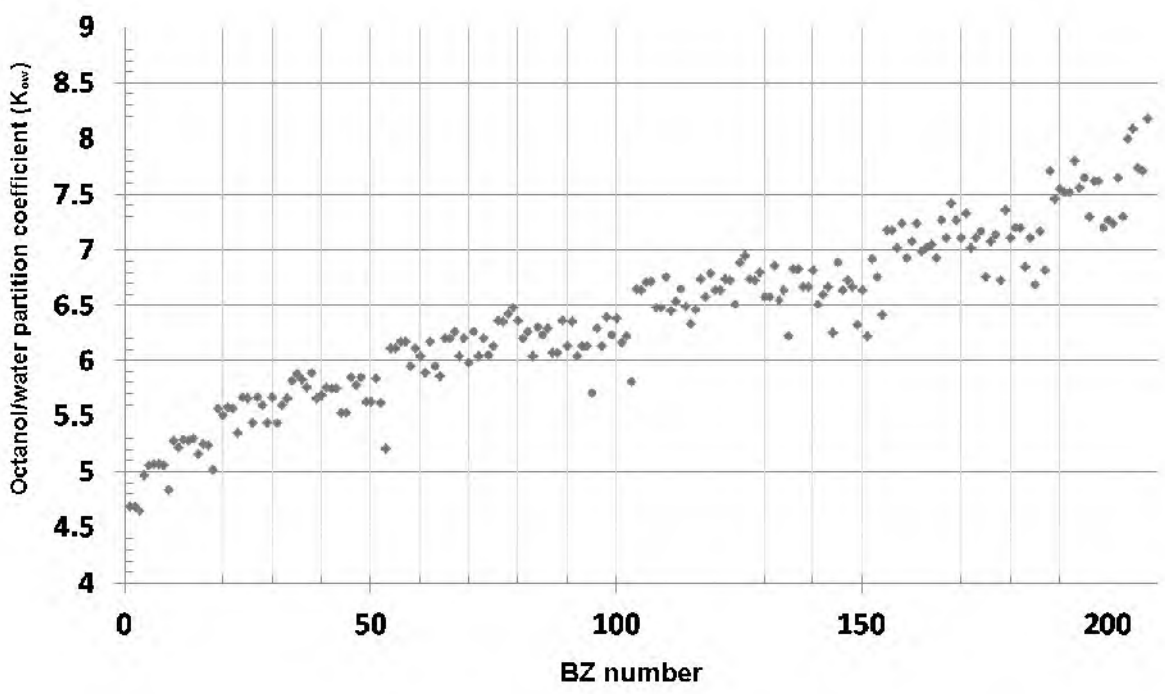
PCBs have never been used as single compounds, but rather as complex mixtures. The commercial products were manufactured to yield a certain degree of chlorination to fulfil technical requirements, generally between 21% and 68% chlorine.

Trade names for commercial products are given in Table 1.7. The most well known are Aroclor, Clophen, Phenochlor, Kanechlor, Pyralene, Fenclor, and Delor. The Aroclors, which were manufactured in the USA, are identified by a four-digit numbering code in which the first two digits indicate the type of mixture and the last two digits indicate the approximate chlorine content by percentage weight. Thus Aroclor 1242 is a chlorinated biphenyl mixture with an average chlorine content of 42%. The exception to this code is Aroclor 1016, which has an average chlorine content of 41% (Hutzinger *et al.*, 1974). Similarly, the Kanechlors are identified by a three-digit value indicating the average chlorine content (300 for 30%). Other products of similar chlorination content have been produced by different companies in Europe, Japan, and China.

Table 1.8 indicates equivalencies between main commercial formulations of PCBs. [The Working Group noted that these should be considered as approximate.] Since different production yield slight differences in the congener mixture, mixtures with comparable chlorine content but from different manufacturers (e.g. Aroclor 1260 and Clophen A60) show varying compositions, although with strong similarities (Johnson *et al.*, 2000).

The homologue composition of the commercial PCB products varies greatly according to chlorination degree achieved (Table 1.9). For example, Aroclor 1242 is a mixture of mono- to heptachlorobiphenyls, while Aroclor 1260 contains penta- to octachlorinated homologues. The concentrations of single congeners within each homologue group also differ between different products and batches (Fig. 1.4). About 130 of the 209 congeners have been identified in commercial formulations at concentrations above 0.05%. Generally, commercial PCB products consist of about 100–140 PCB congeners, with mono- and non-*ortho* substituted PCBs as minor or trace constituents (Frame *et al.*, 1996a, b; Johnson *et al.*, 2000).

Fig. 1.3 Octanol/water partition coefficient (K_{ow}) of PCB congeners according to the degree of chlorination (BZ number)



BZ, Ballschmiter and Zell; PCB, polychlorinated biphenyl
Compiled by the Working Group

An archetypal distribution of PCB congeners was detected in Aroclor 1254, lot 124–191 (corresponding to the historical G4 production process), while lot 6024 showed a profile characteristic of the A4 production process used between 1974 and 1976 (Kodavanti *et al.*, 2001). Indeed, Aroclor 1254 was produced by two different chlorination procedures (two-step versus single-step chlorination) (Frame *et al.*, 1996a, b). The differences in composition of the two lots are given in Table 1.10. Although Aroclor 1254 A4 probably represented less than 1% of the total production of Aroclor 1254, this PCB product was extensively used by standard suppliers and thus by researchers (Frame, 1999).

Chiral PCB congeners are important constituents of both technical and environmental mixtures of PCBs. For example, the total concentration of chiral PCB congeners in the commercial mixtures Aroclor 1242 and Aroclor 1260 is 6% and 30% by weight, respectively (Kania-Korwel *et al.*, 2007). Chiral enantiomers may have different biological and toxicological properties (Püttmann *et al.*, 1989; Rodman *et al.*, 1991). There is evidence that PCB atropisomers differ in their biological activities (Kania-Korwel *et al.*, 2006, 2008). They have been found in non-racemic proportions in many species (Lehmler *et al.*, 2010; Wong & Warner, 2009). While physical and chemical processes in the environment generally affect the two enantiomers of a known compound at the same rate, biological processes may result in the enrichment of one of the enantiomers, because of enantio-selective interactions with biological macromolecules (Buser & Mueller, 1993).

Table 1.7 Trade names for commercial PCB products[a, b]

Asbestol (trans, cap)	Hydol (trans, cap)
Askarel	Montar
Bakola 131 (trans, cap)	Nepolin
Biclor (cap)	No-Flamol (trans, cap)
Chlorextol (trans)	Phenoclor (trans, cap)
Chlorinol	Pydraul
Clophen (trans, cap)	Pyralene (trans, cap)
Clorphen (trans)	Pyranol (trans, cap)
Delor	Pyroclor (trans)
Duconol (cap)	Saf-T-Kuhl (trans, cap)
Dykanol (trans, cap)	Santotherm FR
EEC-18	Santovac 1 and 2
Elemex (trans, cap)	Siclonyl (cap)
Eucarel	Solvol (trans, cap)
Fenchlor (trans, cap)	Sovol
Elemex (trans, cap)	Therminol FR
Hivar (cap)	

[a] Each trade name may correspond to one or several products with varying chlorine content (see Table 1.8).
[b] Products may be used in transformers (trans) or capacitors (cap).
PCB, polychlorinated biphenyl
From IPCS (1993)

1.1.4 *Contaminants and impurities of commercial products*

Commercial PCB products have been reported to be contaminated with other chlorinated aromatic compounds, such as polychlorinated naphthalenes and PCDFs (IARC, 1978). Vos & Koeman (1970) were able to identify tetrachlorodibenzofurans, pentachlorodibenzofurans, and chlorinated naphthalenes in samples of Phenoclor DP-6 and Clophen A60. Bowes *et al.* (1975) examined samples of Aroclor 1248, 1254 and 1260 produced in 1969, samples of Aroclor 1254 from 1970 and Aroclor 1016 from 1972, and samples of Aroclor 1260, Phenoclor DP-6 and Clophen A60. They found PCDFs in all Aroclor preparations except Aroclor 1016, and in Clophen A60 and Phenoclor DP-6 (Table 1.11). The levels of PCDFs were in the low microgram per gram range (Erickson, 2001), but additional PCDFs may be formed from PCBs on heating. Impurities such as 2,3,7,8-tetrachlorodibenzofuran and 2,3,4,7,8-pentachlorodibenzofuran have been reported in different amounts under various manufacturing conditions in Aroclor 1248, Aroclor 1254, Clophen A-60, Phenoclor DP-6, and Kanechlor 400 (de Voogt & Brinkman, 1989). Rappe & Gara (1977) confirmed by capillary gas chromatography–mass spectrometry (GC–MS) that 2,3,7,8-tetrachlorodibenzofuran was one of the main PCDFs in "Yusho oil," as reported by Nagayama *et al.* (1976).

The proportion of impurities may vary between batches. For example, Aroclor 1254 with lot numbers 6024 and 124–191, which were produced by the same company by two different production processes, showed a 3.4-fold difference in the total concentration of PCDFs (Table 1.10).

It is important to note that PCDDs are not found in commercial PCB preparations (Erickson, 2001).

Overall, differences in composition as well as the presence of toxicologically relevant impurities may have had a significant impact on the results of toxicological studies with commercial

Table 1.8 Comparison of commercial PCB products based on percentage chlorination

Average number of chlorine atoms/ molecule	Range of chlorination (%)	Aroclor (USA)	Clophen (Germany)	Phenoclor (France)	Pyralene (France)	Kanechlor (Japan)	Fenoclor (Italy)	Delor (former Czechoslovakia)	PCB (China)
1.15	21	1221							
2	32–33	1232			2000	200			
2.5	38				1500				
3	40–42	1242, 1016	A30	CP3	3000	300	42	2; 103	PCB_3
4	48	1248	A40	DP4		400		3; 104	
5	52–54	1254	A50	DP5		500	54	4 and 5; 105	PCB_5
6–6.8	60–62	1260, 1262	A60	DP6		600	64	106	
8.7	68	1268					70		
10	71	1270					DK		

PCB, polychlorinated biphenyl
Adapted from de Voogt & Brinkman (1989), Erickson (1997), and Johnson *et al.* (2000)

Table 1.9 Homologue composition and physical properties of selected commercial PCB products

	Aroclor							Kanechlor		
	1221	**1232**	**1016**	**1242**	**1248**	**1254**	**1260**	**300**	**400**	**500**
Composition (%)										
Biphenyl	11	6	< 0.01	-	-	-	-	-	-	-
Monochlorobiphenyl	51	26	1	1	-	-	-	-	-	-
Dichlorobiphenyl	32	29	20	17	1	-	-	17	3	-
Trichlorobiphenyl	4	24	57	40	23	-	-	60	33	5
Tetrachlorobiphenyl	2	15	21	32	50	16	-	23	44	27
Pentachlorobiphenyl	0.5	0.5	1	10	20	60	12	0.6	16	55
Hexachlorobiphenyl	-	-	< 0.01	0.5	1	23	46	-	-	13
Heptachlorobiphenyl	-	-	-	-	-	1	36	-	-	-
Octachlorobiphenyl	-	-	-	-	-	-	6	-	-	-
Nonachlorobiphenyl	-	-	-	-	-	-	-	-	-	-
Properties										
Relative molecular mass	200.7	232.2	257.9	266.5		328.0	357.7			
Colour	Clear	Clear	Clear	Clear		Light yellow	Light yellow			
Density (g/cm^3 at 25 °C)	1.18	1.26 1.27	1.37	1.38	1.41 1.44	1.50 1.54	1.56 1.62			
Viscosity (cP at 38 °C)	5	8	20	24	70	700	Resin			
Physical state	Oil	Oil	Oil	Oil		Viscous liquid	Viscous liquid			
Boiling point (°C)	275–320	290–325	325–356	325–366		365–390	385–420			
Water solubility (µg/L at 25 °C)	200 15 000[a]	1450[a]	240 420	240	52 54	12	3			
Vapour pressure (10^{-6} kPa at 25 °C)	893	613	53	53	53	11	5.3			
Henry's law K (atm.m^3/mol, 25 °C)	3.5×10^{-3}		2.9×10^{-4}	5.2×10^{-4}		2.0×10^{-3}	4.6×10^{-3}			
Log K_{ow}[b]	2.8	3.2	4.4	4.1	6.1	6.5	6.9			
Flashpoint (°C)	141–150	152–154	170	176–180	193–196	None to boiling	None to boiling			

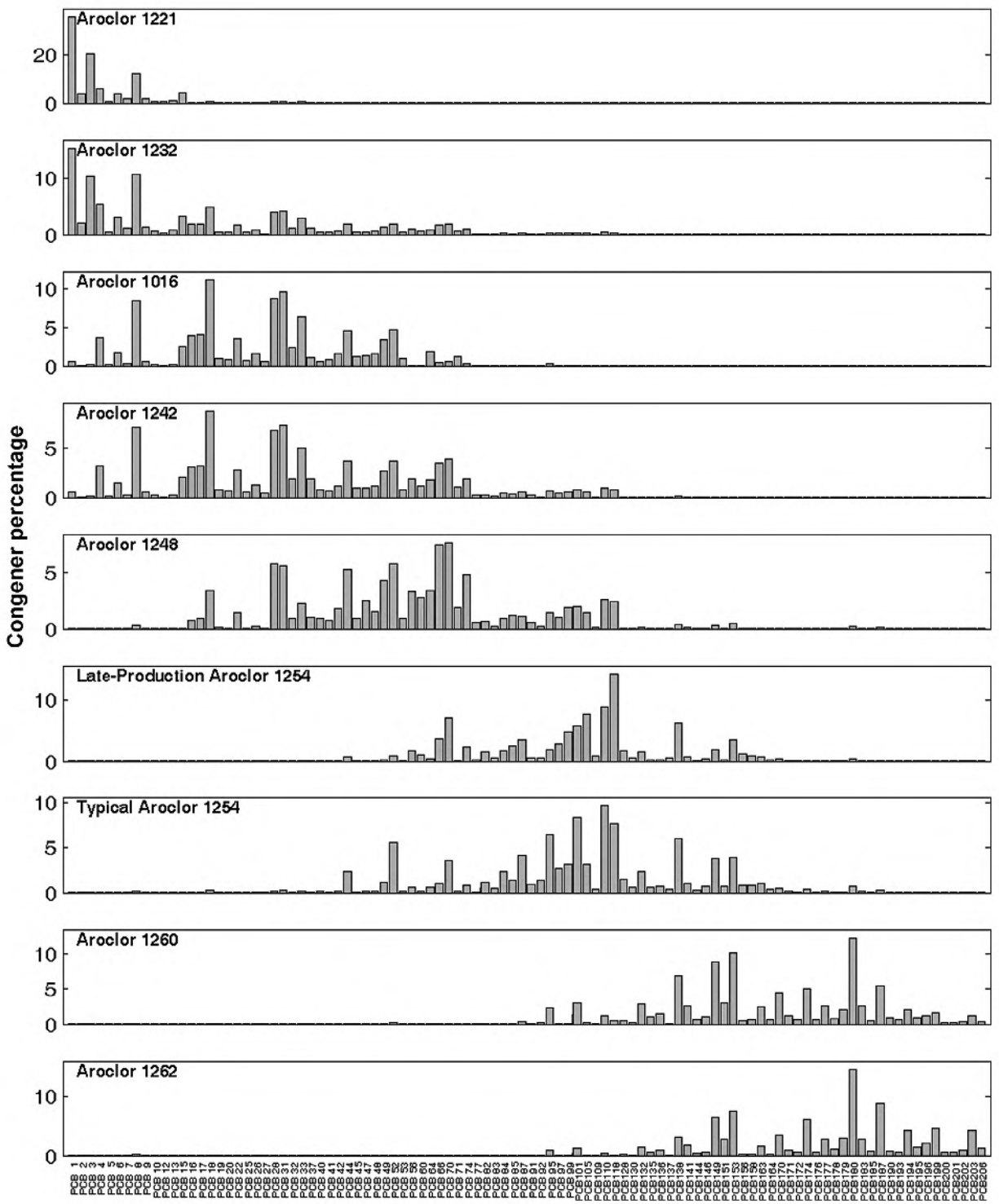
[a] Estimated value

[b] Log K_{ow} represents an average value for the major components of the Arochlor mixture. The Henry's law constants were estimated by dividing the vapour pressure by the water solubility (Cohen & Mercer, 1993; Erickson, 1997).

PCB, polychlorinated biphenyl

From Hutzinger *et al.* (1974), Pellet *et al.* (1993), and Lindell (2012).

Fig. 1.4 Congener-specific composition of Aroclor formulations



Only the 100 most abundant congeners are shown in this figure.
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Table 1.10 Chemical profile and impurities (polychlorodibenzodioxins, polychlorodibenzofurans and polychlorinated naphthalenes) in lots 124–191 and 6024 of Aroclor 1254

PCBs and impurities	Aroclor 1254	
	Lots 124–191 (G4 process)	Lot 6024 (A4 process)
Non-ortho *congeners*		
PCB-77	0.01 mg/g	27.2 mg/g
PCB-81	0.01 mg/g	0.28 mg/g
PCB-126	0.17 mg/g	3.24 mg/g
PCB-169	0.01 mg/g	0.02 mg/g
Mono-ortho *congeners*		
PCB-105	51.00 mg/g	130.00 mg/g
PCB-114	0.05 mg/g	0.78 mg/g
PCB-118	127.00 mg/g	124.00 mg/g
PCB-123	0.57 mg/g	2.14 mg/g
PCB-156	4.80 mg/g	51.00 mg/g
PCB-157	0.36 mg/g	26.30 mg/g
PCB-167	ND	ND
PCB-189	ND	ND
PCDFs		
2,3,7,8-TetraCDF	129.9 ng/g	350.1 ng/g
1,2,3,7,8-PentaCDF	295 ng/g	1920.2 ng/g
2,3,4,7,8-PentaCDF	821 ng/g	4049.2 ng/g
1,2,3,4,7,8-HexaCDF	1638.1 ng/g	4571.4 ng/g
1,2,3,6,7,8-HexaCDF	733.7 ng/g	3190.5 ng/g
1,2,3,7,8,9-HexaCDF	ND	ND
2,3,4,6,7,8-HexaCDF	213.3 ng/g	1333.3 ng/g
1,2,3,4,6,7,8-HeptaCDF	581.8 ng/g	1506.5 ng/g
1,2,3,4,7,8,9-HeptaCDF	533.3 ng/g	1459.4 ng/g
1,2,3,4,6,7,8,9-OctaCDF	356 ng/g	945.6 ng/g
Σ polychlorinated dibenzofurans (PCDF)	11.3 µg/g	38.7 µg/g
Σ polychlorinated dibenzo-p-dioxins (PCDD)	< 2 ng/g	< 2 ng/g
Σ polychlorinated naphtalenes	155 µg/g	171 µg/g
Σ non-*ortho* congeners-TEQ	17.3 µg WHO-TEQ/g	353 µg WHO-TEQ/g
Σ mono-*ortho* congeners-TEQ	5.51 µg WHO-TEQ/g	10 µg WHO-TEQ/g
Σ PCDF-TEQ	0.54 µg WHO-TEQ/g	2.25 µg WHO-TEQ/g
Total PCDD+PCDF+PCB-TEQ	23.4 µg WHO-TEQ/g	365.3 µg WHO-TEQ/g

CDF, chlorodibenzofuran; ND, not detected; PCB, polychlorinated biphenyl; PCDFs, polychlorodibenzofurans; TEQ, toxic equivalent
Adapted from Kodavanti *et al.* (2001) and EFSA (2005)

Table 1.11 Concentrations of chlorodibenzofurans in Aroclor, Clophen, and Phenoclor

Commercial PCB mixture (date of production)	Polychlorodibenzofurans (concentrations in mg/g)			
	Tetra-CDF	Penta-CDF	Hexa-CDF	Total
Aroclor 1248 (1969)	0.5	1.2	0.3	2.0
Aroclor 1254 (1969)	0.1	0.2	1.4	1.7
Aroclor 1254 (1970)	0.2	0.4	0.9	1.5
Aroclor 1260 (1969)	0.1	0.4	0.5	1.0
Aroclor 1260 (lot AK3)	0.2	0.3	0.3	0.8
Aroclor 1016 (1972)	< 0.001	< 0.001	< 0.001	-
Clophen A60	1.4	5.0	2.2	8.6
Phenoclor DP6	0.7	10.0	2.9	13.6

CDF, chlorodibenzofuran
Adapted from Bowes *et al.* (1975)

PCB products and mixtures (EFSA, 2005). Consistent interpretation of the results of such studies, especially differentiation of the effects caused by respective PCBs, may only be achieved if the congener composition of these mixtures is known. The determination of the content in specific congeners was not feasible in most cases due to the lower sensitivity of analytical techniques available in the past.

1.2 Analysis

1.2.1 General considerations

Past and current methods for the chemical analysis of PCBs have been reviewed recently (Le Bizec *et al.*, 2015). Since the 1960s, PCBs have been determined using GC techniques with electron capture detection (ECD), initially using packed columns. Today the separation has been improved by the use of capillary columns and the selectivity by the use of MS detectors. Increase in sensitivity, expressed as decreasing detection limits, has been achieved as analytical techniques have improved.

Originally, PCB concentrations were determined on the basis of commercial products, e.g. various Aroclor products with different chlorination levels. Later, PCB concentrations were determined based on homologue groups, while today congener-specific analysis is a common practice. These methodological changes, including differences in the basis of quantification, are an obstacle when comparing older with more recent studies.

Even when comparing studies from the same period, it can be difficult to compare PCB concentrations reported by different laboratories, if information on data quality is not available and if the results for different numbers of congeners are summarized. Often "total" PCB concentrations are reported, summing up all the congeners included in the laboratory's method and assumed to approach the true total PCB concentration. Operational sum parameters have been defined to harmonize congener lists and improve comparability, for example, the six indicator PCB congeners (PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, and PCB-180), expressed as PCB_6. The six congeners were not selected from a toxicological point of view, but were considered as indicators for the different PCB patterns in various sample types and are most suitable for evaluating non-dioxin-like PCBs (NDL-PCBs) (EFSA, 2005). This parameter is used, for example, in the European food and feed regulation (EC, 2011a).

Some agencies, such as the International Council for the Exploration of the Sea (ICES), recommend reporting PCB_7, which includes the

mono-*ortho* congener PCB-118 in addition to the PCB_6 (ICES, 2012; Webster *et al.*, 2013). In the Arctic Monitoring and Assessment Programme (AMAP), the sum of 10 PCB congeners is often reported (PCB-28, PCB-31, PCB-52, PCB-101, PCB-105, PCB-118, PCB-138, PCB-153, PCB-156, PCB-180), which includes the six indicator PCBs. However, reports of individual concentrations in scientific studies have the advantage of allowing sum calculations as required. In food analyses, PCB concentrations are preferred to compliance/non-compliance reports (EFSA, 2005).

Depending on the sample type and the purpose of the study, PCB concentrations may be reported in different units. Concentrations in solid samples are generally reported in mass per mass. Normalizations to dry weight or lipid weight are common for abiotic matrices (e.g. soil and sediment) and those with a high lipid content (e.g. fatty food products), respectively. For liquid and air samples, the concentrations are often given in mass per volume. However, as liquid volumes are susceptible to small changes during sample storage and cannot be determined as precisely as masses, concentrations in small liquid volume samples (e.g. blood) are increasingly related to mass instead of volume.

Apart from the adjustment of mass for fresh weight (also referred to as raw weight, wet weight), lipid normalization of PCB concentrations in blood samples is also common. (Schisterman *et al.*, 2005; Phillips *et al.*, 1989; Grimvall *et al.*, 1997). [The Working Group has acknowledged that a variety of lipid determination methods for blood are used and that there is no consensus on how to determine lipid concentrations.]

Given the low concentrations of PCBs in some matrices, reliable quality assurance and quality control are particularly important, including for example monitoring of recovery rates and procedural blanks, duplicate analyses, analyses of in-house reference material and external quality control in proficiency testing schemes.

The transport and storage of samples can be a source of error through PCB loss or contamination. Studying the effects of storage conditions on PCBs in biological material, De Boer & Smedes (1997) generally did not find temperature effects as long as the temperature was < 5 °C, or downward trends in PCB contents. Practical guidance on the storage and transport of marine samples intended for PCB analysis is given by OSPAR (1999, 2002) and Webster *et al.* (2013).

[The Working Group stressed the importance of how the "non-detects" were reported and treated in the data analysis. There are a variety of methods used and there is currently no global consensus.]

1.2.2 Analytical tools

Instrumental analysis is essentially identical for all matrices. Dioxin-like PCBs (DL-PCBs) are often analysed together with dioxins and furans by gas chromatography-high resolution mass spectrometry (GC-HRMS). For this purpose, DL-PCBs are separated from other PCB congeners as part of the clean-up and fractionation process, for example using activated carbon, porous graphite columns, or 2-(1-pyrenyl) ethyldimethylsilyated (PYE) silica (Hess *et al.*, 1995).

Gas chromatography-electron capture detection (GC-ECD) provides low detection limits and high precision, but is less specific than MS, as it separates PCB congeners only by retention time. MS adds a second dimension in terms of different mass spectra. Therefore, ^{13}C-labelled PCB congeners can be separated from the native molecule on a mass basis. In contrast, as retention times are identical to the native analogues, ^{13}C-labelled PCB congeners cannot be used in GC-ECD analyses.

Due to lower selectivity and the risk of interference, GC-ECD is often based on two GC capillary columns of different polarity (dual column GC) (Covaci & Schepens, 2001). Webster

et al. (2013) recommend that retention times be checked for shifts between analytical runs, usually with the help of characteristic peaks, for example those added as injection standards. Coelution of PCB-138 and PCB-163 occurs on many common capillary columns.

Among the MS techniques, electron capture negative ionization (ECNI) is very sensitive for detection of penta- to decachlorinated PCBs (Webster *et al.*, 2013). However, electron impact (EI) has better selectivity than ECNI and comparable sensitivity when combined with large-volume injection, which requires rigorous sample clean-up (Covaci *et al.*, 2002a). Suitable target and qualifier ions for PCBs are listed by Webster *et al.* (2013).

Some studies have applied gas chromatography-ion trap mass spectrometry (GC-ITMS), for example for the analysis of PCBs in human milk (Gómara *et al.*, 2011). GC-ITMS with its MS/MS option offers increased selectivity while being less expensive than HRMS (Webster *et al.*, 2013). Triple quadrupole mass spectrometry (LRMS/MS) operated in the selected reaction monitoring mode has also been shown to provide selectivity and sensitivity comparable to that of HRMS in food analyses (Ingelido *et al.*, 2012).

Bioassays are an alternative method of determining PCB concentrations and have been suggested as screening tools for monitoring PCDD/Fs and DL-PCBs in foodstuffs by the European Commission Directive 2002/69 (EC, 2002). The dioxin-responsive chemically activated luciferase (CALUX or lux) assay is mechanism-specific and uses the interaction with the aryl hydrocarbon (Ah) receptor. Differences between results of the bioassay and of the conventional targeted high resolution gas chromatography-high-resolution mass spectrometry (HRGC-HRMS) analysis of PCDD/Fs and DL-PCBs have been shown (van Leeuwen *et al.*, 2007), possibly caused by other compounds capable of interactions with the AhR (Vorkamp *et al.*, 2012).

Enzyme-linked immunosorbent assays (ELISA) have been successfully applied to PCB analyses in environmental samples, showing reasonable agreement with conventional GC analyses, but with a high dependence on sample pretreatment (Johnson & Van Emon, 1996; Deng *et al.*, 2002). Recent developments include, for example, immunosensors for applications in situ (Lin *et al.*, 2008) and immunoaffinity chromatography for sample purification (Van Emon & Chuang, 2013).

1.2.3 Analysis of environmental samples

Selected methods for analysis of PCBs in environmental matrices are presented in Table 1.12.

Supplementary material on analysis of PCBs in soil and sediment is available online at: http://monographs.iarc.fr/ENG/Monographs/vol107/suppl_S1.pdf.

(a) Air and dust

Both active and passive sampling are used for PCB analysis in air. Passive sampling has been applied to the analysis of outdoor air using semi-permeable membrane devices (Ockenden *et al.*, 2001) and polyurethane foam (Mari *et al.*, 2008). Vegetation is used as a natural passive sampler, for example tree bark integrating atmospheric PCB concentrations over the life time of the tree (Hermanson and Hites, 1990) or pine needles reflecting up to several years of PCB exposure (Kylin *et al.*, 1994).

Polyurethane foam has also been used for indoor air collection (Hazrati & Harrad, 2006), but active sampling is often the preferred method (EPA, 1999; Kohler *et al.*, 2005). To account for concentration differences and the limited air volume in an indoor setting, outdoor air is usually sampled by high-volume sampling, while low-volume sampling is used for indoor air.

Once retained on a solid matrix (filter, sorbent), PCBs are solvent-extracted using the same techniques as commonly applied for soil,

Table 1.12 Selected methods of analysis of PCBs in environmental matrices

Sample matrix	Sample preparation	Assay method	Detection limit[a]	Reference
Air	Collection on sorbent/filter, solvent extraction, evaporation, acid treatment and/or other clean-up (if necessary), separation of dioxin-like and non-dioxin-like PCBs if required.	GC-ECD; GC-HRMS	0.03–10 pg/m^3; 10 pg/m^3	McConnell *et al.* (1998), Mari *et al.* (2008)
Dust	Sieving of samples (during sampling or afterwards), solvent extraction, evaporation, acid treatment, back extraction, clean-up, evaporation.	GC-MS	NA	Harrad *et al.* (2009)
Water	Liquid-liquid extraction or SPE of unfiltered or filtered water, evaporation, clean-up if necessary. Alternative technique: Passive sampling	GC-ECD; GC-HRMS	0.22–3 ng/L; 0.004–0.5 ng/L	Hope *et al.* (1997), EPA (2008a)
Soil	(Water removal), extraction, evaporation, clean-up, including sulfur removal, separation of dioxin-like and non-dioxin-like PCBs if required.	GC-HRMS	1.5 ng/kg; 0.4–46 ng/kg	Wang *et al.* (2010), EPA (2008a)
Sediment	(Water removal), extraction, possibly in combination with sulfur removal, evaporation, clean-up, including sulfur removal, separation of dioxin-like and non-dioxin-like PCBs if required.	GC-ECD; GC-MS; GC-HRMS	NA; 0.4–46 ng/kg	Webster *et al.* (2013), EPA (2008a)

[a] Detection limits are given for individual PCB congeners

PCB, polychlorinated biphenyl; ECD, electron capture detection; EI, electron impact; GC, gas chromatography; HRMS, high-resolution mass spectrometry; MS, mass spectrometry; NA, not available; SPE, solid-phase extraction

sediment or biota. Before extraction, recovery/internal standards are added, e.g. PCB congeners that are not present in the environment, or ^{13}C-labelled PCB congeners. Extraction is often performed by Soxhlet (EPA, 1999; Menichini *et al.*, 2007). Ultrasonic extraction and pressurized liquid extraction (PLE) have also been described (Aydin *et al.*, 2007; Mari *et al.*, 2008). Barro *et al.* (2005) applied headspace-solid phase micro extraction, which does not involve solvents.

Whether or not clean-up steps are required depends on potential interferences from the matrix (e.g. particles) and co-extracted compounds as well as on expected concentrations. Adsorption chromatography can be applied, for example using alumina (Zhang *et al.*, 2011a) or silica. As all PCB congeners are acid stable, acid treatment is possible.

Dust for PCB analysis has been collected in several ways, for example from the residents' vacuum cleaner bags (Franzblau *et al.*, 2009; Knobeloch *et al.*, 2012), by vacuuming (Wilson *et al.*, 2001; Harrad *et al.*, 2009) and from air conditioning units (Tan *et al.*, 2007). Dust samples originating from vacuum bags might be sieved, but cut-off sizes differ, e.g. 150 µm (Wilson *et al.*, 2001) and 1 mm (Knobeloch *et al.*, 2012) have been described.

Extraction techniques are basically the same as described for sorbents, including Soxhlet extraction (Dirtu & Covaci, 2010), PLE (Harrad *et al.*, 2009) and ultrasonic extraction (Wilson *et al.*, 2001). Before extraction, internal/recovery standards should be added, as described for air samples. Due to interferences from the matrix and co-extraction of other compounds, clean-up of dust samples will be required. PLE can be combined with simultaneous clean-up by adding adsorption materials to the cells; however, additional clean-up steps may be necessary (Harrad *et al.*, 2009). Various sorbents have been used for clean-up of dust samples, including Florisil (Wilson *et al.*, 2001; Harrad *et al.*, 2009), silica gel (Dirtu & Covaci, 2010), and combinations of both (Knobeloch *et al.*, 2012). As described for air samples, acid treatment has also been applied (Harrad *et al.*, 2009).

(b) Water

The PCB content in a water sample is strongly influenced by the amount of suspended particulate matter (SPM) that adsorbs PCBs. Depending on the objectives of the analysis, different approaches can be chosen, resulting in different fractions to be analysed:

- Unfiltered water includes dissolved components and those bound to colloids and SPM.
- Filtered water gives PCB concentration on SPM (residue on the filter) and dissolved or bound to colloids (filtrate).
- Passive sampling targets the dissolved fraction.

Passive sampling devices integrate PCB concentrations over time, which reduces temporal variability. Common formats for water sampling include semipermeable membrane devices, low density polyethylene, and silicone rubber (Lohmann *et al.*, 2012). Passive sampling techniques have been applied for analysis of PCBs in river water (Grabic *et al.*, 2010) and seawater (Granmo *et al.*, 2000; Fernandez *et al.*, 2012).

In water bodies with a low SPM content, e.g. seawater, PCB concentrations will likely be low, and large amounts of water will have to be sampled and processed, while avoiding contamination. Guidelines for seawater sampling and the subsequent analysis of organic contaminants have been established by OSPAR (OSPAR, 2013). Studies have shown that the critical part of such analysis occurs outside the laboratory, i.e. during sampling, transport, and storage (Wolska *et al.*, 2005). As described for air and dust, recovery/internal standards should be added before extraction.

PCBs from water samples are typically extracted by either liquid–liquid extraction (LLE), i.e. the direct extraction of PCBs with a non-polar solvent (Hope *et al.*, 1997), or solid-phase extraction (SPE), where PCBs are retained on a solid phase and subsequently eluted with a non-polar solvent (Russo *et al.*, 1999). The United States Environmental Protection Agency (EPA) method 1668B for determination of PCBs in several matrices describes SPE, continuous LLE, and separatory funnel extraction as suitable extraction methods for aqueous samples (EPA, 2008a).

The amount of SPM in the sample is a critical factor, as LLE might be insufficient and SPE cartridges might become blocked by samples with a high SPM content (Erger *et al.*, 2012). Alternatively, SPM might be removed by filtration and analysed separately, for example by Soxhlet or ultrasonic extraction. This could be the method of choice for water samples with a high SPM content, for example wastewater samples or landfall leachate (Zorita & Mathiasson, 2005).

To what extent purification is necessary depends on the nature of the sample, its SPM content, PCB concentration and that of interfering compounds. Although sampling only freely dissolved PCBs, some passive sampling approaches add a clean-up step after extraction, for example using acid silica or aluminium (Grabic *et al.*, 2010). Surface-water samples, however, have often been analysed without clean-up (Hope *et al.*, 1997; Erger *et al.*, 2012), while other studies have included adsorption chromatographic steps (Khim *et al.*, 2001). Gel permeation chromatography (GPC) may be used for water extracts that contain organic compounds of high relative molecular mass (EPA, 2008a).

PCB exposure from snow can be considered insignificant, with the exception of polar regions where snow may be a source of drinking-water. Analytical methods are similar to those for water (Carrera *et al.*, 1998).

1.2.4 Analysis of biological samples

Several matrices have been analysed to determine internal exposure to PCBs, or body burden, including adipose tissue, meconium, placenta, blood, umbilical cord blood, human milk, and hair (Table 1.13).

(a) Tissues (adipose tissue and placenta)

The analytical methods applied to the analysis of PCBs in tissues such as adipose and placenta are similar to those used for environmental samples. The characteristically high lipid content of adipose and other tissues, however, requires rigorous lipid removal before instrumental analysis.

Different ways of sample pretreatment have been applied after or as part of the homogenization procedure, for example sample drying with Na_2SO_4 or hydromatrix (Covaci *et al.*, 2002a; Saito *et al.*, 2004), melting of fat (De Saeger *et al.*, 2005), mixing with base (Kim & Fisher, 2008) and addition of ethanol for protein precipitation (Whitcomb *et al.*, 2005).

Extraction is generally carried out with a non-polar solvent such as toluene or hexane, in some cases in a mixture with acetone, dichloromethane or propanol (Guvenius *et al.*, 2002; Saito *et al.*, 2004; Fernandez *et al.*, 2008). The extraction could often proceed by shaking or rotating, for example in an Ultra Turrex or Vortex (Guvenius *et al.*, 2002). Other extraction techniques are the same as those applied in environmental analyses, including ultrasonic extraction (Suzuki *et al.*, 2005), Soxhlet (Fernandez *et al.*, 2008), PLE (Saito *et al.*, 2004), and MAE (Li *et al.*, 2006). Supercritical fluid extraction with carbon dioxide (sometimes modified with dichloromethane) has also been applied (Stellman *et al.*, 1998). For the extraction of placenta, Gómara *et al.* (2012) additionally described the preparation of a suspension that was liquid–liquid extracted. As for other matrices, recovery/internal standards are generally added before extraction.

Table 1.13 Selected methods for analysis of PCBs in biological matrices

Sample matrix	Sample preparation	Assay method	Detection limit[a]	Reference
Adipose tissue	Pre-treatment (drying and/or protein denaturation), extraction, evaporation, lipid removal, further clean-up, separation of dioxin-like and non-dioxin-like PCBs if required.	GC-ECD; GC-HRMS	0.009–1.1 ng/g lipid; 0.002–0.2 ng/g lipid	Whitcomb *et al.* (2005), Fernandez *et al.* (2008)
Placenta	Pretreatment (drying and/or protein denaturation), extraction, evaporation, lipid removal, further clean-up, separation of dioxin-like and non-dioxin-like PCBs if required.	GC-ECD; GC-MS (ECNI)	NA	Gómara *et al.* (2012), Ma *et al.* (2012)
Blood	Protein denaturation, extraction, evaporation, lipid removal, further clean-up, separation of dioxin-like and non-dioxin-like PCBs if required.	GC-ECD; GC-MS; GC-HRMS	10–100 pg/mL; 2–5 pg/mL	Covaci & Schepens (2001), Lu *et al.* (2012)
Urine (hydroxylated PCBs)	Acidification, extraction, evaporation, derivatization.	GC-MS (EI)	0.02–0.04 ng/mL	Hong *et al.* (2005a, b)
Human milk	Drying or protein denaturation + fat globules dispersion, extraction, evaporation, lipid removal, further clean-up, separation of dioxin-like and non-dioxin-like PCBs if required.	GC-ECD; GC-MS; GC-HRMS	NA; 0.01–0.03 ng/mL; NA	Duarte-Davidson *et al.* (1991), Covaci *et al.* (2001), Fürst (2006)
Hair	Washing, incubation with HCl, extraction, evaporation, lipid removal, further clean-up.	GC-ECD; GC-MS (EI)	0.3–2 ng/g	Covaci *et al.* (2002b)

[a] Detection limits are given for individual PCB congeners

ECD, electron capture detection; ECNI, electron capture negative ionization; EI, electron impact; GC, gas chromatography; HCl, hydrochloric acid; HRMS, high-resolution mass spectrometry; MS, mass spectrometry; NA, not available

A common method for lipid removal is treatment of the sample with acid, usually sulfuric acid (Whitcomb *et al.*, 2005). GPC is another suitable method (Ma *et al.*, 2012), but may not achieve complete removal of lipids. The use of partially deactivated neutral aluminium for lipid removal has also been described (Stellman *et al.*, 1998).

For further clean-up of the extracts, the same techniques are applied as in the environmental analyses, either individually or in combinations. These include silica gel (Suzuki *et al.*, 2005; Fernandez *et al.*, 2008), alumina (Covaci *et al.*, 2002a), Florisil (Whitcomb *et al.*, 2005) and GPC (Saito *et al.*, 2004). Impregnating the silica gel with acid is a common way of combining adsorption chromatography with lipid removal (Covaci *et al.*, 2002a; Fernandez *et al.*, 2008).

Some studies have analysed PCB metabolites in adipose tissue and placenta, e.g. hydroxylated PCBs and methylsulfonyl-PCBs. These methods usually included a fractionation by adsorption chromatography and elution with different solvents (Guvenius *et al.*, 2002; Saito *et al.*, 2004). In the method by Gómara *et al.* (2012), hydroxylated PCBs were separated from the parent compounds during liquid-liquid extraction (LLE). After derivatization, the fraction containing hydroxylated PCBs was cleaned up in the same way as described for the parent compounds.

(b) Blood (including umbilical cord blood)

Numerous studies have analysed PCBs in blood, mostly in serum, but also in plasma (Schettgen *et al.*, 2011). The analytical methods used generally do not differ for serum and plasma. Given the low lipid content of blood, PCB concentrations are generally low and the sample amount available for analysis may be a challenge. Most studies work with volumes of 0.5–2 mL. Methods have recently been developed to extract PCBs from only 50 µL of plasma and from dried blood spots (Lu *et al.*, 2012).

Umbilical cord blood has often been analysed in combination with maternal blood, using the same methods. Given the lower lipid content and usually lower PCB concentrations in cord blood, adjustments of the sample intake might be useful; however, sample availability is usually the limiting factor.

Apart from the addition of internal standards, the first step in PCB analysis of serum, plasma or cord blood is generally the denaturation of protein, e.g. by addition of formic acid (Kang *et al.*, 2008), methanol (Korrick *et al.*, 2000), or acetonitrile (Agudo *et al.*, 2009). Different extraction techniques have been described, among which the simple mixing of the sample with solvent (Apostoli *et al.*, 2005; Schettgen *et al.*, 2011). LLE has also been used (Kawashiro *et al.*, 2008; Bachelet *et al.*, 2011) as well as SPE on C_{18} or hydrophilic–lipophilic balanced reversed phase sorbent (Covaci & Schepens, 2001; Lee *et al.*, 2011). Guvenius *et al.* (2003) used Lipidex 5000, a lipophilic gel, for extraction of PCBs from cord blood.

Since they are present at low concentration, lipids are not always removed from the extract (Lu *et al.*, 2012). Lipids can be removed by direct addition of acid to the extracts (Atuma & Aune, 1999) or by clean-up methods on acidified silica (Covaci & Schepens, 2001). Further clean-up sorbents include Florisil (Whitcomb *et al.*, 2005), alumina (Stellman *et al.*, 1998), neutral silica gel (Atuma & Aune, 1999), or combinations of these (Guvenius *et al.*, 2003; Apostoli *et al.*, 2005).

To account for the low concentrations of PCBs in blood, extracts are often reduced to very small volumes, e.g. 50 µL (Covaci & Schepens, 2001). This is achieved by addition of non-volatile keepers (Covaci & Schepens, 2001), or by evaporation to dryness (Apostoli *et al.*, 2005) and reconstitution in the desired solvent or a solution of syringe standards in this solvent. Evaporation to dryness carries the risk of loss of volatile PCB congeners.

Hydroxylated PCB metabolites have been analysed in blood and umbilical cord blood. Guvenius *et al.* (2003) used the same extraction method as for parent PCBs, but obtained hydroxylated PCBs in an isolated fraction, based on different elution solvents. Park *et al.* (2009) treated the sample with hydrochloric acid and 2-propanol, and extracted hydroxylated PCBs by LLE. Hydroxylated PCBs require derivatization to non-polar molecules before separation by GC (Sandau *et al.*, 2000).

(c) Urine

A few studies have assessed PCB metabolites (hydroxylated PCBs) in urine samples. Hydroxylated PCBs are more polar than their parent compounds and act as weak acids, which has to be taken into account in extraction, clean-up, and separation by GC.

Hong *et al.* (2005a, b) presented two methods for the extraction of hydroxylated PCBs from urine. The first method combined SPE testing of four different phases, with five derivatization methods. Best recoveries and GC separations were found for hydroxylated PCBs extracted on a C_2 phase and derivatized with iodopropane under basic conditions (Hong *et al.*, 2005a). The second method used headspace solid-phase microextraction and on-fibre derivatization, achieved by placing the needle in the headspace of a solution of bis(trimethylsilyl)trifluoroacetamide (BSTFA) (Hong *et al.*, 2005b). The derivatized hydroxylated PCBs were transferred to the GC injector by thermic desorption. Several fibre materials were tested, of which polydimethylsiloxane-divinylbenzene (PDMS-DVB) gave the highest signal in the analysis.

(d) Human milk

Breast milk is the most extensively analysed matrix for the estimation of PCB body burden in humans. The first studies date back to the 1970s (Musial *et al.*, 1974), and programmes for the biomonitoring of human milk have been established in several countries or regions (Wilhelm *et al.*, 2007; Krauthacker *et al.*, 2009; Cerná *et al.*, 2012). Analytical methods are very diverse: the milk samples may be treated as liquid, or the lipid phase may be isolated, or the sample may be freeze-dried and treated as solid.

When the whole milk sample is treated as a liquid, the first steps usually include protein denaturation and dispersion of fat globules by addition of sodium oxalate, or acetic acid and methanol, sometimes in combination with ultrasound treatment (Dmitrovic & Chan, 2002; Fürst, 2006). Before or after this step, internal standards are usually added, and the sample is extracted by LLE (Chovancová *et al.*, 2011), or SPE (Covaci *et al.*, 2001; Dmitrovic & Chan, 2002). Hexane is a commonly used solvent, although a large variety of solvent combinations and solvent sequences have been described in the literature.

In some studies, the lipid phase of the milk sample is separated or extracted and a defined amount of fat used for further analysis (Fürst, 2006; Pérez *et al.*, 2012).

In the third approach, milk samples are freeze-dried and a defined amount is extracted with techniques commonly applied to solid samples, e.g. Soxhlet extraction (Duarte-Davidson *et al.*, 1991) and PLE (She *et al.*, 2007). Matrix solid-phase dispersion has also been described (Gómara *et al.*, 2011). However, freeze-drying always runs the risk of loss of volatile PCBs and cross-contamination.

As described for other human matrices, lipids in the extract are removed before instrumental analysis. Furthermore, the extracts usually contain co-extracted compounds that are likely to interfere with PCBs in the instrumental analysis. The clean-up techniques therefore generally include lipid destruction by acid treatment, either directly in the extract (Duarte-Davidson *et al.*, 1991), or by acidified silica gel (Covaci *et al.*, 2001). Alternatively, GPC has been used, but usually in combination with acid treatment (She *et al.*, 2007). Further clean-up techniques include

adsorption chromatography on neutral or basic silica (She *et al.*, 2007), alumina (Chovancová *et al.*, 2011), and Florisil (Pérez *et al.*, 2012), also in combinations (Fürst, 2006). Ingelido *et al.* (2007) described clean-up by supercritical fluid extraction.

(e) Human hair

With a lipid content of about 2% (Altshul *et al.*, 2004), hair accumulates lipophilic compounds such as PCBs and has the advantage of being sampled non-invasively. However, to what extent hair PCB content reflects internal exposure to PCBs is difficult to determine, even if the hair is washed before analysis to avoid co-extraction of dust particles. Comparisons of serum and hair samples showed weak correlations for most PCB congeners and considerably higher PCB concentrations in hair, also on a lipid-normalized basis (Altshul *et al.*, 2004). Effects of hair colour (natural or dyed) cannot be ruled out (Covaci *et al.*, 2002b).

Sample amounts of less than 1 g are sufficient for detection of PCBs. The hair samples are washed, and cut or pulverized, and then spiked with internal or recovery standards and incubated with hydrochloric acid (Covaci *et al.*, 2002b). Extraction techniques applied in hair analyses include LLE (Covaci *et al.*, 2002b), Soxhlet (Zhang *et al.*, 2007), and ultrasonic extraction (Barbounis *et al.*, 2012). The same methods for lipid removal and extraction clean-up as for other biological matrices have been used, e.g. adsorption chromatography on acidified silica gel, alumina (Covaci *et al.*, 2002b), and Florisil (Zhang *et al.*, 2007). A comparison between three laboratories analysing the same hair sample but using different internal standards, extraction techniques and analytical instruments (GC-ECD, GC-LRMS and GC-HRMS) showed good agreement, with a relative standard deviation of 15% (Gill *et al.*, 2004).

1.2.5 Analysis of food samples

Food items are regularly analysed for PCBs in various national and international food-monitoring programmes (Fromberg *et al.*, 2011; EFSA, 2005), and market-basket or duplicate-diet studies have been performed to identify PCB intake from food (Voorspoels *et al.*, 2008; Fromme *et al.*, 2009).

These studies have often applied methods that are sufficiently versatile to allow analysis of different kinds of food item with varying lipid content and consistency. The first step is often a drying of the food material with sodium sulfate (Voorspoels *et al.*, 2008; Schecter *et al.*, 2010), followed by the addition of recovery or internal standards, and Soxhlet extraction using hexane:acetone (Voorspoels *et al.*, 2008), or toluene (Kiviranta *et al.*, 2004). The clean-up usually includes lipid removal by acid treatment, either as direct addition to the extracts (Fromme *et al.*, 2009), or via acid-impregnated silica gel (Voorspoels *et al.*, 2008). Further clean-up steps can include neutral and basic silica gel (Son *et al.*, 2012), alumina (Kiviranta *et al.*, 2004), and Florisil (Schecter *et al.*, 2010); however, the extent of purification and fractionation is highly dependent on the target analytes.

Food monitoring sometimes focuses on DL-PCBs, which are analysed together with dioxins and furans. These are separated from other PCB congeners by fractionation on a carbon column, which separates the molecules by planarity (Fernandes *et al.*, 2004). Given the low concentrations of DL-PCBs, the fractions are sometimes further purified before instrumental analysis (Fromme *et al.*, 2009).

Some studies have used more specific methods for different food items, for example, protein denaturation and dispersion of fat globules in dairy products, by the addition of sodium oxalate, or potassium oxalate and ethanol (Fromberg *et al.*, 2011; Sirot *et al.*, 2012), followed by LLE. In other studies using cows' milk, the samples

are freeze-dried before extraction (Lake *et al.*, 2013), or the fat is separated using a detergent (Pérez *et al.*, 2012). The clean-up steps may be the same as for other lipid-containing matrices. For the analysis of butter and vegetable oil, Roszko *et al.* (2012) described a dialysis method based on low-density polyethylene semi-permeable membranes, followed by GPC and common column clean-up.

Numerous studies have dealt with analysis of PCBs in fish, as summarized by Domingo & Bocio (2007). Analyses of meat and fish basically follow the same methods (Su *et al.*, 2012). Samples are often dried as the first step, e.g. by freeze-drying (Abalos *et al.*, 2010; Liu *et al.*, 2011) or addition of anhydrous sodium sulfate (de Boer *et al.*, 2010). After addition of internal standards, the samples are extracted on a Soxhlet apparatus (Su *et al.*, 2012), by PLE (Pérez-Fuentetaja *et al.*, 2010), or ultrasonic extraction (Son *et al.*, 2012). The clean-up techniques are the same as described for other food matrices, including acid treatment (Su *et al.*, 2012), acid and neutral silica gel, and alumina (Liu *et al.*, 2011), and Florisil (Villa *et al.*, 2011), sometimes in an automated PowerPrep system (Abalos *et al.*, 2010). A rapid extraction and purification method was presented by Kalachova *et al.* (2011), combining PCB partitioning into ethyl acetate and lipid removal on a silica gel microcolumn.

Eggs are commonly analysed for PCBs, with a focus on the egg yolk (Kiviranta *et al.*, 2004; Voorspoels *et al.*, 2008). While the same methods could be applied as for other food samples, recent publications have only equilibrated the sample with solvents (Fromberg *et al.*, 2011; Rawn *et al.*, 2012). The clean-up steps include lipid removal by direct acid treatment and adsorption chromatography on acid silica and Florisil (Rawn *et al.*, 2012).

Fruit and vegetables are analysed less frequently than lipid-rich food items. In the methods described by Grassi *et al.* (2010) and Sirot *et al.* (2012), freeze-drying, extraction using Soxhlet or PLE, and acid treatment were applied, in a manner very similar to that used for analyses of other food items.

1.3 Production and uses

1.3.1 Production processes

PCBs have commonly been synthesized commercially by catalytic chlorination of biphenyl. The catalysts used include iron, iodine, and chlorides of aluminium, tin, and antimony. The synthesis is performed as a one-step chlorination process, or in two steps with further chlorination of residues from the first step. The crude products are purified by alkali wash to remove hydrogen chloride and ferric chloride, blown with air, and sometimes also by distillation (IARC, 1978). The degree of chlorination is controlled by the time (range, 12–36 hours) in the reactor.

The manufacturing process for Aroclors involved the chlorination of biphenyl with anhydrous chlorine in the presence of a catalyst, such as iron filings or ferric chloride. In 1974–1977, "late production" Aroclor 1254 was made by a two-stage chlorination procedure. In the first stage, biphenyl was chlorinated to 42% chlorine content by weight as for Aroclor 1242. This was then fractionated to give a distillate (Aroclor 1016). The residue (mostly mono-*ortho* tetrachlorobiphenyls and higher homologues) was further chlorinated to 54% chlorine by weight, resulting in a lot (Monsanto lot KI-02–6024) with markedly higher levels of the high non-*ortho* and mono-*ortho* PCB congeners than the Aroclor 1254 lots produced earlier. The differences between the early and late lots of Aroclor 1254 are discussed in more detail above (see Section 1.1.3 and Table 1.10).

1.3.2 Production volumes and trends

Although the commercial production of PCBs began in the 1920s, it was not until after 1945 that production reached substantial volumes. Production peaked in the 1960s and 1970s, and had ceased in most countries by the end of the 1970s or early 1980s.

Estimates of the total cumulative worldwide production of PCBs indicate that 1 to 1.5 million tonnes (or more) of commercial PCB products were manufactured. Production volumes from former Czechoslovakia, France, Germany, Italy, Japan, China, Poland, the Russian Federation and the former Soviet Union, Spain, the United Kingdom, and the USA, as reported by Tatsukawa (1976), de Voogt & Brinkman (1989), Jiang *et al.* (1997), AMAP (2000), Holoubek *et al.* (2001a), and Sułkowski *et al.* (2003), add up to around 1 325 000 tonnes for 1930–1993 (Table 1.14).

In the USA, annual production peaked in 1970 with a total volume of 39 000 tonnes. From 1957 to 1971, 12 different types of Aroclor with chlorine contents ranging from 21% to 68% were produced in the USA by Monsanto Chemicals Co. (see Section 1.1). In addition, Geneva Industries produced a smaller amount of PCBs from 1972 to 1974 (EPA, 2008b).

In China, the production of PCBs began in 1965 and was gradually stopped between 1974 and the 1980s. According to preliminary investigation and analysis, 7000–10 000 tonnes of PCBs were produced in China from 1965 to 1974, with 9000 tonnes as PCB_3 [similar to Aroclor 1242] and 1000 tonnes as PCB_5 [similar to Aroclor 1254] (Xing *et al.*, 2005; NIP China, 2007).

Information from the Democratic People's Republic of Korea (NIP Korea DPR, 2008) indicated that production of PCBs has been ongoing at two sites since the 1960s. The initial production capacity for PCBs was 1200 tonnes per year, with a tendency to increase until the 1980s; however, capacity has decreased since the early 1990s, and the average annual production volume in 2001–2006 was 411.6 tonnes. The total amount produced up to 2006 could be estimated at around 30 000 tonnes. According to this report, the Democratic People's Republic of Korea planned to reconsider its production of PCBs in 2012.

The commercial products were marketed under more than one hundred different trade names, depending on place of manufacture, production process, and chlorine content. Aroclors comprised at least 10 different commercial PCB products, under the names Aroclor 1016, 1221, 1232, 1242, 1248, 1254, 1260, 1262, 1268, and 1270. It should be noted that Aroclor 5460 was not a PCB product, but consisted of polychlorinated terphenyls. Other commercial PCB products include Clophen (four products), Delor (three products), Fenclor (five products), Kanechlor (five products), Phenochlor (four products), Pyralene (three products), Sovol, and Therminol (see Section 1.1.3).

1.3.3 Uses

Due to the physical and chemical properties of PCBs, such as non-flammability, chemical stability, high boiling point, and high dielectric constants, PCBs were widely used in several industrial and commercial open and closed applications (Table 1.15). PCBs have also been used in corresponding military applications, but detailed information on military use is typically very scarce.

As a result of the production process, PCBs were never used as individual congeners, but as technical products composed of multiple congeners. The commercial PCB products were generally used as such, but mixtures with other compounds were also produced to obtain specific properties. For example, the PCB product Sovol may have been mixed with α-nitronaphtalene to increase volatility, and sold as Nitrosovol (UNEP, 1988). Similarly, Galbestos was a mixture of PCBs and asbestos used on galvanized steel and

Table 1.14 Volume and duration of PCB production in countries with known production (by production volume)

Producer	Country	Duration		Volume (tonnes)	Reference
		Start	Stop		
Monsanto	USA	1930	1977	641 246	de Voogt & Brinkman (1989)
Bayer AG	Germany, western	1930	1983	159 062	de Voogt & Brinkman (1989)
Orgsteklo	Russian Federation	1939	1990	141 800	AMAP (2000)
Prodelec	France	1930	1984	134 654	de Voogt & Brinkman (1989)
Monsanto	United Kingdom	1954	1977	66 542	de Voogt & Brinkman (1989)
Kanegafuchi	Japan	1954	1972	56 326	Tatsukawa (1976)
Orgsintez	Russian Federation	1972	1993	32 000	AMAP (2000)
Caffaro	Italy	1958	1983	31 092	de Voogt & Brinkman (1989)
2.8 Vinalon and the Sunchon Vinalon Complex	Democratic Republic of Korea	1960[a]	2012[b]	30 000[c]	NIP Korea DPR (2008)
SA Cros	Spain	1955	1984	29 012	de Voogt & Brinkman (1989)
Chemko	Former Czechoslovakia	1959	1984	21 482	Schlosserová (1994)
Xi'an	China	1965	1980	10 000	Jiang *et al.* (1997), NIP China (2007)
Mitsubishi	Japan	1969	1972	2 461	Tatsukawa (1976)
Electrochemical Co.	Poland	1966	1970	1 000	Sułkowski *et al.* (2003)
Zaklady Azotowe Tarnow-Moscice	Poland	1974	1977	679	Sułkowski *et al.* (2003)
Geneva Industries	USA	1972	1974	454	EPA (2008b)
Total		*1930*	*2012*	*1 355 810*	

[a] During the 1960s
[b] "The Ministry of Chemical Industry will, by 2012, take measures to dismantle the PCBs production process and establish a new process of producing an alternative."
[c] Estimated from Republic of Korea 2008, National Implementation Plan for the Stockholm Convention on Persistent Organic Pollutants.
PCB, polychlorinated biphenyl
Adapted from Breivik *et al.* (2007)

galvanized corrugated sliding panels in various industrial and military applications.

(a) Closed applications

The predominant applications for PCBs were in dielectric fluids in capacitors and transformers. These applications are considered to be closed applications, since PCBs are not expected to leak out of the system. However, transformers had occasionally to be topped up with PCBs so that these systems were not completely closed.

While applications in hydraulic and heat transfer, and cooling systems are also usually considered to be closed applications, there have been reports of accidental leaks from such systems, and thus these applications are often referred to as "normally closed."

During the 1960s, dielectric fluid in capacitors and transformers represented 50–60% of the sales of PCBs in the USA (IARC, 1978). In 1972, Monsanto restricted its sale of PCBs to capacitor and transformer applications (Erickson, 2001); after this date, these applications represented some 99% of the total use of PCBs in the USA (Durfee *et al.*, 1976). In China, PCB_3 [similar to Aroclor 1242] was used primarily in power capacitors applied in electricity production, distribution and transmission, while PCB_5 [similar to Aroclor 1254] was used mainly as a paint additive (see Table 1.8).

Table 1.15 Industrial uses of PCBs

System/category	Aroclor									DecaCB
	1221	1232	1016	1242	1248	1254	1260	1262	1268	
Dielectric fluids										
Capacitors	✓		✓+	✓+		✓				
Transformers			✓	✓		✓+	✓+			
Hydraulic/lubricants/heat-transfer fluids										
Heat transfer				✓	✓	✓				
Hydraulic fluids		✓		✓	✓	✓	✓			
Vacuum pumps					✓	✓	✓			
Gas transmission turbines	✓			✓						
Immersion oil for microscopes							✓			✓
PCBs incorporated into products and materials										
Rubber	✓	✓		✓+	✓	✓			✓	
Synthetic resins					✓	✓	✓	✓	✓	
Carbonless copy paper				✓+						
Pipeline valve grease									✓	
Adhesives	✓	✓		✓+	✓	✓				
Wax extenders				✓+		✓			✓	
Caulk and joint sealants						✓[a]				
Insulation and other building materials						✓			✓	
De-dusting agents						✓	✓			
Inks						✓				
Cutting oils						✓				
Wire and cable coatings						✓	✓			
Die or investment casting										✓
Pesticide extenders						✓				

[a] Also others
✓ Denotes use of given Aroclor in a specific end-use
✓+ Denotes principal use
PCB, polychlorinated biphenyl
Adapted from Johnson *et al.* (2000) and Erickson & Kaley (2011)

As production and use of PCBs became banned, outdated PCB-containing equipment (equipment filled with PCBs as dielectric fluid) was generally removed from use and stored for disposal (Xing *et al.* 2005). In this equipment, about 6000 tonnes of PCBs came from capacitors (NIP China, 2007).

(b) Open applications

PCBs were also used in several open applications as a major constituent of permanent elastic sealants and as flame-retardant coatings (Heinzow *et al.*, 2007).

The use as plasticizer in sealants (caulking material) and flooring material was common in many countries, representing up to 15–20% of the total use of PCBs in Sweden (Jansson *et al.* 1997). The sealants were mainly used in outdoor applications, but indoor use was not uncommon. Use in flooring material was limited to indoor use.

Sealants that were mixed with PCBs were mainly of the polysulfide type. The mixing was often performed on site. Information on concentrations to be used were not available to the Working Group; however, from a technical point of view, PCB concentrations were likely to be above 5%. Sealants analysed some 40 years after application often contained concentrations of PCBs of 5–15%, with concentrations of up to 35% being reported. The concentration may vary not only between sites, but also within a building. These variations may be the result of use of sealants with different PCB content, or of secondary processes, such as migration out of the matrix. There are reports indicating that inner parts of sealants could contain higher concentrations than the superficial parts (Johansson *et al.*, 2003).

In addition to the use as sealants and flame-retardant coatings, PCBs have also been used in other open applications, such as in inks, adhesives, microencapsulation of dyes for carbonless duplicating paper, conveyor belts, rubber products, paints, pesticide fillers, plasticizers, polyolefin catalyst carriers, immersion oil for microscopes, cutting and lubricating oils, surface coatings, wire insulators, and metal coatings (ATSDR, 2000; Erickson, 2001; Erickson & Kaley, 2011). Also, use in small ballasts for fluorescent lights could be regarded as an open application, especially after long-lasting usage.

(c) Disposal of equipment containing PCBs

Improper handling of electronic waste (e-waste) has been identified as a source of environmental contamination with PCBs, especially for old equipment (Leung *et al.*, 2006). Dismantling of ships has also been identified as a potentially important source of occupational exposure to and environmental contamination with PCBs (Basel Convention, 2003).

With the complete ban on the use of PCBs, stockpiles awaiting elimination have successively appeared in many countries.

In 2000, 23 companies worldwide had facilities for the disposal of equipment containing PCBs, of which 11 were in Europe. The use of solvent for decontamination represents the most common procedure of disposal, followed by destruction by incineration, dechlorination with sodium, retrofilling and vitrification. The most common technology used for destruction of PCBs is by incineration, with an efficiency of between 99% and 99.99999% (IOMC, 1998). For exemple, France has an installed capacity for incineration of PCB residues amounting to around 20 000 tonnes per year (INERIS, 2013).

1.4 Environmental occurrence and exposure

PCBs are found worldwide at measurable levels in all environmental media (soils and sediments, water, air), in wildlife, and also probably in the body of every human. Human exposure to PCBs occurs mostly via ingestion of

contaminated food (see Section 1.4.7), but also via inhalation and dermal absorption.

Soils are natural sinks for persistent and lipophilic compounds such as PCBs; PCBs are absorbed by the organic carbon of the soil, and once absorbed they are relatively persistent (Buckley-Golder, 1999) (see Section 1.4.5). PCBs enter the soil via different pathways: industrial releases from manufacture, use and disposal, accidental releases, atmospheric deposition, application of sewage sludge, and erosion and leachate from nearby contaminated areas. PCBs in organic liquids may be dissolved by soils and then migrate with the solvent.

The congener patterns of PCBs in soils and sediments change over time as a result of the activity of aerobic bacteria (that degrade less chlorinated congeners) and anaerobic bacteria (that can cause partial dechlorination of more highly chlorinated congeners) (Hardell *et al.*, 2010). The patterns found in environmental biota are often referred to as "weathered," since they result from alterations in the composition of a mixture (e.g. resulting from bio accumulative and metabolic processes in higher biological organisms and through bacterial action, exposure to ultraviolet radiation, etc.). "Weathering" processes result in PCB patterns with either a higher chlorinated fraction or congeners with higher bioaccumulative properties compared with the commercial products. "Weathering" must be considered when assessing PCB-associated risks based on studies with experimental animals exposed to commercial PCB products.

Water is a major pathway for migration of PCBs, both in solution and particulate-bound, although PCBs are lipophilic and generally not very soluble in water (see Section 1.4.6). Less chlorinated PCB congeners have greater solubility than more highly chlorinated congeners.

Air is another major pathway for PCB migration (see Sections 1.4.3 and 1.4.4). PCBs are semi-volatile compounds and, as with water solubility, less chlorinated congeners are more volatile than more highly chlorinated ones (Totten *et al.*, 2006). There is extensive evidence that PCBs in aquatic systems exchange with PCBs in air (Bamford *et al.*, 2002). Air transport of PCBs can occur in either the vapour phase or particulate-bound, thus contributing to global pollution and PCB contamination of remote regions of the earth. PCBs in air come from several direct or indirect sources, including industrial facilities, military sites, contaminated bodies of water, landfills and hazardous waste sites, electric arc furnaces, incineration and other forms of combustion, sewage sludge applied to agricultural lands, and construction materials, including in paints (Hu & Hornbuckle, 2010), caulking, light ballasts, floor sealants, and adhesives and plasticizers in older buildings (Wallace *et al.*, 1996).

PCBs from soil, sediment, air and water enter the food-chain by uptake and bioaccumulation in plants and animal fats. There is significant biological magnification of PCB concentration as PCBs move up the food-chain. PCB concentrations vary depending on the degree of bioaccumulation, and are usually highest in carnivorous fish coming from contaminated waters. PCBs are found in the fat of all meat animals, in all dairy products containing fat, and in eggs (ATSDR, 2000; IOM, 2003), sometimes at high concentrations due to local contamination of grasses, and feeding practices in some countries (see Section 1.4.2). Also, it is not uncommon to feed domestic animals with fish meal or oil, or waste animal fats, which results in recycling of PCBs (IOM, 2003). For example, farmed salmon fed with concentrated fish meal or fish oil containing significant amounts of PCBs showed elevated concentrations of PCBs (Hites *et al.*, 2004). PCBs found in food are typically of higher chlorination, since they are less volatile and more biologically persistent in plants and animals than the lower congeners.

Another important route of exposure to PCBs is inhalation; however, it is difficult to determine the relative contribution of inhalation

compared with ingestion. Harrad *et al.* (2006) have suggested that inhalation may account for 4–63% (median, 15%) of overall exposure in humans. PCBs may be attached to indoor dust, which can be either ingested or inhaled. Individuals who spend significant periods of time in the presence of either outdoor or indoor vapour-phase PCBs will have continuous exposure that is not reflected in measurements of "total" PCBs, because the less chlorinated congeners are more rapidly metabolized and excreted by the human body (Fig. 1.5; Johansson *et al.*, 2003). Concentrations of different PCB congeners were measured in blood from individuals living in houses where PCB-containing sealant was used. Concentrations of most congeners were only slightly elevated (1.2 to 3.2 times), but the two congeners with a low level of chlorination (PCB-28 and PCB-66) were detected at much higher concentrations (30 and 9 times, respectively) in contaminated flats than in control flats.

Dermal absorption of PCBs may occur primarily in the occupational setting, but also through contact with contaminated sediments or other applications to the skin (Wester *et al.*, 1987, 1993). Less chlorinated congeners are more rapidly absorbed through the skin than more highly chlorinated congeners (Garner & Matthews, 1998).

Congener patterns in the general human population are always different from any pattern found in commercial PCB products (Patterson *et al.*, 2009). The factors that may explain this are:

- The general public is exposed to multiple sources of PCBs, only rarely to a single commercial product.
- There may be more than one route of exposure for almost all matrices/animals/humans.
- Dechlorination occurs to varying degrees in sediments, soils, water and air. Commercial PCB products will volatize to some degree, and in doing so, will lose less chlorinated congeners.
- PCBs ingested by fish and animals will be metabolized (to less chlorinated and hydroxylated congeners) to different degrees. Thus most food stuffs will demonstrate a shift in the congener profile compared to the commercial product.
- When inhalation is the major route of exposure, there is selective exposure to the more volatile, less chlorinated and less persistent congeners.
- Genetic differences among individuals may confer differences in metabolic activity and selective metabolism of different congeners.

1.4.1 Diffuse sources of PCBs worldwide

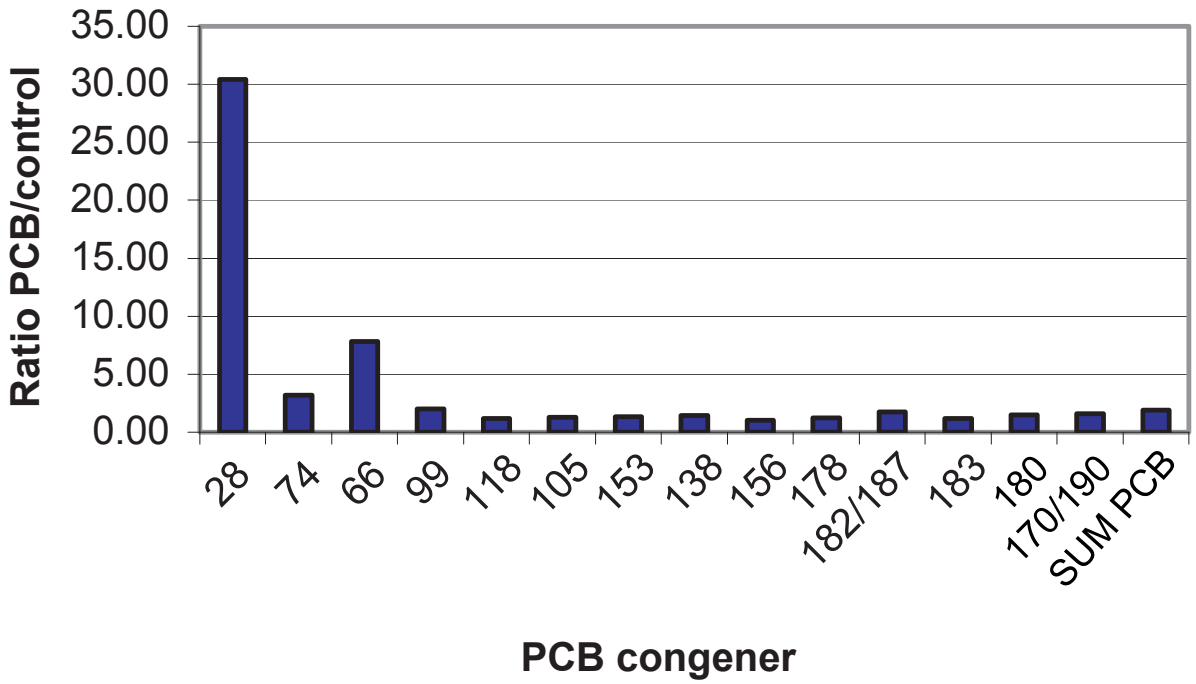
(a) North America

The two Monsanto facilities that manufactured PCBs in the USA were located in Anniston, Alabama, and Sauget, Illinois. In Anniston, more than 400 000 tonnes of PCBs were produced, at least 4550 tonnes were discarded in two landfills, and at least 20.5 tonnes were released into the atmosphere (Hermanson & Johnson, 2007). Many of the large industries using PCBs manufactured by Monsanto were located near major bodies of water, and PCBs were released into the environment as a result of unintentional leaks, volatilization during the production process, and migration from associated landfills and waste products. There was also production, at lower quantities, by Geneva Industries in Houston, Texas (de Voogt & Brinkman, 1989). As a result, contamination has occurred in many rivers and streams near these sites of production (see Section 1.4.6(a)).

(b) Europe

In western Europe, many chemical plants are located along major rivers (i.e. Rhine, Rhone, and Seine) and there have been several isolated incidents of organic chemical pollution. The Seine estuary remains one of the most polluted

Fig. 1.5 Blood PCB concentrations in individuals living in PCB-contaminated flats relative to individuals living in control flats



PCB, polychlorinated biphenyl
From Johansson et al. (2003)

in Europe (RNO, 2012). Also, the Venice lagoon in Italy is particularly polluted owing to the proximity of an important industrial district (the Marghera Harbour) (see Section 1.4.6(b)).

In the Slovak Republic, the Chemko Chemical Co. (based in the Michalovce district) produced 21 000 tonnes of commercial PCB mixtures between 1959 and 1984 (Delor 103, 104, 105, 106, Delotherm DK and DH, Hydelor 137). Improper disposal from the Chemko plant via release of effluent directly into the Laborec river resulted in long-term environmental contamination.

During the conflict of the former state union of Serbia and Montenegro throughout the 1990s, the burning or damaging of industrial and military targets resulted in the release of large amounts of PCBs into the environment: more than 1000 electro-transformer stations that contained PCB oil were damaged. After the bombardment of Kragujevac, Serbia, 2500 kg of PCB-based oil from the transformers of the Zastava automobile industry were spilled.

A French inventory reported that the number of installed transformers containing at least 100 kg of PCBs was 100 000 units in 1987, corresponding to 50 000 tonnes of fluids containing 60% PCBs (Pyralene), and to 50 000 tonnes of carcasses with 5% of PCB residues. The 250 000 medium-voltage capacitors represented about 3000–5000 tonnes of pure PCBs, while the low-voltage capacitors represented 1500–2000 tonnes of hardly extractable PCBs.

In Spain, an inventory in 1997 reported some 6000 tonnes of PCBs, although the amount of material containing or contaminated with PCBs could reach 200 000 tonnes.

(c) Asia

Contamination of soil and sediments has been reported in the Russian Federation, China, Viet Nam, and Japan. Such contamination may originate from PCB producing plants (e.g. China, Japan, Democratic People's Republic of Korea), or from e-waste recycling facilities (e.g. China). In addition, two major accidents of food contamination occurred in Taiwan, China and Japan (see Section 1.4.2(a)).

(d) South and Central America

There has been no manufacture of PCBs in South and Central America, but there has been widespread use of PCB-containing transformers and other PCB-containing devices.

(e) Africa

There has been no manufacture of PCBs in Africa, but there has been widespread use of PCB-containing transformers and other PCB-containing devices. In Africa, several studies showed an increase in the number of sources of PCBs, due to leakage and wrongly disposed transformers, shipwrecks, and biomass burning.

Another major source of exposure is the importing of e-waste and increase of e-waste recycling facilities, usually illegal, but common in Ghana, Senegal, Nigeria, Kenya, and the United Republic of Tanzania. A report by the United Nations Environment Programme (UNEP) documented issues concerning e-waste in South Africa, Kenya, Uganda, Morocco, and Senegal (UNEP, 2009).

In spite of the lack of homogenous data, an attempt has been made to compare the main PCB stocks that reside in the various countries of the region. [These data should only be seen on the relative scale since lacking the accuracy to make them valuable in the absolute sense.]

In Algeria, the national inventory of electrical equipment and PCB wastes identified 6770 appliances and around 4000 tonnes of oil to remove. The deposit of transformers, capacitors and various equipment containing PCBs was estimated at 1700 tonnes in Tunisia and 1150 tonnes in Morocco (Business Med, 2010).

1.4.2 Accidental releases into the food-chain

(a) Asia

Cooking oil contaminated by Kanechlor has been the source of two accidental mass poisonings in western Japan (later called "Yusho," oil disease in Japanese) and in Taiwan, China (later called "Yucheng," oil disease in Chinese). Commercial PCB mixtures were used as heat-transfer media in oil tanks; leakage of the pipes caused exposure to the PCB mixture and PCB pyrolytic products, mainly PCDFs and polychlorinated quaterphenyls (PCQs) (Masuda *et al.*, 1986). Patients from both countries have been exposed to comparable quantities of PCBs and PCDFs. The PCB/PCDF concentrations in the Yusho oil were higher (several hundred ppm to 3000 ppm) than those in the Yucheng oil (53 to 100 ppm) (Guo *et al.*, 2003); however, on average, Yucheng patients consumed the contaminated oil for a longer duration than the Yusho patients.

(i) Yusho incident, Japan

In 1968, the Yusho incident involved approximately 1800 people who ingested rice oil contaminated by Kanechlor 400 and its pyrolytic products, mainly in Fukuoka and Nagasaki prefectures (Masuda, 1994a, b; Kuratsune, 1996; Matsueda *et al.*, 1993; Todaka *et al.*, 2007a; Nagayama *et al.*, 1977; Tanabe *et al.*, 1989; Masuda *et al.*, 1998; Ohta *et al.*, 2008a). Affected people developed a "strange skin disease," including acne-form eruption, follicular accentuation, and pigmentation, as well as eye discharge and swelling of eyelids. The mean concentrations of seven PCB congeners (PCB-105, PCB-118, PCB-138, PCB-153, PCB-157, PCB-170, and PCB-180) detected in blood were 6.7 ppb and 3.84 ppb (95% confidence

interval, 3.54–4.17), 5 and 20 years after being exposed, respectively (Masuda & Yoshimura, 1982). Mortality data among registered Yusho patients were identified by follow-up studies to 1990 and 2007. The first of these two reports (Ikeda & Yoshimura, 1996) reported serum PCB concentrations in the range of 0 to 35 ppb in 1972, and a decrease to about 5 ppb in 1984 (Iida *et al.*, 1999) (see Sections 1.4.9(b)(iii) and (c)(iv) for additional data on PCB concentrations in blood and adipose tissue, respectively).

(ii) Yucheng incident, Taiwan, China

In 1978–9, the Yucheng incident involved approximately 2000 people who ingested rice oil contaminated with Kanechlor 500 and its pyrolytic products (Hsu *et al.*, 1985). After a few months, these people developed chloracne, hyperpigmentation, severe fatigue, peripheral neuropathy, and other signs and symptoms similar to Yusho disease. On the basis of a dietary questionnaire, it was estimated that Yucheng patients had consumed on average about 1 g (range, 0.7–1.4) of PCBs and 3.8 mg (range, 1.8–5.6) of PCDFs (Lan *et al.*, 1981). Another study estimated the intakes of PCBs, PCDFs, and PCQs by Yucheng patients at 673, 3.8, and 490 mg, respectively (Masuda *et al.*, 1986). DL-PCBs contributed to approximately 30% and 20% of the total TEQ (toxic equivalent) in Yucheng men and women, respectively. Compared with the general population in Taiwan, China, the mean total serum PCB concentrations in the Yucheng victims were still nine times higher 15 years after exposure (see Sections 1.4.9(b)(iii) and (c)(iv) for additional data on PCB concentrations in blood and adipose tissue, respectively).

(b) Europe

In Europe, the "Belgian dioxin crisis" was caused by the accidental release of 50 kg of a commercial PCB mixture contaminated with 1 g of dioxins commonly found in transformers, to a stock of recycled fat used for the production of 500 tonnes of animal feed. In May 1999, it appeared that more than 2500 poultry and pig farms could have been contaminated. Chickens showed the classical signs of oedema disease.

In Ireland in 2008, a tank for storage of pork fat was contaminated with heat-transfer fluid containing PCBs (Hovander *et al.*, 2006). [The Working Group noted that the label of "dioxin crisis" attributed to these episodes of PCB feed contamination was inappropriate.]

1.4.3 Outdoor air

PCBs in outdoor air may be a significant source of exposure. Concentrations of PCBs in air depend on a variety of factors, including temperature and proximity to local sources. Temperature is particularly important in controlling the cycle of volatilization and precipitation. Proximity to local sources, such as industrial facilities, landfills, or contaminated bodies of water, results in elevated air concentrations of both vapour phase and particulate-bound PCBs that dissipate with distance at different rates, resulting in both local and distant contamination. Combustion and other high-temperature processes generate PCBs, in particular during combustion of highly chlorinated compounds; however, this route of unintentional formation is considered to contribute little to total airborne PCBs. Migration to the outdoor environment has also been shown to occur as a result of erosion of exposed sealants.

(a) North America

PCB concentrations in outdoor air vary greatly between urban and rural sites in North America, and may be very high near industrial facilities and other contaminated sites (Table 1.16). These differences reflect primarily the impact of local sources and dilution in air, but also the deposition of PCBs at lower temperatures.

The major sources in Chicago are from landfills, sewage sludge drying beds, and transformer storage yards (Hsu *et al.*, 2003). Shen *et al.*

Table 1.16 PCB concentrations in outdoor air in North America

Reference	Location, sources	PCBs measured	PCB concentration in pg/m³ as mean and/or range	Comments
Vorhees *et al.* (1997)	Near a PCB-contaminated site, New Bedford Harbor, Massachusetts	"PCB concentrations"	400–61 000	
	Comparison neighbourhood		100–8200	
Hung *et al.* (2001)	Canadian Arctic	Sum of 102 congeners	28 in 1993; 23 in 1997	PCB-28, PCB-52, and PCB-118 showed little or no decline over time
Hermanson *et al.* (2003)	Near the former Monsanto PCB-manufacturing facility in Anniston, Alabama	Sum of 120 congeners	8700–82 000 [annual average, 27 000]	
Totten *et al.* (2004)	Urban sites (Camden and Jersey City, New Jersey)	Sum of 116 congeners	Average, 3250 and 1260, respectively	
	Remote and suburban areas at various sites near the New York City metropolitan region		Averages of 150–220	
Sun *et al.* (2006)	Six sites near near USA–Canadian Great Lakes (Lake Michigan near Chicago)	Sum of 84 congeners	± 100–1400	
Hermanson & Johnson (2007)	Near the former Monsanto PCB-manufacturing facility in Anniston, Alabama	PCBs in tree bark	171 927 ng/g (ppb) lipid near the site, to 35 ng/g (ppb) lipid at a distance of 7 km	Tree bark serves as passive vapour-phase air sampler
Sun *et al.* (2007)	Six sites distant from urban areas near USA–Canadian Great Lakes (Lakes Superior and Huron)	Sum of 84 congeners	60–86	
	Six sites near near USA–Canadian Great Lakes (Lake Erie)		± 1.1–230	
Palmer *et al.* (2008)	Near the contaminated Hudson River, downstream communities	Sum of 84 congeners	Median, 711	Concentrations were higher closer to the river than further away, and higher in warmer than cooler months of the year. The congener pattern in air was primarily PCBs with three or four chlorines
	City upstream of the industrial sites that caused the contamination		Median, 431	
Palmer *et al.* (2008)	Contaminated portion of the Hudson River	Sum of 84 congeners	102–4011 (median, 711)	
	Community upstream of the contamination		80–2366	
Harrad *et al.* (2009)	Toronto, Canada	Sum of 8 congeners	100–1400 (mean, 350)	
Li *et al.* (2010)	North America	Sum of PCBs	79 (49–120)	
	Remote sites in Alaska and rural sites in the lower 48 states of the USA		1–50	
	Large urban areas like Chicago		1000 and 150 000	
Persoon *et al.* (2010)	Cleveland, Ohio	Sum of 151 congeners	1730–4240	
	Chicago, Illinois		1130–2690	

PCB, polychlorinated biphenyl

(2006) found large relative differences in air PCB concentrations between urban, rural and remote sites, with the highest concentrations in Toronto, Canada, and the Eastern third of the USA [absolute concentrations could not be quantified] using results from passive air samplers in 31 stations in Canada and the USA.

(b) Europe

In Europe, the reported PCB concentrations in outdoor air range from ~10 up to ~1000 pg/m^3 in western European countries and from ~50 up to ~9000 pg/m^3 in eastern European contries.

Measurement in the Baltic region showed PCB concentrations in southern Norway to be rather high and similar to those in urban areas (Backe *et al.*, 2000; Agrell *et al.*, 2001). Results from the Czech national monitoring system and European Monitoring and Evaluation Programme (EMEP) background monitoring stations also showed relatively high PCB concentrations in this country (EC, 2004). Typical values for background sites usually range up to ~100 pg/m^3 and up to several 100s pg/m^3 for contaminated areas (Kocan, 2000, 2001).

PCB concentrations in outdoor air may also be measured in precipitation as total deposition rates (ng/m^2 per day). In southern Sweden (Backe *et al.*, 2002), PCB concentrations ranged from 1.18 to 81.4 ng/L, with no seasonal trends. In Paris, France, average PCB concentrations (sum of seven congeners) in rain during 1986–2001 remained approximately constant at about 40 ng/L (Chevreuil *et al.*, 2001).

Declining concentrations of PCBs have been observed since the early 1960s and 1970s, decreasing by 67% in France (EC, 2004) and by 78% in the United Kingdom (CITEPA, 2013) over 20 years. The difference observed between the steady concentrations in rain and the decrease in general atmospheric emissions may be partly explained by water solubility limits and differences between point sources and global emissions.

Air concentrations of the seven indicator PCBs 28, 52, 101, 118, 153, 138 and 180 were measured at four locations in the Czech Republic, Finland, Sweden, and the Netherlands, from 1996 to 2001. Measured values did not vary noticeably during this period at any location (Fig. 1.6). This suggests that a steady-state has been reached between degradation and environmental cycling, with an ongoing low-level input from existing equipment and contaminated material (Holoubek *et al.*, 2003).

(c) Asia

Limited information on the concentrations of PCBs in air and dust has been reported in Asian countries (Table 1.17). One of the most extensive studies reported results for outdoor air samples from 55 sites in Japan, 20 in China, 30 in the Republic of Korea, and 1 in Taiwan, China. The range of concentrations was 100–1000 pg/m^3.

(d) South and Central America

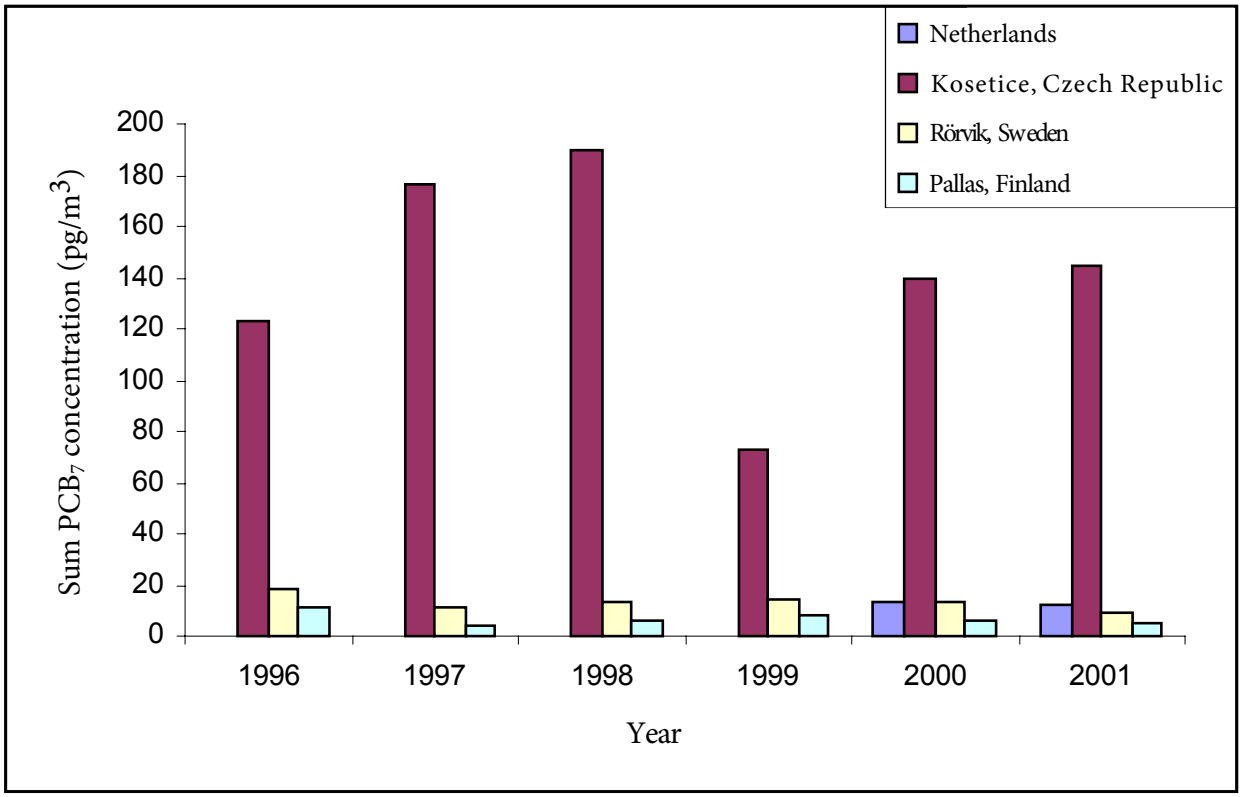
Shen *et al.* (2006) found large relative differences in air PCB concentrations between urban, rural and remote sites using passive air samplers in 4 stations in Mexico, Belize, and Costa Rica. One site in Mexico had higher concentrations than sites in Central America and in Canada.

Li *et al.* (2010) reviewed information from various research groups around the world and reported the average concentration of the sum of PCBs in air to be 66 pg/m^3 (range, 9–670 pg/m^3) for South America, and 59 pg/m^3 (range, 17–150 pg/m^3) for Central America.

(e) Africa

Only recently have data from passive air samplers deployed on the African continent become available. PCB concentrations have been reported as very high in Senegal (500 pg/m^3) (Klánová *et al.*, 2009), Côte d'Ivoire, and the Gambia (up to 300 pg/m^3) (Gioia *et al.*, 2011). Concentrations in some areas in South Africa, Kenya, Egypt, the Democratic Republic of the

Fig. 1.6 Annual average atmospheric concentrations of seven indicator PCBs (PCB_7) from four European Monitoring and Evaluation Programme stations in Europe, 1996–2001



PCB_7, sum concentration of PCB-28, PCB-52, PCB-101, PCB-118, PCB-153, PCB-138, and PCB-180
From Holoubek *et al.* (2003)

Congo, Ghana, Mali, and the Sudan were also high, and comparable to those in urban areas in more developed countries. These levels could not be explained by biomass burning or primary emissions, and were probably due to e-waste dumps. Lower concentrations have been measured in the Congo, Ethiopia, Mauritius, Nigeria, the Togolese Republic, Tunisia, and Zambia.

(f) Vegetation used for monitoring studies

Plant foliage is a reliable proxy for monitoring levels of vapour-phase compounds in outdoor air since it bioaccumulates organic pollutants. Several researchers have used vegetation, grass, conifer needles, mosses, pollen, and leafy vegetable species (cabbage and lettuce) as biomonitors to evaluate patterns of PCB contamination (Larsen *et al.*, 1985; Reischl *et al.*, 1989; Kylin, 1994; Simonich & Hites, 1995). This method has been employed in high-mountain ecosystems (Daly & Wania, 2005), and in several countries, including the Czech Republic (Holoubek *et al.*, 1994), Poland (Migaszewski, 1999), western Finland (Sinkkonen *et al.*, 1995), Germany (Reischl *et al.*, 1987), Italy (Gaggi *et al.*, 1985), and France (Granier & Chevreuil, 1992).

1.4.4 Indoor air

PCBs have been shown to migrate into surrounding materials, such as concrete or wood, and to indoor air. The major sources are PCB-containing caulk, paint (where PCB-11 is the main marker), floor sealants, and ballasts

Table 1.17 PCB concentrations in outdoor air and dust in Asian countries

Reference	Country, region Date of study	Sources	PCBs measured Comments	Concentrations
Iwata *et al.* (1995)	Russian Federation, Lake Baikal May 1992	Six outdoor air samples from research vessel	Kanechlors 300, 400, 500, 600 as standards	Range, 8.7–23 pg/L
McConnell *et al.* (1996)	Russian Federation, Lake Baikal June 1991	A total of 19 outdoor air samples	Aroclors 1242 and 1254 as standards	Mean, 196 ± 65 pg/m^3
Hogarh *et al.* (2012)	Taiwan, China; China; Japan; Republic of Korea March–May, 2008	Outdoor air samples from 55 sites in Japan (37 rural, 4 suburban and 14 urban), 20 in China (3 rural and 17 urban), 30 in the Republic of Korea (12 rural, 2 suburban and 16 urban), and 1 in Taiwan, China	Sum of 202 congeners	Japan, 40–760 pg/m^3 China, 300–2500 pg/m^3 Taiwan, China, about 317 pg/m^3 Republic of Korea, 36–600 pg/m^3
Thacker *et al.* (2013)	India, central and western regions 2009–2010	Outdoor air samples from various cities	Sum of dioxin-like PCBs	Range, 0.0001 × 10^{-1} to 0.0295 ng TEQ/Nm^3

PCB, polychlorinated biphenyl; TEQ, toxic equivalent

in lighting devices. Outgassing from contaminated dust may also contribute. Joint sealants are increasingly recognized as important diffuse sources of indoor air contamination by PCBs.

(a) North America

PCBs have been measured in indoor air in several studies (Vorhees *et al.*, 1997; Vorhees *et al.*, 1999; Herrick *et al.*, 2004; Colt *et al.*, 2005; Franzblau *et al.*, 2009; Harrad *et al.*, 2009). In the USA it was reported that indoor air concentrations of PCBs were 5–300 times greater than those in outdoor air (Wallace *et al.*, 1996), and that concentrations were higher in older buildings. The concentrations of PCBs in indoor air in North America are summarized in Table 1.18.

(b) Europe

The highest indoor concentrations (up to 7500 ng/m^3) have been reported in buildings constructed between 1960 and 1975 from prefabricated concrete elements sealed with elastic materials containing PCBs (Balfanz *et al.*, 1993). Joint sealants containing PCB were discovered in various public buildings in Europe (Kohler *et al.*, 2005; Wilkins *et al.*, 2002). Estimated indoor PCB concentrations in contaminated sections were the lowest in microenvironments such as cars (8.92 ng/m^3), and were inversely related to the degree of chlorination of the PCB mixtures used (Hammar 1992; Harrad *et al.*, 2006; Kuusisto *et al.*, 2006, 2007; Frederiksen *et al.*, 2012). The concentrations of PCBs in indoor air in Europe are summarized in Table 1.19.

(c) Asia

Indoor floor dust samples (n = 43) collected from rural homes and mosques in Gujarat, Pakistan, showed median total PCB concentrations of 0.67 ng/g (range, 0.3–6.1 ng/g) (Ali *et al.*, 2012). The PCB profile was dominated by PCB-153 (> 60% of the sum of PCBs), with concentrations between < 0.2 and 2.4 ng/g. These PCB concentrations were 10 times lower than those reported in house dust in Singapore (Tan *et al.*, 2007).

1.4.5 Soil and sediments

PCBs can enter soil and sediments through various routes. Sediments constitute an important sink for PCBs entering the marine environment. Sewage sludges are monitored for PCBs in countries where they are largely used (60%) in agriculture. The dumping of incinerator-related materials and/or the inadequate management of commercial PCBs have resulted in significantly elevated PCB concentrations.

1.4.6 Water

Inputs of PCBs to the hydrological cycle are principally via discharges of sewage and industrial effluents, urban run-off, leachates from solid waste landfill sites, atmospheric deposition and, of increasing concern, via agricultural run-off (Scrimshaw *et al.*, 1996).

Water can contain PCBs either in solution or bound to particulates. While PCBs are not very water-soluble, water can be a significant source of exposure to less chlorinated congeners that have a greater solubility than more highly chlorinated congeners. PCB concentrations in sea and freshwater are summarized in Table 1.20.

(a) North America

(i) Drinking-water

In the USA, the EPA has set a goal for PCBs in drinking-water of zero, and a maximum contaminant concentration of 500 ng/L (500 ppt), with sources being primarily landfills, and discharge of waste chemicals (EPA, 2014). While conventional treatment of drinking-water will remove particulate-bound PCBs, those that are soluble are often not completely removed. Solubilities of individual PCB congeners vary from about 4 ppm for monochlorobiphenyl to as low as 0.0007

Table 1.18 PCB concentrations in indoor air in North America

Reference	Location	Source	PCBs measured	Concentration	Comments
Vorhees *et al.* (1997)	New Bedford Harbor, Massachusetts, USA	18 homes	Sum of 65 congeners	Geometric mean concentration, 18 ng/m³ (range, 7.9–61 ng/m³)	
		Comparison neighbourhood		Geometric mean concentration, 10 ng/m³ (range, 5.2–51 ng/m³)	
Vorhees *et al.* (1999)	New Bedford Harbor, Massachusetts, USA	House dust in homes surrounding the Superfund site	Sum of 65 congeners	1400 (range, 320–23 000) ng/g dry weight	
		Comparison neighbourhood		60 (15–290) ng/g	
Herrick *et al.* (2004)	Greater Boston, USA	24 university buildings		> 36 200 ppm 111–395 ng/m³	One third of the 24 buildings investigated contained caulk at concentrations > 50 ppm (the EPA limit)
Colt *et al.* (2005)	Four geographical regions in the USA	PCBs in carpet dust, 443 homes of Caucasian Americans who served as controls in a case–control study on non-Hodgkin lymphoma		Specific concentrations not reported	PCB concentration in dust was significantly related to age of the house, being greatest in homes built before 1940, and significantly greater in homes built in 1960–1979 (when PCBs were being manufactured in the USA) than in homes constructed after 1980
Franzblau *et al.* (2009)	Five counties in Michigan, USA	House dust House dust	PCB-123 PCB-118	439 000 ppt 33 600 000 ppt	Dioxin-like PCBs contributed 66.2% of the total WHO TEQ found in dust
Harrad *et al.* (2009)	Texas, USA	20 homes	Sum of 9 tri- to heptachlorinated congeners	200 ng/g (ppb); (range, 0.71–620 ng/g)	
	Toronto, Ontario, Canada	10 homes	Sum of 9 tri- to heptachlorinated congeners	260 ng/g (ppb) (range, 51–820 ng/g)	Levels were more than four times higher than those measured in cities in the United Kingdom and New Zealand

EPA, United States Environmental Protection Agency; PCB, polychlorinated biphenyl

Table 1.19 PCB concentrations in indoor air in Europe

Reference	Country	Source	PCB concentration (mean or range)	Comments
Hammar (1992)	Sweden	Joint sealants	80 ng/m^3	Outside the building, mean concentrations were 0.5–4.6 ng/m^3
Balfanz *et al.* (1993)	Germany	Air from contaminated buildings	Range, > 300–7500 ng/m^3	Indoor PCB concentrations were inversely related to the degree of chlorination of the PCB mixtures used
Wilkins *et al.* (2002)	Denmark (Organization of Sealant Branch's Manufacturers and Distributors)	Dust from public and residential buildings with excessive microbial growth	Estimated inventory of 75 tonnes in caulking materials	Concentration in polluted buildings was 10–20 times higher than the amount found in samples from other buildings
Kohler *et al.* (2005)	Switzerland	Joint sealants in public buildings	> 10 g/kg in 48% of samples	70% of samples contained PCB mixtures such as Clophen A50, Aroclor 1248, and Aroclor 1254
Harrad *et al.* (2006)	United Kingdom	Homes, offices, cars, public microenvironments	8.92 ng/m^3	The least contaminated microenvironment was the car (average, 1391 pg/m^3)
Kuusisto *et al.* (2007)	Finland	Walls/floor	110–540 µg/m^2	Detected PCBs were highly chlorinated
Frederiksen, *et al.* (2012)	Denmark	Air from uncontaminated apartments Elastic sealants from contaminated apartments	168–3843 ng/m^3 187–221 680 mg/kg	Significant correlations were observed between the lower chlorinated congeners in air and sealant

PCB, polychlorinated biphenyl

Table 1.20 PCB concentrations in various types of water around the world

Reference	Type of water	Location	PCB measured	Concentration	Comments
North America					
Jeremiason *et al.* (1994)	Lake	Lake Superior, USA		2.4 ng/L in 1980; 0.18 ng/L in 1992	
Connolly *et al.* (2000)	River	Hudson River		Sometimes > 1300 ng/L	Varied greatly with season and water flow
Rowe *et al.* (2007)	River	Delaware River	Sum of 116 congeners	420–1650 pg/L	
Wang *et al.* (2012)	River	Mississippi River	Sum of 27 congeners	86 and 254 ng/L	
	Lake	Lake Pontchartrain		134–728 ng/L	In some months the PCBs in river water were primarily in the liquid phase, whereas in other months primarily in the sediment
South and Central America					
Rissato *et al.* (2006)	River	Sao Paulo State, Brazil	Sum of seven congeners	0.02–0.5 ng/L	Predominantly lower chlorinated congeners
Africa					
Scarpato *et al.* (2010)	Sea	Tunisia Morocco–Algeria coastal sites	Sum of 10 congeners	10–12 ng/g 7–8 ng/g	PCB contamination evaluated by mussel-caging technique (exposure, 12 weeks)
Jayed *et al.* (2010)	Ocean	Thirteen sites along the Atlantic Moroccan coast	Sum of PCB-28, PCB-153, PCB-138	Wet season: 11 ng/g Dry season: 8.2 ng/g	Concentrations in mussels during wet and dry seasons not significantly different, but values in the northern sites exceeded 2–3 times the medians registered for the other sampling sites
Vorkamp *et al.* (2010)	Ocean	Cape Town harbour Cape Town sea shore Ghana coast	Sum of congeners	81 ng/g dw 15 ng/g dw 5 ng/g dw	Bivalve samples
Europe					
Nondek & Frolikova (1991)	Lake	Sumava lakes, Czech Republic		1900 ng/g	Contamination due to atmospheric transport to non-industrialized areas
Winkels *et al.* (1998)	River	River Danube, Czech Republic		< 5 ng/g dw	Contamination due to flood disaster in the Moravian part of the Czech Republic in July 1997

Table 1.20 (continued)

Reference	Type of water	Location	PCB measured	Concentration	Comments
Fillmann *et al.* (2002)			Sum of seven congeners	≤ 700 ng/g dw (2–196 µg/kg ww in fish)	
UNEP (2002)	River	Krupa, Sana and Lepenica rivers, Balkan area, Slovenia		380 ng/L (in 1988) 100 ng/L (in 1997)	The factory in Semič was storing 5–6 tonnes of waste oil containing PCBs
Desmet *et al.* (2012)	River	Rhone river, France	Sum of PCB_7	1–40 ng/g dw	Concentrations consistently lower than those found during the previous decade (Burns & Villeneuve, 1987). Maximum PCB concentration was identified in 1960–75. The downward trends in concentration followed emission reductions, although soil concentrations decreased at much slower rates (Tolosa *et al.*, 1995)
ADEME (1998), Blanchard *et al.* (2001)	Wastewater	Wastewater treatment plants, France	Sum of seven congeners	Input water, 100–300 ng/L Output water, 15–54 ng/L	In 1999, average concentration was 15–26 ng/L. High levels of DL-PCBs in eel from Dutch freshwater were reported in a screening of Dutch fishery products (Van Leeuwen *et al.*, 2002)
Asia					
Kucklick *et al.* (1994)	Lake	Lake Baikal, Siberia, the Russian Federation, June 1991	61 PCB congeners using standards of Aroclor 1242, 1254, and 1260	Mean, 560 ± 180 pg/L for dissolved phase, and 420 ± 400 pg/L for particulate phase	
Iwata *et al.* (1995) May 1992	Lake	Lake Baikal, the Russian Federation, June 1991	Kanechlors 300, 400, 500, 600 as standards	Range, 8.7–23 pg/m^3	
McConnell *et al.* (1996) June 1991	Lake	Lake Baikal, the Russian Federation, June 1991	Aroclors 1242, 1254 as standards	Mean, 1 324 ± 96 pg/m^3	

DL-PCB, dioxin-like polychlorinated biphenyl; dw, dry weight; PCB, polychlorinated biphenyl; ww, wet weight

ppm for the decachlorobiphenyl (Erickson, 1997). Thus under certain circumstances, drinking-water can still be a source of exposure to less chlorinated congeners.

(ii) Sea and freshwater

The USA–Canadian Great Lakes are contaminated by multiple sources of PCBs (Bhavsar *et al.*, 2007; Turyk *et al.*, 2012). It has been shown that industrial sites on rivers feeding Lake Erie received the largest quantities of PCBs, with 26% derived from atmospheric deposition (Kelly *et al.*, 1991). The Hudson River in New York is highly contaminated with PCBs because of releases from two large capacitor plants (Carpenter & Welfinger-Smith, 2011), the Fox River in Wisconsin is highly contaminated because of releases from a manufacturer of carbonless copy paper, and a paper mill (Imamoglu *et al.*, 2004), and the St Lawrence River and several of its tributaries have been contaminated by releases from aluminium foundries operated by companies that discarded hydraulic fluids containing PCBs in drains (Fitzgerald *et al.*, 1996). The Hudson and Fox Rivers are being dredged to remove these contaminants.

(b) Europe

(i) Sea

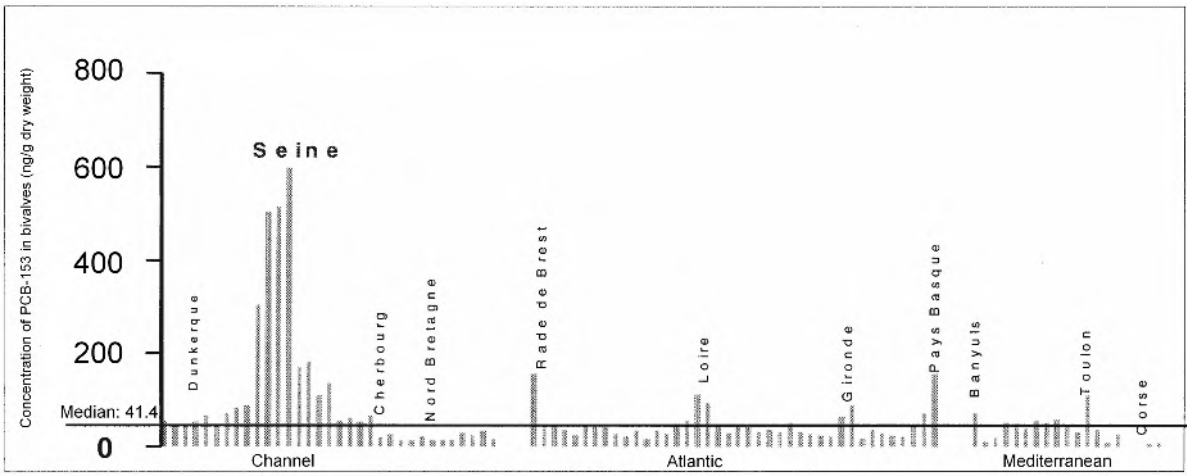
An extensive review of data obtained during the 1980s has been published (Tolosa *et al.*, 1995). In general, the concentrations of PCBs for all the investigated areas in the Mediterranean Sea were similar except in the Ligurian Sea where concentrations were higher. Predictably, the highest concentrations were reported in urban and industrial wastewaters (e.g. from Marseille and Barcelona) as well as in river discharges (e.g. from the Rhone), and decreasing concentration gradients have been found in transects offshore from these sources. PCB concentrations in the suspended particulate matter from coastal and open Western Mediterranean waters were of 5–35 pg/L in 1990, of the same order of magnitude as those reported in other regions, e.g. North Sea and North Atlantic. A more recent study covering the whole Western basin also shows a spatial gradient from the continental shelf (3.5–26.6 pg/L) towards the open sea (1.7–6.6 pg/L); a relatively important enrichment (8.4 pg/L) in open sea stations located in higher productivity frontal zones was observed (Dachs *et al.*, 1997). The dissolved PCBs (Σ12 congeners) amounted to 28–63 pg/L. Total concentrations of PCBs in estuarine and coastal sediment samples of the Mediterranean Sea ranged from 0.04 to 1684 ng/g dw (Koci, 1998; Vale *et al.*, 2002; Vojinovic-Miloradov *et al.*, 2002; Cardellicchio *et al.*, 2007).

During 1974–82, PCB concentrations decreased by a factor of 3 in offshore Monaco (Burns & Villeneuve, 1987), while the surface sediments of the Adriatic coast did not show a temporal trend (Picer & Picer, 1991).

Concentrations of PCBs in ocean water are usually in the low picogram per litre range. The general trend for concentrations in the Baltic Proper suggests an increase in PCB concentrations from the early 1970s onwards (ICES, 2000). This is an opposing trend to the decreasing concentration trends for PCBs in biota from the Baltic Proper (HELCOM, 1996; Roots, 1996).

The monitoring of PCBs in coastal areas may be based on measurements in mussels. Trends in PCB concentrations in the Seine estuary in France are reported in Fig. 1.7 (RNO, 2012). The rate of decrease was 3.5% per year. As reported by the Arctic Monitoring and Assessment Programme (AMAP), several time-series of PCB-153 concentrations in blue mussels from around Iceland showed significant decreasing trends; however, one time-series from a fjord system showed a significant increase (Rigét *et al.*, 2010). Active mussel watching (mussel transplantation) has also been applied in monitoring programmes in Africa (see Table 1.20).

Fig. 1.7 PCB contamination along the coast of France



Concentrations of PCB-153 in mussels or oysters (used as "sentinel species") sampled from coastal areas of France. The Seine estuary and bay are heavily exposed to manmade chemicals of terrestrial origin derived from the urbanized and industrialized river Seine.
Data from The French pollution monitoring programme (Réseau National d'Observation de la qualité du milieu marin)
Reproduced from Abarnou *et al.* (2002), with permission from the publisher

(ii) Freshwater

The major source of freshwater contamination in Europe comes from diffuse leaching of products from users, households, and industries into wastewater streams (UNEP, 2002). The areas most polluted by flood disasters are in Poland (the River Odra) (Wolska *et al.*, 1999; Protasowicki *et al.*, 1999) and in the Czech Republic. The River Danube is a major source of contamination to the Black Sea; however, many chlorinated hydrocarbons have been banned by several European and other countries in the past 10 years (Winkels *et al.*, 1998; Covaci *et al.*, 2002c; Fillmann *et al.*, 2002; see Table 1.20).

Industrial contamination is known to have occurred in Germany (the Rivers Elbe and Rhine and their tributaries) (Brauch, 1993), in former Czechoslovakia (the Sumava Lakes) (Nondek & Frolikova, 1991), in England and Ireland (where however approximately a 50% decline in concentrations between 1970 and 1990, was recorded) (Sanders *et al.*, 1992; Harrad *et al.*, 1994) and in Slovenia through the dumping of industrial waste in the Krupa river during the manufacture of transformers. PCB contamination also occurred in the Balkan area, in the cities of Pancevo, Novi Sad, Belgrade, Kragujevac, in Serbia, after military intervention by NATO in spring 1999.

1.4.7 Food products

Since the early 1990s, food has been identified as the major route of human exposure to lipophilic and persistent organochlorines such as PCBs, PCDDs, and PCDFs. In populations that are not exposed to other known sources, dietary intake contributes to about 90% of the total daily intake of dioxin-like compounds including dioxin-like PCBs, and of this, food of animal origin contributes about 90% in various regions of the world (Schecter *et al.*, 1997; Büchert *et al.*, 2001; Llobet *et al.*, 2003a, b; Päpke & Fürst, 2003; Schecter *et al.*, 2003a, b; Charnley & Doull, 2005; Huwe & Larsen, 2005).

Similarly, it is generally accepted that the major route of exposure to non-dioxin-like PCBs, namely to PCB_6, is dietary intake, by consumption of fatty foodstuffs (IARC, 1978; IPCS, 1993; EFSA, 2005; Lindell, 2012). However, inhalation can also be a significant source of exposure (see Section 1.4.4).

Human food can become contaminated by PCBs via three main routes:

- uptake from the environment, by fish, birds, livestock (via food-chains), and crops;
- contamination of animal feed, by regular practices or accidentally;
- direct contamination of food, accidentally.

Data on PCB concentrations in food are reported in many different ways, making comparisons difficult. The number of congeners analysed differs between studies and often congeners are summed according to groups, such as indicator PCBs, DL-PCBs, or some other number of congeners. When using TEQs, the scheme used should be noted; also some studies report TEQ on the basis of bioassays such as the CALUX system as biological equivalents (BEQ). Results have been reported with different reference units (wet weight, dry weight, or lipid weight). Further difficulties in interpretation arise since different parts of fish or seafood are analysed (muscle, liver, skin, etc.) and PCB concentrations are also sometimes reported on the basis of prepared food (to account for changes by cooking or frying). Finally, the objectives of a study may bias the sampling strategy, often resulting in reporting of higher concentrations.

(a) PCB concentrations in food

Concentrations of DL-PCBs in various meats and dairy products from selected countries and regions are presented in Table 1.21.

(i) Polar regions and North America

PCB concentrations in food for polar regions and North America are summarized in Table 1.22. Domingo & Bocio (2007) reviewed the concentrations of PCB and PCDD/PCDF in marine species and human intake through fish and seafood consumption by different region-specific sections.

The traditional food items for indigenous peoples in the Arctic include lipid-rich tissue of high trophic-level animals. After long-range transport and biomagnification of PCBs in the Arctic marine food-chain, PCBs accumulate in edible animals like fish, seals and whales (AMAP, 2004). This dietary exposure led to PCB concentrations in Arctic inhabitants that exceeded those of individuals living at temperate latitudes (Dewailly *et al.*, 1993), but levels have been shown to decrease (AMAP, 2009). Likewise, PCBs in traditional food items have generally decreased (Rigét *et al.*, 2010).

(ii) Africa

Loutfy *et al.* (2006) investigated levels of WHO-TEQs from diet in Egypt, and determined a range of 6.59–9.98 pg TEQ/kg per day, with about 40% of this value due to DL-PCBs. This value exceeds the maximum WHO tolerable daily intake (TDI) of 4 pg TEQ/kg per day. The primary source was found to be dairy products, in which PCB concentrations were several times higher than in such products in more developed countries. Loutfy *et al.* (2007) determined the concentrations of PCDD/PCDF and dioxin-like PCBs in samples of fish and seafood (mullet fish, bolti fish, bivalves and crab) randomly acquired in local markets in Egypt. The upper-bound concentrations of dioxin-like PCBs ranged from 0.14 (bivalves) to 0.76 (mullet) pg WHO-TEQ/g wet weight, respectively.

Adu-Kumi *et al.* (2010) reported an average TEQ for dioxin-like PCBs in fish from two lakes in Ghana to be 0.7 pg WHO-TEQ/g.

(iii) Australia and New Zealand

In 2000–2001, 168 samples of 22 foods collected for the Australian Total Diet Survey were analysed for DL-PCBs and compared with those from other areas of the world (Table 1.21; Food Standards Australia New Zealand, 2004).

A more recent study reported PCB concentrations from composite samples of Australian farmed yellowtail kingfish (mean, 21 µg/kg; range, 8.6–29 µg/kg), mulloway (mean, 5.4 µg/kg; range,

Table 1.21 Concentrations of dioxin-like PCBs in selected foods from various countries and regions

Food	PCB concentration (range of means), pg TEQ/g lipid					
	Australia	Europe[a]	New Zealand[a,b]	North America[a]	Netherlands[c]	United Kingdom
Beef	0.03–0.11	–	0.0036–0.092	0.5	1.24	0.25–0.31[f]
Pork	0.04–0.07[d]	0.8	0.15–0.43[e]	0.02–1.7	0.23	–
Lamb	0.02–0.06	–	0.01–0.045	–	–	–
Poultry	0.18–0.24	0.7	0.018–0.14	0.3	1.72	0.47–0.53
Fish	9.46–9.5	0.03–9[h]	0.77	0.11–0.28[h]	0.412[g,h]	3.57–3.57
Eggs	0.04–0.11	0.2–0.6	0.05–0.11	0.029[h]	0.87	0.11–0.20
Milk	0.04–0.11	0.2–1.8	0.027–0.15	0.5	0.69	0.34–0.43
Bread	0.0003–0.005	–	0.00099–0.004	–	–	0.06–0.15
Butter	0.021–0.086	–	0.15–0.15	–	0.96	–

[a] Results reported in international toxic equivalents (I-TEQ), which are 10–20% lower than WHO-TEQs
[b] Results reported in the range of lower to middle bound
[c] Results reported as lower bound only
[d] Assumes bacon is representative of all pork products
[e] Pork meat
[f] Carcass meat
[g] Lean fish
[h] Reported on a fresh-weight basis
From Food Standards Australia New Zealand (2004)
PCB, polychlorinated biphenyl; TEQ, toxic equivalent

4.7–6 µg/kg) and manufactured feed (Padula *et al.*, 2012). The mean concentration of DL-PCBs was 2.1 pg TEQ/g (range, 1.2–2.8 pg TEQ/g) in kingfish, and 0.51 pg TEQ/g (range, 0.41–0.61 pg TEQ/g) in mulloway.

(iv) Asia

Concentrations of specific PCB congeners in samples of food from Asia are summarized in Table 1.23. In Japan, a study sponsored by the Ministry of Health and Welfare showed a more than 50% decrease in concentrations of three non-*ortho* substituted PCBs in human milk samples between 1973 and 1996 (Environment Agency of Japan, 1999). A report from the Republic of Korea demonstrated regular dietary exposure (Son *et al.*, 2012; Table 1.23). In China, Liu *et al.* (2011) determined concentrations of seven indicator PCBs in marine fish. The sum of PCB_7 ranged from 0.3 to 3.1 µg/g wet weight, with median and mean values of 6.4 ng/g wet weight and 398 ng/g wet weight, respectively (Table 1.23). The average concentrations and contributions of the seven specific congeners at four different sites are presented in Table 1.24. [It was noted that the concentrations found in this study were higher than in other parts of the world.]

(v) Europe

The major contributors to total exposure in Europe appeared to be milk and dairy products for almost all groups of infants and toddlers (Barr *et al.*, 2006; Becker *et al.*, 2009), and fish and seafood products for most of the adolescents, adults, elderly and very elderly groups (Langer *et al.*, 2007; Fréry *et al.*, 2009; ANSES, 2011).

The most comprehensive assessment of PCB concentrations in food was undertaken by the European Food Safety Agency (EFSA) (EFSA, 2005, 2010, 2012). For the 27 European Union Member States, and Switzerland and Norway, in a report that took all food groups together, the

Table 1.22 PCB concentrations in marine foods and estimated dietary intake in polar regions and North America

Country	Food analysed	PCB concentration	Estimated dietary intake	Comments	References
Polar regions					
Inuit of Quebec, Canada	ΣPCB10 in: Polar bear fat Seal blubber Arctic char muscle	 7 µg/g lipid 1 µg/g lipid 150 ng/g lipid		Female consumers of these foods had higher PCB concentrations in milk than a group in Southern Quebec	Dewailly *et al.* (1993)
West Greenland	ΣPCB10 in: Minke whale, beluga and narwhal blubber Halibut liver, kittiwake liver and muscle, minke whale skin, and seal blubber	 > 500 ng/g 50–500 ng/g	23 µg/day per person (3 µg/day per person if blubber food items are excluded from the diet)	Compared with the marine animals, concentrations in food sources from the terrestrial environment were characterized as low	Johansen *et al.* (2004)
North-western Territory, Canada	Food including cooked sucker flesh, raw beluga mattak (skin/blubber) and boiled Canada goose meat	Foodstuffs in the 50–500 ng/g group (Berti *et al.*, 1998)	Mean, 23 ng/kg bw per day Median, 11 ng/kg bw per day	Provisional tolerable daily intake was 300 ng/kg bw per day, based on Health Canada	Johansen *et al.* (2004)
Canada	Fish products from retail market	Geometric mean WHO-TEQ (pg/g wet weight): 0.06 (shrimp), 0.08 (tilapia), 0.92 (salmon)		No information on human exposure	Rawn *et al.* (2006)
North America					
USA (California coast)	Samples of a variety of fish	Mean I-TEQ: 109 pg/g lipid (non-*ortho* PCBs 77, 126, 169)		No information on human exposure	Brown *et al.* (2006)
USA, Maryland, Washington, DC, and North Carolina	Commercially wild caught and farm-raised fish	Bluefish, 800 ng/g ww (highest) Coho salmon, 0.35 ng/g ww (lowest)			Hayward *et al.* (2007)
USA	Salmon and canned sardines	Salmon: PCB-153, 1.2 ng/g ww; PCB-138, 0.93 ng/g ww Canned sardines: PCB-153 and PCB-138, 1.8 ng/g ww		Six of seven NDL-PCBs congeners were detected, with PCB-153 and PCB-138 at highest levels	Schecter *et al.* (2010)

NDL-PCB, non-dioxin-like polychlorinated biphenyls; ww, wet weight

Table 1.23 PCB concentrations in food in Asia

Country, region	Date	Source	PCBs measured	Concentration	Reference
Russian Federation					
Lake Baikal, Siberia	June 1991	Pelagic sculpin, omul, Baikal seal	61 PCB congeners using standards of Aroclor 1242, 1254, and 1260	Ranges, 2.7–2.8 mg/kg of lipid for pelagic sculpin, and 0.73–1.6 mg/kg of lipid for omul	Kucklick *et al.* (1994)
Lake Baikal, Siberia	May–June 1992	Five species of 35 fresh fish samples collected from Lake Baikal in 1993	Total PCBs using an equivalent mixture of Kanechlors 300, 400, 500, and 600 as standards	Mean, 1.7 ± 0.96 µg/g lipid	Nakata *et al.* (1995)
Lake Baikal, Siberia	1993	Three species of fish collected from Lake Baikal in 1993	Total PCBs using an equivalent mixture of Kanechlors 300, 400, 500, and 600 as standards	350 ± 350 ng/g ww	Nakata *et al.* (1997)
China					
Shanghai and its vicinity	2000–1	Various fish and seafood	Kanechlor-300, 400, 500, 600 as standards	Range, 0.20 (shrimp and mussel) to 2.5 (mackerel) ng/g ww	Nakata *et al.* (2002b)
North-eastern, Bohai Sea coastline	Early 2000s	Bivalves and gastropods	PCB mixture (EPA 68A-LCS)	Range, 62.3–344.9 ng/g lipid, for bivalves Range, 81.6–583.6 ng/g lipid, for gastropods	Zhao *et al.* (2005)
Dalian, Tianjin, and Shanghai		Fish and shellfish collected from local supermarkets	PCB-138 and PCB-153 were dominant, followed by PCB-101 and PCB-180	3.60 (0.83–8.04) ng/g ww Estimated daily intake: 1.83 ng/kg bw	Yang *et al.* (2006)
Guangzhou and Zhoushan	2003–4	Seafood (mainly harvested locally) purchased from local markets in Guangzhou and Zhoushan	PCBs 81, 77, 123, 118, 114, 105, 126, 167, 156, 157, 169, 189	Range, 1510–10 200 pg/g lipid	Jiang *et al.* (2007)
South China Sea, Bohai Sea, East China Sea, and Yellow Sea	2006–9	Marine fish	7 PCB congeners (28, 52, 101, 118, 138, 153, and 180); details in Table 1.24	Mean, 398 ng/g ww Median, 6.4 ng/g ww Range, 0.3–3100 ng/g ww	Liu *et al.* (2011)
South, Daya Bay and Hailing Bay	July 2007, December 2007	Fish	PCBs 31/28, 52, 44, 99, 149/118, 153, 138, 180, 170, 194, 101, 110, 147, 146, 187	Range, 1.5–4.0 ng/g ww	Yu *et al.* (2011a, b)
Nanjing	July, 2006	Fish and meat from 10 markets	PCBs 8, 18, 28, 52, 44, 66, 101, 81, 77, 123, 118, 114, 105, 153, 126, 138, 128, 187, 167, 156, 157, 170, 180, 189, 169, 195, 206, 209	Range, 0.87–15 ng/g ww for different fishery product; 5.1–20 ng/g ww for meat product	Su *et al.* (2012)
Fengjiang town (Taizhou)	2005–9	Rice hulls from a waste electrical and electronic-equipment dismantling area	PCBs 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 28, 52, 101, 138, 153, 180, 3, 15, 19, 202, 205, 208, 209 (dry weight basis)	44.1 ng/g (range, 12.8–124 ng/g) in 2005, 16.3 ng/g (range, 5.44–24.9 ng/g) in 2006, 9.01 ng/g (range, 2.57–22.8 ng/g) in 2007, 7.90 ng/g (range, 3.08–16.5 ng/g) in 2008, 7.39 ng/g (range, 3.80–10.7 ng/g) in 2009	Fu *et al.* (2012)

Table 1.23 (continued)

Country, region	Date	Source	PCBs measured	Concentration	Reference
Japan					
Ariake Sea		Shrimp, mussel, and mackerel		Range, 0.20–2.5 ng/g ww	Nakata *et al.* (2002a)
Japan		Fish and shellfish	PCB-126 and PCB-118 were the highest contributing congeners	In 1999: 0.98×10^{-3} WHO-$TEQ_{PCDD/PCDF/PCB}$ In 2004: 0.91×10^{-3} WHO-$TEQ_{PCDD/PCDF/PCB}$	Sasamoto *et al.* (2006)
Hirakata city, Osaka Prefecture	Unspecified	Domestic and imported seafood purchased from three food markets	PCBs 81, 77, 123, 118, 114. 105, 126, 167, 156, 157, 169, 180, 170, 189	Range, 13–40 182 pg/g ww	Ohta *et al.* (2008b)
Lao People's Democratic Republic					
Vientiane (Agent Orange-non-sprayed capital)	2001	Meat, fish, and dairy products from food markets	PCBs 37, 77, 126, 169, 81, 28, 33, 55, 60, 66, 74, 105, 114, 118, 122, 123, 124, 156, 157, 167, 189, 52, 101, 128, 138, 153, 170, 180, 187, 194, 206, 209	Range, 0.004–0.186 pg TEQ/g in fish samples; 0.011– 0.063 pg TEQ/g in meat and dairy products	Schecter *et al.* (2003a)
Republic of Korea					
		Muscle of sport and market fish	22 PCB congeners	23.0 (4.48–95.6) ng/g ww (sport fish) 8.91 (2.96–68.2) ng/g ww (market fish)	Yim *et al.* (2005)
		40 species of marine organism	DL-PCBs	0.4×10^{-3} ($0.008–0.6) \times 10^{-3}$ WHO-TEQ ww	Moon & Ok (2006)
	2005 to 2007	26 marine species (n = 78) collected annually during 2005–2007 from a large fish market in Busan	PCBs 8, 18, 28, 29, 44, 52, 87, 101, 105, 110, 118, 128, 138, 153, 170, 180, 187, 194, 195, 200, 205, 206	Range, 0.2–41 ng/g ww	Moon *et al.* (2009)
Singapore					
Singapore (cont.)	June 2002 to June 2003	Twenty types of seafood from local supermarkets	PCBs 17, 18, 28/31, 33, 44, 49, 52, 70, 74, 82, 87, 90, 101, 95, 99, 105, 110, 118, 128, 132, 138, 149, 151, 153, 156, 169, 170, 171, 177, 180, 183, 187, 194, 199, 201, 205, 206, 208, 209	Mean, 3.72 ng/g ww (range, 0.61–28.47 ng/g ww)	Bayen *et al.* (2005)

DL-PCBs, dioxin-like polychlorinated biphenyl; ww, wet weight

Table 1.24 PCB concentrations in marine fish from China

PCB	PCB concentration (n/g ww)							
	South China Sea		Boahi Sea		East China Sea		Yellow Sea	
	Average concentration (n/g ww)	Contribution (%)	Average concentration (n/g ww)	Contribution (%)	Average concentration (n/g ww)	Contribution (%)	Average concentration (n/g ww)	Contribution (%)
PCB-25	0.10	5.0	6.7	10.7	38.8	7.5	111.9	11.1
PCB-52	0.13	6.4	4.6	7.3	40.8	7.8	64.2	6.4
PCB-101	0.35	17.3	8.6	13.7	48.3	9.3	88.1	8.7
PCB-118	0.22	10.7	12.1	19.3	43.9	8.4	106.2	10.5
PCB-138	0.66	32.4	11.2	17.8	167.1	32.1	336.5	33.4
PCB-153	0.39	18.8	16.3	26.0	136	26.2	248.8	24.7
PCB-180	0.19	9.4	3.3	5.3	45.0	8.7	52.4	5.2
Σ 7 PCBs	2.0	–	62.8	–	520	–	1008	–

PCB, polychlorinated biphenyl; ww, wet weight
Data from Liu *et al.* (2011)

upper bound (lower bound) for the 50th, 90th and 95th percentiles were < 0.005 (< 0.005), 0.02 (0.01) and 0.03 (0.01) pg WHO_{2005}-TEQ/g wet weight for PCDD/PCDF, respectively. For the total TEQ, the upper bound (lower bound) concentrations were 0.01 (< 0.005), 0.04 (0.02) and 0.07 (0.04) pg WHO_{2005}-TEQ/g wet weight, respectively (EFSA CONTAM, 2012). Infant formulae showed upper bound concentrations below the current maximum levels (0.2 pg WHO_{2005}-TEQ/g wet weight), with highest concentrations found in ready-to-eat meals containing fish or meat. Overall, a decrease in concentrations of DL-PCBs was observed for the three food groups available: "raw milk and dairy products," "hen eggs and egg products" and "muscle meat from fishes other than eels." Feed and food of animal origin contained higher concentrations of PCDD/PCDF and DL-PCBs combined (the non-*ortho* PCBs were the main contributors to the total TEQs) than foods from plant origin. PCB-153, PCB-138, and PCB-180 represented altogether 36.9–97.8% of the sum of PCB_6. The maximum levels were exceeded in 9.7% of the food samples and 2.3% of the feed samples for PCDD/PCDF and DL-PCBs combined, and in 3.0% of the food samples and 2.4% of the feed samples for the PCB_6. With respect to food categories, lower PCB concentrations were found in meat from sheep, eggs from battery rearing, farmed salmon and trout, and farm milk (which however showed higher concentrations of PCDD/PCDF and DL-PCBs combined than milk from bulk) (EFSA, 2012).

The Baltic Sea area is heavily contaminated with persistent organochlorine compounds, including PCBs (Kiviranta *et al.*, 2003), as is clearly attested by samples of fatty fish from the eastern coast in Sweden (Svensson *et al.*, 1995). In the most contaminated feed group, the highest relative contribution to the WHO_{2005}-TEQ_{total} came from non-*ortho* PCBs, up to twice the average contribution (EFSA, 2012).

(b) Estimated daily dietary intake

In Europe, more than 90% of PCB exposure in the general population is via food consumption (EFSA, 2005; Table 1.25). Average daily dietary intakes of the sum of PCB_6 are in the range of 10–45 ng/kg bw for adults, and two and a half times higher in children. Limited exposure data for young children indicate that the average daily intake (breastfeeding excluded) of the sum of PCB_6 is about 27–50 ng/kg bw. Overall, the non-*ortho* PCBs represented 21.0–74.9% of the WHO_{2005}-TEQ_{total} of PCDD/PCDF and DL-PCBs combined in food (EFSA, 2012), and the mono-*ortho* PCBs represented no more than 12% of the WHO_{2005}-TEQ_{total}. In the most contaminated samples, such as products from aquatic animals and from ruminants, the relative contribution of the non-*ortho* PCBs ranged from 34.2% to 86.1%. Most likely due to an effect of the European risk management measures, a decrease in exposure to the sum of PCB_6 was observed between 2002–2004 and 2008–2010 in most but not all population groups, and it was estimated between 2.0% and 75.6%.

In the USA, the daily dietary intake of PCBs for adults decreased from 1978 (0.027 µg/kg bw) until 1986–1991 (< 1 ng/kg bw) (IPCS, 2003). Mean daily intakes for infants during the same period decreased from 11 to < 1 ng/kg bw. However, trends during 1991–1997 did not appear to decrease, and ranges of daily dietary intake were 3–5 ng/kg bw for adults, and 2–12 ng/kg bw for children of different ages (IPCS, 2003).

Daily dietary intake of PCBs from countries in Asia are presented in Table 1.26. In China, the estimated daily intake from four food groups of animal origin ranged from 0.09 to 0.59 pg TEQ/kg bw for DL-PCBs, which is lower than the daily intake in some developed countries (Liu *et al.*, 2013). A survey of food items on the market and typical consumption patterns in Japan reported a daily intake for the general population of 2.60 pg TEQ/kg bw per day (Koizumi *et al.*, 2005). Of

Table 1.25 Dietary exposure to PCBs for an average consumer on the European market

Food group	Mean ΣPCBs (ng/g)	Consumption (g/day)			Exposure (ng/day)		
		Italy	France	Sweden	Italy	France	Sweden
Cereals and cereal products	0.0213	270	218	292	6	5	6
Fruits and vegetables	0.0495	498	313	387	25	15	19
Eggs	0.73	18	17	15	13	12	11
Fats and oils	5.05	38	18	24	192	91	121
Meat and meat products	1.52	134	117	143	204	178	218
Offals	0.74	3	3	7	2	2	5
Fish and fish products	12.50	43	32	35	538	400	438
Milk	0.17	124	106	343	21	18	59
Cheese and dairy products	0.98	87	100	45	86	98	44
Total (ng/kg bw per day)		–	–	–	18.1	13.7	15.4
Total (ng/kg bw per day) for a high consumer of meat and meat products		–	–	–	22.0	17.6	18.9
Total (ng/kg bw per day) for a high consumer of fish and fish products		–	–	–	40.4	31.8	33.3

PCB, polychlorinated biphenyl
Adapted from EFSA (2005)

Table 1.26 Estimated daily dietary intake of PCBs in Asia

Country, region	Date	Source	PCBs measured	Mean daily intake	Reference
Japan, Fukuoka Prefecture	1969–70	Patients Individual consumption of oil was estimated by taking into account age, sex and the number of meals at home	PCBs, PCDFs, and PCQs	Estimated total intake: PCBs, 633 mg PCDFs, 3.4 mg PCQs, 596 mg	Hayabuchi *et al.* (1979)
Japan, eight sites from Hokkaido to Okinawa	1995 survey	Food duplicate study 40 women (mean age, 52 years)	11 PCB congeners (74, 99, 118, 138, 146, 153, 156, 163, 170, 180, and 182)	165.9 ng/day	Koizumi *et al.* (2005)
Japan, 75 different areas of 25 prefectures	Not reported	Food duplicate study 374 subjects, 86 men and 288 women (mean age, 48.0 years; range, 17–72 years)	12 PCBs	Mean PCB intake, 0.59 pg/kg bw per day Median PCB intake, 0.39 pg/kg bw per day	Arisawa *et al.* (2008)
Republic of Korea	2010	Estimated dietary intake 200 individual food samples from 40 different foodstuffs	62 PCB congeners, including 7 indicator PCBs and 12 DL-PCBs (PCB-1, 3, 4, 8, 10, 15, 18, 19, 22, 33, 37, 44, 49, 54, 70, 74, 87, 95, 99, 104, 110, 112, 128, 149, 151, 155, 158, 168, 170, 171, 177, 178, 183, 187, 188, 191, 194, 199, 201, 202, 205, 206, 208, and 209)	9.9 ng/kg bw per day	Son *et al.* (2012)

DL-PCBs, dioxin-like polychlorinated biphenyls; PCDFs, polychlorinated dibenzofurans; PCQs, polychlorinated quaterphenyls; ww, wet weight

these, 2.41 pg TEQ/kg bw per day was from ingestion of food, while inhalation and soil ingestion contributed only to 0.19 pg TEQ/kg bw per day. A "typical" Japanese person receives 120.7 pg TEQ per day through food consumption (mainly fish/shellfish, followed by meat/eggs).

In specific subpopulations with high dietary PCB exposure, such as Baltic Sea fishermen, the daily intake from fish of the sum of PCB_6 was estimated at 40 ng/kg bw, corresponding to a total daily intake of the sum of non-dioxin-like PCBs of 80 ng/kg bw, before taking into account the rest of the diet (Lindell, 2012).

In breastfed infants, the most recent WHO study of PCB exposure reported a mean daily intake of about 1600 ng/kg bw (range, 230–7300 ng/kg bw per day) for total PCB_6. Thus, exposure of infants to PCB_6 (and DL-PCBs) through human milk is about two orders of magnitude higher than the average daily intake by adults.

1.4.8 Occurrence in manufactured products other than commercial PCB preparations

In addition to commercial PCB preparations, many manufactured products contain PCBs as a result of contact with PCB products, as contaminants during manufacture, or as degradation products of other chlorinated compounds. For example, PCBs have been found in various paint pigments (Hu & Hornbuckle, 2010; Kuusisto *et al.*, 2006). Electronic equipment contains PCBs, which are released during dismantling.

Since the sampling and determination of the presence of PCBs is a difficult process, the Basel Convention has established a so-called "grey list" of materials and equipment that are suspected to contain PCBs (Basel Convention, 2003):

- Cable insulation
- Rubber and felt gaskets
- Thermal insulation material including fibreglass, felt, foam and cork
- Transformers, capacitors (also contained in electronic equipment)
- Voltage regulators, switches, bushings and electromagnets
- Adhesives and tapes
- Oil, including that contained in electrical equipment and motors, anchor windlasses, hydraulic systems
- Surface contamination of machinery and other solid surfaces
- Oil-based paint
- Caulking
- Rubber isolation mounts
- Foundations mounts
- Pipe hangers
- Light ballasts
- Plasticizers.

1.4.9 Population biomonitoring

(a) Blood

The presence of PCBs in serum or blood may reflect exposure from any source (Dewailly *et al.*, 1988). Results from different studies in humans have indicated that measurements of PCBs in serum generally reflect cumulative past exposure. Many PCB congeners can remain in the body for years after exposure, although some of the less chlorinated congeners are more volatile and consequently show shorter residence times.

(i) North America

Hopf *et al.* (2009a) provided an extensive review of reports on background levels of PCBs in the USA population. They concluded that serum concentrations increased up to 1979 and decreased after that, but that the background levels are still of concern. The NHANES survey over the period 2002–2004 reported increasing

concentrations of PCBs with age, and concentrations were higher in men than in women, and higher in African-Americans and Caucasians than in Mexican-Americans (Patterson *et al.* 2009). Sjödin *et al.* (2004) showed a decline in concentrations of PCB-153 between 1985 and 2002 in pooled samples from the NHANES study.

Several studies have looked at specific populations living near specific contaminated sites or eating contaminated fish (Table 1.27).

Serum concentrations for the sum of 17 congeners in Viet Nam veterans were 167.5 ng/L lipid adjusted, of which the major portion (116.6 ng/L) were di-*ortho* congeners (Schecter *et al.*, 1996).

Jarrell *et al.* (2005) determined the sum of 24 congeners in pregnant women in Canada, and reported a mean value of 0.78 ng/L wet weight.

Because the less chlorinated PCBs are more volatile, teachers working in a school where caulk containing PCBs was used showed serum congener profiles that were enriched in less chlorinated congeners (Herrick *et al.*, 2011).

DeCaprio *et al.* (2005) reported finding a pattern of PCB congeners in serum specific of young native Americans living near a PCB-contaminated waste site. This pattern was not clearly observed in older individuals because it was obscured by the greater concentrations of more persistent congeners, coming primarily from dietary exposure.

(ii) Europe

Several European studies on human biomonitoring have reported blood PCB concentrations in adults or children (summarized in Table 1.28). Past environmental contamination in industrial areas has polluted surrounding soils and forage, leading in turn to high blood PCB concentrations in the adult population. Age-related accumulation of PCBs has been observed in many studies (Patterson *et al.*, 1994; Apostoli *et al.*, 2005; Park *et al.*, 2007), and may be partially explained by historical high levels of exposure in the 1970s.

In Germany, Environmental Surveys (GerES) were carried out in 1998 (Becker *et al.*, 2002) and during 2003–2006 (Becker *et al.*, 2009). GerES data show mean blood concentrations for the sum of PCBs of 1.3–1.7 µg/L in 1998 and of 286 ng/L in the more recent survey, with strong difference (factor of 5.6) between age groups 18–25 and 66–69 years. In Belgium in 2007–2011 (Schoeters *et al.*, 2011), the Flemish Human Environmental Survey reported average blood PCB concentrations of 333 ng/g lipid. Average concentrations in the United Kingdom in 2003 were 170 ng/g lipid (Thomas *et al.*, 2006). In Spain in 2004–2008, concentrations of the most common PCBs were in the range of 21.8 to 38.9 ng/g lipid (Ibarluzea *et al.*, 2011). In France, blood analysis in the general adult population was first carried out in 1986 (Dewailly *et al.*, 1988) and then in 2006–2007 (French Nutrition and Health survey; Fréry *et al.*, 2013). The reported blood PCB concentrations in populations in industrial polluted areas such as Italy (Turci *et al.*, 2004; Apostoli *et al.*, 2005; Turrio-Baldassarri *et al.*, 2008) and Slovakia (Jursa *et al.*, 2006) were high compared with those in non-occupationally exposed populations such as in Sweden (Salihovic *et al.*, 2012). In the Faroe Islands (Denmark), high concentrations of PCBs and hydroxylated PCBs in serum samples from pregnant women were attributed to the traditional diet, made of pilot whale meat, blubber and other marine food (Fängström *et al.*, 2002).

The most frequently detected di-*ortho*-chlorine-substituted PCBs in population studies are PCB-138, PCB-153, and PCB-180 (Glynn *et al.*, 2000), accounting for 65–78% of the measured sum of total PCBs (Needham *et al.*, 2005). The seven PCB indicator congeners (118, 138, 153, 156, 170, 180, and 194) contributed to 99% of the total PCB levels, with a modest contribution from dioxin-like congeners (Apostoli *et al.*, 2005).

In several countries in the European Union, a clear decrease in blood concentrations of PCBs has been observed in the last two decades. Overall,

Table 1.27 Serum concentrations of PCBs after consumption of PCB-contaminated fish, North America

Country, region	Sample	PCBs measured	Mean ng/g (ppb)	Reference
North Canada, Nunavik	Inuit women, *n* = 159	Sum of 14 congeners	313.2 ± 2 Range, 71.3–1951.3	Muckle *et al.* (2001)
USA, St Lawrence River	Native American adults, *n* = 753	Sum of 101 congeners	4.39 ± 4.18	DeCaprio *et al.* (2005)
USA, St Lawrence River	Native American adolescents		0.71 ± 0.668 (if not breastfed) 0.95 ± 0.806 (if breastfed)	Schell *et al.* (2008)
USA, Great Lakes	Fish consumers, *n* = 293 Fishing-ship captains, men	Sum of 89 congeners in µg/L (ppb) wet weight	4.2 (2.7), in 1994–95 2.8 (2.0), in 2001–05 6.3 (5.0), in 1994–95 1.2 (0.9) – 3.8 (3.0), in 2001–05	Knobeloch *et al.* (2009)
USA, Anniston, Alabama	Adult residents, *n* = 394	Sum of 35 congeners	4.72 ± 11.05 Range, 0.09–170.42	Goncharov *et al.* (2011)

PCB, polychlorinated biphenyl

mean whole blood concentrations of PCB-138, PCB-153, and PCB-180 appear to have decreased by approximately 80% in 20 years (Link *et al.*, 2005; Hagmar *et al.*, 2006; Agudo *et al.*, 2009; AMAP, 2009). Nevertheless, compared with North America (CDC, 2005), serum concentrations of PCB-138, PCB-153, and PCB-180 were higher by two- to fivefold in Germany in 1998 (Heudorf *et al.*, 2002), or Italy in 2001–2003 (Turci *et al.*, 2004; Apostoli *et al.*, 2005; Needham *et al.*, 2005). Similarly, serum concentrations of hydroxylated PCBs and methylsulfonyl-substituted metabolites of PCBs were higher by two to threefold in a contaminated area in a study in Slovakia (Hovander *et al.*, 2006).

(iii) Asia

In Asia, PCB concentrations in several biological samples (including serum or whole blood, umbilical cord blood, hair, breast milk, adipose tissue, liver, kidney, and lung tissues) showed a wide range (Table 1.29; Schecter *et al.*, 2003a). Data specific to the Yusho and Yucheng patients are presented in Table 1.30 and Table 1.31, respectively.

(iv) South and Central America

Rodríguez-Dozal *et al.* (2012) analysed serum samples from pregnant women in Mexico for 19 congeners and Aroclor 1260. For Aroclor 1260 [calculated as the sum of PCB-138 and PCB-153 multiplied by 5.2], they reported regional differences (mean concentration, 31.1 ng/g lipid) and elevated concentrations from residents of Merida (maximum, 546.2 ng/g lipid). Trejo-Acevedo *et al.* (2012) measured serum PCB concentrations (sum of 14 congeners) from children living in a malaria-endemic area of Mexico, and reported a mean serum PCB concentration of 5892 ± 3895.7 ng/g lipid. In an analysis of PCB congeners in maternal blood of women in Sao Paulo State, Brazil, PCB-118, PCB-138, and PCB-153 were detectable in more than 70% of samples, and their concentrations were almost double in women from industrial areas compared with women from rural areas (Rudge *et al.*, 2012).

(v) Africa

Röllin *et al.* (2009) reported overall low blood concentrations of PCBs (99, 118, 138, 153, 170, 180 and 187) in delivering mothers from seven geographical regions in South Africa. Large regional differences were observed, with women

Table 1.28 Blood concentrations of PCBs in various European countries

Country	Reference/study	Period	Age (years)	Number	PCBs measured	Mean	95th percentile
France	Dewailly *et al.* (1988)	1986	Men: 38	569	Σ 7 PCBi	4020 ng/L	5000 ng/L
	Fréry *et al.* (2009)	2005	30–65	1030	138, 153, 180	347 ng/g lipid	714 ng/g lipid
	ANSES (2011)	2009–10	18–75	606 16	138, 153, 180	305 ng/g lipid 681 ng/g lipid	1368 ng/g lipid 3150 ng/g lipid
	Fréry *et al.* (2013)	2006–7	18–74	386	Σ 6 PCBi	287 ng/g lipid 1858 ng/L	721 ng/g lipid 4977 ng/L
Germany	GerES III GerES IV (2008)	1998 2003–6	18–69 7–14	2815 1079	138, 153, 180	1570 ng/L 286 ng/L	5000 ng/L 980 ng/L
United Kingdom	Thomas *et al.* (2006)	2003	22–80	151	Σ 31 congeners	170 ng/g lipid	670 ng/g lipid
Belgium	Schoeters *et al.* (2011)	2007–11	50–65	1530	138, 153, 180	333 ng/g lipid	
Italy	Turci *et al.* (2004)	2001–3		162	Total PCBs	2480 ng/L	5240 ng/L
	Apostoli *et al.* (2005)	2003	20–79	311	Σ 24 congeners	897 ng/g lipid	2643 ng/g lipid
	Turrio-Baldassarri *et al.* (2008)	2004	Men: 51	94	Σ 6 congeners	866 ng/g lipid	
Slovakia	Jursa *et al.* (2006)	2001–2	20–70	315	Σ 45 congeners	CA:5863 ng/g lipid RA:1245 ng/g lipid	Max: 55 334 ng/g lipid Max: 9015 ng/g lipid
	Park *et al.* (2007)	2002–4	Pregnant	CA: 762 RA: 341	118, 153, 105, 138, 180, 170	CA: 734 ng/g lipid RA: 351 ng/g lipid	CA:2105 ng/g lipid RA: 469 ng/g lipid
Spain	Agudo *et al.* (2009)	1992–6	35–64	953	138, 153, 180	459 ng/g lipid	
	Ibarluzea *et al.* (2011)	2004–8	Pregnant	1259	138, 153, 180	88 ng/g lipid	
Sweden	Salihovic *et al.* (2012)	2001–4	70	Men: 495 Women: 517	138, 153, 180	Men: 600 ng/g lipid Women: 517 ng/g lipid	753 ng/g lipid 664 ng/g lipid

CA, contaminated area; PCB, polychlorinated biphenyl; RA, reference area; PCBi, indicator PCBs

Table 1.29 PCB concentrations in biological samples from populations in Asia

Country, region, Year	Subjects, participants	Samples	PCBs measured	Mean concentrations (standard deviation or range)	Reference
Taiwan, China 1994	Pooled blood of 50 women	Blood serum	PCBs 28, 52, 74, 66, 101, 153, 138, 187, 183, 156, 157, 180, 170	386 ng/g lipid	Guo *et al.* (1997)
Central Taiwan, China 2001	30 primiparous women (mean age, 27.8 yr; range, 20–35 yr)	Breast milk	PCBs 77, 81, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189	4.87 (SD 8.04) pg TEQ/g lipid	Chao *et al.* (2003)
Central Taiwan, China 2000–1	20 pregnant women; mean age, 28 yr (range, 25–35 yr)	Placenta, milk, venous blood, and cord blood	12 DL-PCBs and 6 indicator PCBs	DL-PCBs: 5292 pg/g lipid in placenta, 10 170 pg/g lipid in milk, 9496 pg/g lipid in venous blood, and 3577 pg/g lipid in cord blood Indicator PCBs: 32 457 pg/g lipid in placenta, 55 425 pg/g lipid in milk, 36 416 pg/g lipid in venous blood, and 37 758 pg/g lipid in cord blood	Wang *et al.* (2004)
Taiwan, China 2004	Pooled blood plasma of 10 blood donors	Blood plasma	33 PCB congeners included PCB-8, 37, 44, 49, 52, 60, 66, 70, 74, 77, 82, 87, 99, 101, 105, 110, 114, 118, 126, 128, 138, 153, 156, 157, 158, 166, 169, 170, 179, 180, 183, 187, and 189.	187 ng/g lipid	Hsu *et al.* (2005)
East China July 11–13, 2006	64 male workers, aged 18–60 yr	Hair	PCBs (1668A-LCS, 1668A-IS)	Mean 1 600 pg/g dw (55 400–7 200 000 pg/g dw)	Wen *et al.* (2008)
China, Zhejiang April 2007 to January 2008	Surgical patients newly diagnosed for cancer (mean age, 65 yr; range, 32 to 94 yr)	Kidney, liver and lung tissues	27 PCB congeners	Median (range) in ng/g lipid: 382.15 (86.92–1403.92) (kidney); 460.00 (89.19–1742.57) (liver); 304.64 (104.85–373.25) (lung)	Zhao *et al.* (2009)
China, Shenzhen July to November 2007	60 samples from primiparous women living in areas not polluted by POPs (mean age, 28 yr; range 20–34 yr)	Breast milk	PCBs 28, 52, 101, 138, 153, 180, 77, 81, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189	DL-PCBs: median (range): 4580 (1964–13 967) pg/g fat Indicator PCBs: 13.2 (3.4–39.2) pg/g fat	Deng *et al.* (2012)
China, Zhejiang Province 2008	74 women in rural areas (mean age, 25.0 yr; range, 19–29 yr) and in urban areas (mean age, 26.5 yr; range, 22–29 yr)	Breast milk	PCBs 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189, 28, 52, 101, 138, 153, 180	42 774 ± 27 841 pg/g lipid (urban group) 26 546 ± 11 375 pg/g lipid (rural group)	Shen *et al.* (2012)
India, six different locations, 2009	55 mothers, reproductive age, ranged 21–38 yr	Breast milk	$^{13}C_{12}$-labelled PCBs	3.1 to 5 400 ng/g lipid weight	Devanathan *et al.* (2012)

Table 1.29 (continued)

Country, region, Year	Subjects, participants	Samples	PCBs measured	Mean concentrations (standard deviation or range)	Reference
India, Bangalore and Chidambaram 2007	25 e-waste recycling workers	Serum	62 PCB congeners	360 pg/g ww	Eguchi *et al.* (2012)
India, Bangalore and Chidambaram 2007	20 residents near a coastal area	Serum	62 PCB congeners	140 pg/g ww	Eguchi *et al.* (2012)
Islamic Republic of Iran, Ahvaz and Noushahr cities, and the countryside of Noushahr November 2007 to January 2008	16 pregnant women in Noushahr (mean age, 26 yr; range, 16–43 yr) 21 pregnant women in Ahvaz (mean age, 27 yr; range, 18–36 yr) 19 pregnant women in countryside of Noushahr (mean age, 25 yr; range, 15–36 yr)	Hair	PCBs 28, 52, 101, 118, 138, 143, 153, 180	Median (range): 9 (4–140) ng/g in Noushahr 8 (4–14) ng/g in Ahvaz 2 (undetected –15) ng/g in Noushahr countryside	Dahmardeh Behrooz *et al.* (2012)
Japan, Fukuoka Prefecture April to June, 1991	Nine normal women (mean age, 30 yr; range, 25–32 yr)	Breast milk	PCB-77, PCB-126, PCB-169	Mean coplanar PCBs, 21.3 pg TEQ/g fat Mean PCB-77: 12.4 pg/g fat; Mean PCB-126: 183.7 pg/g fat; Mean PCB-169: 65.7 pg/g fat TEFs as proposed by the NATO-CCMS (1988), and those of the coplanar PCBs were calculated using data reported by Safe (1990).	Matsueda *et al.* (1993)
Japan September 1994 to November 1996	31 normal volunteers (age, 20–61 yr)	Sebum, and blood	PCB-77, PCB-126, PCB-169	Mean PCBs, 447.3 pg/g lipid (sebum), and 204.6 pg/g lipid (blood)	Iida *et al.* (1999)
Japan 1998–9	28 patients with various illnesses (age, 19–87 yr)	Liver and adipose tissue	Non-*ortho*-PCBs	Mean (range): 20 (2.8–91) TEQ/g lipid (liver tissue) 17 (2.7–57) pg TEQ/g lipid (adipose tissue)	Takenaka *et al.* (2002)
Japan 1999–2000	80 women (mean age, 36.9 yr; range, 26–43 yr)	Serum	36 PCBs	Median, 0.46 (25th percentile, 0.35; 75th percentile, 0.66) nmol/g lipid	Tsukino *et al.* (2006)

Table 1.29 (continued)

Country, region, Year	Subjects, participants	Samples	PCBs measured	Mean concentrations (standard deviation or range)	Reference
Japan, Fukuoka Prefecture 2002–3	127 normal controls (age, 68.0 yr; SD, 5.4 yr)	Blood/serum	PCB-77, PCB-126, PCB-169	11.9 pg TEQ/g lipid	Todaka *et al.* (2007a)
Japan Born 1950–86	15 samples from 9 healthy subjects	Preserved umbilical cord	Dioxin-like PCBs (81, 77, 123, 118, 114, 105, 126, 167, 156, 157, 169, 189)	Mean (range): 2700 (250–12 000) pg/g	Aozasa *et al.* (2008)
Japan, Sapporo City July 2002 to July 2004	101 primiparous pregnant women (mean age, 28.8 yr; range, 18–40 yr) and 94 multiparous pregnant women (mean age, 32.3 yr; range, 28–47 yr)	Blood	PCBs 28, 44, 47/48, 49, 52/69, 56/60, 63, 66, 70, 71, 74, 85, 87, 92, 93/95/98, 99, 101, 107/108, 110, 117, 128, 130, 132, 134, 135, 137, 138, 139, 141, 146, 147, 151, 153, 163/164, 165, 170, 172, 177, 178, 179, 180, 181, 182/187, 183, 191, 194, 195, 196/203, 198/201, 200, 202, 205, 206, 207, 208, and 209	Mean (range): 114.5 ± 61.0 (42.2–329.3) ng/ g lipid (primiparous) 100.2 ± 48.2 ng/g lipid (31.5–258.0) (multiparous)	Todaka *et al.* (2008a)
Japan, Sapporo City, Hokkaido Prefecture July 2002 to July 2004	60 mothers (mean age, 31 yr; range, 21–47 yr)	Blood and breast milk	PCBs 77, 81, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189	Mono-*ortho* PCBs, 13.4 ± 5.8 ng/g lipid (blood) and 14.4 ± 8.2 ng/g lipid (breast milk) Non-*ortho* PCBs 97 ± 10 pg/g lipid (blood); and 60 ± 28 pg/g lipid (breast milk)	Todaka *et al.* (2008b)
Japan, Fukuoka and Nagasaki prefectures Born 1970–3	Five babies born to healthy mothers	Preserved umbilical cord	DL-PCBs (77, 81, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189)	0.1 pg TEQ/g dw	Nagayama *et al.* (2010)
Japan, Sapporo City July 2002 to October 2005	119 primiparous mothers (mean age, 30 yr; range, 21–40 yr)	Blood and breast milk	Non-*ortho* PCBs, mono-*ortho* PCBs, and 56 NDL-PCBs	120.2 ± 67.3 ng/g lipid (blood) 90.4 ± 51.6 ng/g lipid (breast milk)	Todaka *et al.* (2010)
Japan, Sapporo City July 2002 to October 2005	514 pregnant women (mean age, 32 yr; range, 22–41 yr)	Blood and breast milk	Non-*ortho* PCBs, mono-*ortho* PCBs, and 56 NDL-PCBs	Non-*ortho* PCBs, 77 ± 32 pg/g lipid (blood) and 51 ± 21 pg/g lipid (breast milk) Mono-*ortho* PCBs, 11.7 ± 5.7 pg/g lipid (blood) and 10.0 ± 5.2 ng/g lipid (breast milk) NDL-PCBs, 107 (16–326) ng/g lipid (blood) and 73 (12–252) ng/g lipid (breast milk)	Todaka *et al.* (2011)

Table 1.29 (continued)

Country, region, Year	Subjects, participants	Samples	PCBs measured	Mean concentrations (standard deviation or range)	Reference
Republic of Korea, Kyungpook May 2007 to May 2008	53 female myoma patients (mean age, 47 yr; range, 40–68 yr)	Adipose tissue	PCBs 8, 18, 28, 29, 44, 52, 87, 101, 105, 110, 118, 128, 138, 153, 170, 180, 187, 194, 195, 200, 205, 206	270 ± 140 ng/g lipid	Moon *et al.* (2012)
Russian Federation, Irkutsk Region 1992	Three groups of Siberians	Blood	PCBs 77, 126, 169	Mean TEQ, 2.0–25.2 ppt	Schecter *et al.* (2002)
Viet Nam, areas sprayed with Agent Orange 2006	Potentially exposed persons	Blood	Coplanar PCBs, mono-*ortho* PCBs	Coplanar PCBs TEQ, 1.1–5.6 pg/g lipid Mono-*ortho* PCBs TEQ, 1.8–7.3 pg/g lipid	Schecter *et al.* (2006)

DL-PCB, dioxin-like polychlorinated biphenyl; dw, dry weight; NDL-PCB, non-dioxin-like polychlorinated biphenyl; TEQ, toxic equivalent; ww, wet weight; yr, year

Table 1.30 PCB, PCDF, and PCDD concentrations in biological samples from the Yusho population, Japan

Region Period	Subjects/ participants	Sample	PCB measured	Concentration (mean, median, range)	Reference
Fukuoka, Saga, and Ishigaki cities 1970 in Saga; 1972 in Fukuoka; and 1972 in Ishigaki	*n* = 11 in Saga; *n* = 19 in Fukuoka; and *n* = 12 in Ishigaki	Adipose tissue, and breast milk	Mean PCBs	In Saga, PCBs in adipose tissue, mean, 2.6 (range, 0.5–5.3) ppm fat basis In Fukuoka, PCBs in breast milk, mean, 1.2 (range, 0.3–5.6) ppm fat basis In Ishigaki, PCBs in breast milk, mean, 0.4 (0.1–0.7) ppm fat basis	Masuda et al. (1974)
Japan 1973		Blood (*n* = 41), adipose tissue (*n* = 6), liver (*n* = 5)	PCB-118, 105, 153, 132, 156, 170, 180	Mean, 6.7 ppb in blood Mean, 2.5 ppm in adipose tissue Mean, 0.1 ppm in the liver	Masuda & Yoshimura (1982)
Fukuoka Prefecture 1981	59 Yusho patients aged > 40 years not receiving antihypertensive treatment	Blood/serum	Total PCBs	5.1 ± 2.3 ppb for men 6.4 ± 5.3 ppb for women	Akagi & Okumura (1985)
Japan 1988	259 patients (136 men and 123 women)	Blood/serum	Specific congeners not mentioned	Geometric means of PCBs and triglyceride: 3.84 (95% CI, 3.54–4.17) ppb and 114.3 (95% CI, 106.6–122.6) mg/dL, respectively Arithmetic mean of PCBs: 4.8 ppb (range, 0.6–320 ppb)	Hirota et al. (1993)
Japan September 1994 to November 1996	39 Yusho patients	Sebum, blood serum	PCB-77, PCB-126, PCB-169	428.1 pg/g lipid in sebum, and 390.7 pg/g lipid in blood	Iida et al. (1999)
Japan 2002	279 Yusho patients	Blood/serum	PCB-77, PCB-81, PCB-126, PCB-169	3.383 ± 2.765 (range 0.25–25.1) ppb	Uenotsuchi et al. (2005)
Japan 2002–3	279 Yusho patients in 2002 and 269 Yusho patients in 2003.	Blood/serum	PCB-77, PCB-81, PCB-126, PCB-169	125.0 pg-TEQ/g lipid	Todaka et al. (2005)
Fukuoka Prefecture 2002	279 Yusho patients and 92 Yusho-suspected persons	Blood/serum	PCBs 81, 77, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189	Yusho patients: Non-*ortho* PCBs, 12.3 pg TEQ/g lipid; mono-*ortho* PCBs, 25.0 pg TEQ/g lipid Yusho-suspected persons: Non-*ortho* PCBs, 10.0 pg TEQ/g lipid; mono-*ortho* PCBs, 8.8 pg TEQ/g lipid	Todaka et al. (2007a)

Table 1.30 (continued)

Region Period	Subjects/ participants	Sample	PCB measured	Concentration (mean, median, range)	Reference
Fukuoka Prefecture 2002–5	242 Yusho patients, 74 Yusho-suspected persons in 2004, and 237 Yusho patients and 114 Yusho-suspected persons in 2005	Blood/serum	PCB-77, PCB-81, PCB-126, PCB-169	Yusho patients: 12.3, 11.7, 10.6, and 11.0 pg TEQ/g lipid in 2002, 2003, 2004, and 2005, respectively Yusho-suspected persons: 10.0, 8.3, 8.3, and 10.5 pg TEQ/g lipid in 2002, 2003, 2004, and 2005, respectively	Todaka et al. (2007b)
Japan 2001–3	359 Yusho patients	Blood/serum	PCBs 81, 77, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189	3.14 ng/g blood	Imamura et al. (2007)
Fukuoka Prefecture 2004–7	242, 237, 300, and 96 Yusho patients from 2004 to 2007, respectively, and 74, 113, 125, and 148 Yusho-suspected persons, respectively	Blood/serum	Concentrations of 64 PCB congeners: TriCB-(28, 29), TetraCB-(44, 47/48, 49, 52/69, 56/60, 63, 66, 70, 71, 74), PentaCB-(85, 87, 92, 93/95/98, 99, 101, 105, 107/108, 110, 114, 117, 118, 123), HexaCB-(128, 130, 132, 134, 135, 137, 138, 139, 141, 146, 151, 153, 156, 157, 163/164, 167), HeptaCB-(170, 172, 177, 178, 179, 180, 181, 182/187, 183, 189, 191), OctaCB-(194, 195, 196/203, 198/201, 200, 202, 205), NonaCB-(206, 207, 208), DecaCB-209	Yusho patients: 2004, 645 (40–3032) ng/g lipid; 2005, 760 (40–4723) ng/g lipid; 2006, 667 (74–2432) ng/g lipid; and 2007, 510 (51–2252) ng/g lipid Yusho-suspected persons: 2004, 355 (20–1418) ng/g lipid; 2005, 490 (64–4055) ng/g lipid; 2006, 397 (18–1850) ng/g lipid; and 440 (19–2183) ng/g lipid	Todaka et al. (2009a, b)
Fukuoka Prefecture 2002–8	26 pairs of Yusho mothers and their children (19 mothers, 26 children)	Blood/serum	PCB-77, PCB-81, PCB-126, PCB-169	In the formula-fed group: 12.65 pg TEQ/g lipid for the mothers, and 3.85 pg TEQ/g lipid for the children In the breast-fed group: 10.64 pg TEQ/g lipid for the mothers; and 3.27 pg TEQ/g lipid for the children	Tsukimori et al. (2011)
Japan [Period not specified]	27 Yusho patients	Blood/serum	Hydroxylated PCBs (4-OH-CB109, 4-OH-CB146 + 3-OH-CB153, 4-OH-CB187, 4'-OH-CB172)	Total mean (range), 687 (95–1740) pg/g ww Range of the major hydroxylated PCB metabolites: 4-OH-CB187 (54–906 pg/g ww), 4-OH-CB146 +3-OH-CB153 (32–527 pg/g ww), 4-OH-CB109 (ND–229 pg/g ww) and 4'-OH-CB172 (ND–143 pg/g ww).	Tobiishi et al. (2011)
Japan 1968–2006 (the time of delivery of Yusho descendants)	64 Yusho mothers and 117 descendants (10 with FYD and 107 without FYD)	Maternal blood/serum	DL-PCBs (77, 81, 126, 169)	Black baby group, 57.6 pg TEQ/g lipid Non-black baby group, 31.8 pg TEQ/g lipid	Tsukimori et al. (2012)

Table 1.30 (continued)

Region Period	Subjects/ participants	Sample	PCB measured	Concentration (mean, median, range)	Reference
Umbilical cord					
Japan Yusho victims (1968–2000)	11 samples from 6 Yusho babies	Preserved umbilical cord	DL-PCBs (77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189)	6500 (130–11 000) pg/g in three designated patients 580 (130–1400) pg/g in eight suspected patients	Aozasa et al. (2008)
Fukuoka and Nagasaki prefectures Born 1970–3	7 babies born to Yusho mothers	Preserved umbilical cord	DL-PCBs (77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189)	0.3 pg TEQ/g dw	Nagayama et al. (2010)

dw, dry weight; FYD, fetal Yusho disease; PCB, polychlorinated biphenyl; PCDD, polychlorinated dibenzodioxins; PCDF, polychlorinated dibenzofurans; TEQ, toxic equivalent; ww, wet weight

Table 1.31 PCB concentrations in biological samples from Yucheng patients, Taichung County, Taiwan, China

Date of study	Patients	Sample	PCB measured	Concentration (mean, median, range)	Reference
1979–81	Children (n = 113)	Blood	PCBs	39 000 pg/g	Kashimoto *et al.* (1985)
1992	Mothers (n = 56)	Adipose tissue		2820 ± 300 (SE) ng/g	Guo *et al.* (1997)
1994–6	Adults (n = 42)	Sebum Blood	Dioxin-like PCBs	868.6 pg/g 714.4 pg/g	Iida *et al.* (1999)
1994	Adults (n = 414)	Serum	NR	1500 ng/g lipid (PCB-138 represented 29% of all measured PCBs)	Lung *et al.* (2005)
1994–5	Adults (n = 41)	Blood	NR	2468 ng/g lipid (13.3 ng/g sample) 133 pg/g (PCB TEQ in men) 127 pg/g (PCB TEQ in women)	Hsu *et al.* (2005) Lambert *et al.* (2006)

NR, not reported; PCB, polychlorinated biphenyl; SE, standard error; TEQ, toxic equivalent

from rural areas having the lowest levels of PCBs. PCB-138 and PCB-153 were found in the blood of mothers from all of the 61 sites studied at geometric mean concentrations of 3.56 and 3.2 ng/g lipid, respectively. Ahmed *et al.* (2002) reported the sum concentration of 29 congeners in blood from Egyptian women to be 61.9 ng/g. Weiss *et al.* (2006) reported concentrations of PCB-153 in infertile women in the United Republic of Tanzania to be 0.17 µg/kg. Sum PCB concentrations in serum samples from Bizerte, Tunisia, ranged from 37.5 to 284.6 ng/g lipid, with mean and median value of 136.1 ng/g lipid and 123.2 ng/g lipid, respectively. The PCB profile consisted mainly of persistent congeners such as PCB-138, PCB-153, and PCB-180 (82.7% of the sum of PCBs). PCB concentrations were significantly higher in men ($P < 0.05$) than in women (Ben Hassine *et al.*, 2014).

(b) Human milk

Due to its high fat content, human milk can accumulate large amounts of PCBs, thus making it an ideal matrix for the determination of concentrations of PCBs and other lipophilic compounds, and can be sampled using non-invasive techniques. In addition, human milk represents a good indicator of the body burden of lipophilic non-metabolized PCBs, since fat is mobilized for the production of milk during lactation. Animal studies and mass balance studies for humans have revealed that large amounts of PCBs can be eliminated through lactation (Lindell, 2012). Data are summarized in Table 1.32.

(i) Global assessment

The transfer of PCBs from mother to infants via breast milk is an important source of exposure, and several factors (including maternal residence, age, and parity) can potentially affect levels of contaminants in breast milk. Because of the importance of breastfeeding for infants, contamination of human milk is of specific public concern.

The Stockholm Convention on Persistent Organic Pollutants (POPs) is a guidance document, the objective of which is to document the effectiveness of the implementation of the obligations under the Convention. The World Health Organization (WHO) introduced worldwide measurement campaigns to determine the exposure of infants to dioxin-like PCBs (UNEP, 2012).

Table 1.32 PCB concentrations in human milk, by country

Country, population	PCBs (WHO-TEQ pg/g fat)			Sum indicator PCBs (ng/g fat)[a]			Reference
	Mean	Median	Range	Mean	Median	Range	
Europe, 1992–2003							
Czech Republic	–	15.24	14.32–28.5	–	502	496–1009	Van Leeuwen & Malisch (2002)
Germany	–	13.67	12.8–14.3	–	220	188–238	Ulaszewska *et al.* (2011)
	DL-PCBs: 12.60 (in Duisburg) 6.31 (in Munich)	–	–	–	–	–	
Greece	–	6.56 DL-PCBs	–	–	–	–	Costopoulou *et al.* (2006)
Italy (Milan, Rome, Venice)	–	16.29	11.02–19.33	–	253	195–323	Weiss *et al.* (2003), Ingelido *et al.* (2007), Abballe *et al.* (2008)
	–	–	DL-PCBs, 6.02–19.21 pg WHO_{2005}-TEQ/g lipid	–	–	–	
Norway	–	8.9	6.56–9.61	–	119	106–132	Polder *et al.* (2008)
Spain	–	–	–		(Sum of PCB-138, PCB-153, PCB-180) × 1.7: 1355 (in 1994) 653 (in 2000)	–	Cerná *et al.* (2008)
Spain	–	–	–	–	241	162–467	Schuhmacher *et al.* (2009)
	DL-PCBs, 4.8 pg WHO_{2005} TEQ/g lipid	–	–	–	–	–	
Sweden	–	9.71	–	–	146	–	Norén & Meironyté (2000)
North America							
Canada, *n* = 86 women eating fish from Lake Ontario	–	–	153 (50th percentile)	–	–	–	Stewart *et al.* (2003)
Western Canada, *n* = 47 women	–	38.20	–	–	–	–	Jarrell *et al.* (2005)
Canada, Northern Quebec, Inuit women from Nunavik	–	385.0 ± 1.9 SD	75.7–1915.8	–	–	–	Muckle *et al.* (2001)
USA, North Carolina, *n* = 331 women	–	77	9–708	–	–	–	Pan *et al.* (2010)

Table 1.32 (continued)

Country, population	PCBs (WHO-TEQ pg/g fat)			Sum indicator PCBs (ng/g fat)[a]			Reference
	Mean	Median	Range	Mean	Median	Range	
South and Central America							
Brazil, Rio de Janeiro, *n* = 40 mothers	9.7	–	150	–	–	–	Paumgartten *et al.* (2000)
Africa							
Ghana, *n* = 67 mothers	62	–	15–160	–	–	–	Asante *et al.* (2011)
South Africa, Limpopo Provence	10	–	–	–	–	–	Darnerud *et al.* (2011)
Tunisia	180	–	–	–	–	–	Ennaceur *et al.* (2008)
Zimbabwe	26	–	–	–	–	–	Chikuni *et al.* (1997)
Asia							
Japan	1.30×10^3 (in 1972) 1.51×10^3 (in 1974) 0.20×10^3 (in 1998)						Environment Agency of Japan (1999)
China, *n* = 1237				–	–	–	Li *et al.* (2009)
Total TEQ	5.42	5.11	Upper bound, 2.59–9.92	–	–	–	
Estimated dietary intake of PCDD/PCDF + DL-PCBs in infants	28.0 pg TEQ/kg bw per day	–	14.2–48.6 pg TEQ/kg bw per day	–	–	–	

[a] Indicator PCBs are PCBs 28, 52, 101, 138, 153 and 180

DL-PCB, dioxin-like polychlorinated biphenyl; PCB, polychlorinated biphenyl; TEQ, toxic equivalent

The evaluation of the Stockholm Convention has been applied (with slight changes) for five rounds of the UNEP/WHO survey. Often, human milk from primiparae mothers (for detail, see UNEP, 2012) is preferred to human blood, since sampling is non-invasive and PCBs are easier to detect (due to the higher lipid content of milk). It should be noted that for global assessment, the concentrations of dioxin-like PCBs (DL-PCBs) on a TEQ basis for the last three rounds of the UNEP/WHO survey on mothers' milk may be lower by 30% (range, 2–60%) if WHO toxic equivalency factors (TEFs) for 2005 (WHO_{2005}-TEF) are applied, rather than those for 1998 (WHO_{1998}-TEF). International chemical assessments report that the average concentration of PCBs in human milk fat ranges from 0.5 to 4 μg/g (IPCS, 2003). For the sum of PCB_6, the median is between 10.8–30.7 ng/g lipid, and maxima are between 37.1–65.8 ng/g lipid. Overall, the UNEP/WHO survey showed a correlation between maternal age and concentrations of DL-PCBs in breast milk, and lower concentrations of PCBs in breast milk of multiparous women when compared with primiparous women.

(ii) Americas

The mean concentration of PCBs in whole breast milk in Canadian women steadily increased from 6 μg/kg in 1970 to 12 μg/kg in 1975, and to 26 μg/kg in 1982, before declining to 6 μg/kg in 1986 (IPCS, 2003).

Recent data on concentrations of PCDD/PCDFs and DL-PCBs in human milk from South America were reported only for Brazil (Paumgartten *et al.*, 2000).

(iii) Europe

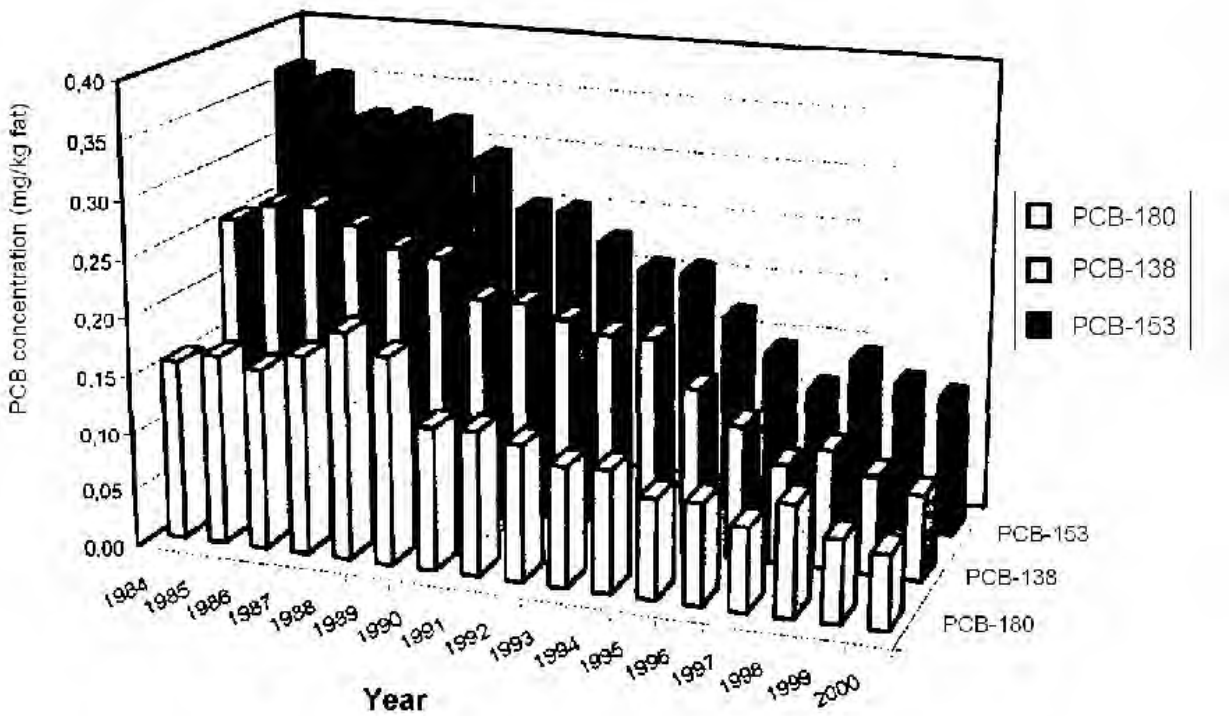
In Europe, concentrations of DL-PCBs (on a TEQ basis) and PCB indicators in human milk are considerably higher than in other regions of the world, a legacy from past exposures. For the sum of PCB_6, the median of 115.3 ng/g lipid is between 3.8 times and 10.7 times higher than in other regions of the world, and maximum concentrations are up to 14.9 times higher (IPCS, 2003). However, a WHO survey identified a decrease in WHO-TEQ PCDD/PCDF and PCB concentrations in human milk over the last decade (Van Leeuwen *et al.*, 2002). It was assumed that this decrease was the result of the ban of PCB use in open systems, and the strict regulations on the use of PCBs and on their disposal in closed systems.

Norén & Meironyté (referenced in IPCS, 2003) reported a steady decrease (from 910 to 324 ng/g lipid) in total PCB concentrations in the breast milk of Swedish women between 1967 and 1997. A declining trend could be observed for the sum of the PCB_6 in Germany, and mean values for the congeners PCB-138, PCB-153 and PCB-180 were approximately 60–70% lower in 2000 than in 1984 (Fürst, 2001; Fig. 1.8). An approximately 74% decrease in DL-PCB concentrations during the last decade was reported in Italy (Di Domenico & Turrio Baldassarri, 1990; Weiss *et al.*, 2003; Abballe *et al.*, 2008). Analyses of milk samples from the Czech Republic also revealed a decline in median concentrations between 1994 and 2000, the strongest decrease being observed between 1994 and 1997 (Cerná *et al.*, 2008). Nevertheless, it should be noted that concentrations in areas with heavy contamination did not show a significant decline in exposure over the past 10 years.

(iv) Asia

In Japan, a time-trend study showed that average PCB concentrations in human milk increased from 1.3 ng/g in 1972 to a peak of 1.5 ng/g in 1974, and then decreased by about 13% in 1998 (Environment Agency of Japan, 1999). In contrast, daily intake of PCBs from breast milk was estimated to decrease from 22.3 μg/g to 0.31 μg/g during this same period. [This trend reflects a change in PCB concentrations in food, due to both a decrease in contamination and more dependence on imported foods, which were less

Fig. 1.8 PCB concentrations in human milk in Germany, 1984–2000



From Fürst (2001)
PCB, polychlorinated biphenyl

contaminated than domestic foods (IPCS, 2003), and is consistent with the observed decline in PCB concentrations in the environment and in human tissues.]

In China, a national investigation of individuals in 12 provinces representing approximately 50% of the total Chinese population reported PCDD/PCDF-TEQ and total-TEQ in human milk from rural areas to be lower than those from urban areas (Li *et al.*, 2009). Positive correlations were found between total-TEQ in human milk and the consumption of aquatic food and meat.

PCB levels in breast milk samples from women in Asia are summarized in Table 1.29 and Table 1.30.

(c) Adipose tissue

(i) North America

Lordo *et al.* (1996) reported PCB concentrations (sum of tetra- to octochlorobiphenyls) in pooled adipose tissue to be 672 ng/g in 1986, compared with 407 ng/g in 1982, and 508 ng/g in 1984. Stellman *et al.* (1998) reported a total PCB concentration of 267 ng/g in breast adipose tissue of healthy women from Long Island, New York. An approximation of Aroclor 1260 [summed concentrations of PCB-138 and PCB-153 multiplied by 5.2] measured in breast adipose tissue, was reported to be 870 ng/g (Aronson *et al.*, 2000). Muscat *et al.* (2003) measured PCB concentrations (sum of 14 congeners) in breast adipose

tissue in women without metastatic breast cancer to be 361 ± 235.9 ng/g and 395.4 ± 279.3 ng/g, in women who did not have recurrence and women who did have recurrence, respectively.

(ii) Europe

The results of a study conducted in 1993–94 suggested that concentrations of PCBs in adipose tissue are the best indicator of long-term exposure or of total body burden of PCBs, compared with human milk or blood (Kocan *et al.*, 1994).

PCB concentrations in adipose tissue of the general population in industrialized countries vary very widely, ranging from < 1000 to 5000 ng/g fat (Falandysz *et al.*, 1994; Holoubek *et al.*, 1995, 2001b). In a comparative study in Europe (Van Bavel *et al.*, 2003), PCB concentrations in the population in Sweden were one third (mean, 661.9 ng/g fat; range, 247.2–1651.2 ng/g fat; Σ37 PCBs) of those in the Hungarian samples.

(iii) South and Central America

Breast adipose tissue in 76 women from an agricultural region of north-eastern Argentina contained eight PCBs at very low levels (only 1.3% above detection limits), but high levels of *p,p*-dichlorodiphenyldichloroethane (DDE) and other pesticides (Muñoz-de-Toro *et al.*, 2006).

The sum of four PCB congeners in children from Nicaragua was 530 ng/g lipid weight (2.0 ng/g wet weight) in those living and working near a waste-disposal site and eating fish from contaminated Lake Managua, 230 ng/g lipid weight (0.9 ng/g wet weight) in those living nearby but not working at the waste site and not eating fish, and 160 ng/g lipid weight (0.6 ng/g wet weight) in those living at a distance from the waste site and not eating fish (Cuadra *et al.*, 2006).

(iv) Asia

PCB concentrations in adipose tissue were reported from Yusho and Yucheng patients (see Table 1.30 and Table 1.31).

(v) Adipose versus serum measurements

Arrebola *et al.* (2012a, b) measured concentrations of three PCB congeners in serum and adipose tissue in adults from Bolivia. PCB-138 had median concentrations of 0.2 ng/mL in serum [33.7 ng/g lipid] and 84 ng/g in adipose tissue [105 ng/g lipid]. The median values for PCB-153 was 0.3 ng/mL in serum [59.0 ng/g lipid], and 52.7 ng/g in adipose tissue [65.8 ng/g lipid]. PCB-180 had median values of 0.1 ng/mL in serum [26.7 ng/g lipid] and 32.8 ng/g in adipose tissue [41.0 ng/g lipid].

(d) Umbilical cord blood, placenta, and fetal tissue

(i) North America

Stewart *et al.* (2000) reported cord blood PCB concentrations from women living along Lake Ontario and eating contaminated fish. The average cord blood PCB concentration was 0.525 ng/g wet weight [25th percentile, 0.174 ng/g wet weight; 75th percentile, 1.11 ng/g wet weight]. In plasma from umbilical cord in Inuit women from northern Canada, the geometric mean for the sum of 14 PCB congeners was 279.9 ng/g lipid (range, 70.8–1420.1 ng/g lipid) (Muckle *et al.* 2001). Dallaire *et al.* (2003) reported changes in concentrations in umbilical cord blood in this population over time, and found a 7.9% annual decrease between 1994 and 2000. Choi *et al.* (2006) measured 51 congeners in cord blood from women living near a PCB-contaminated site in Massachusetts, and reported a geometric mean of 0.40 ng/g (range, 0.068–18.14), with no consistent relationship with residential distance from the waste site. Consumption of meat and local dairy products (but not fish) were associated with higher cord blood PCB concentrations.

In women from New York state, Schecter *et al.* (1998) reported the concentration of three dioxin-like PCBs to be 18.2 pg/g lipid in placenta, giving a TEQ of 1.05. The concentrations of 14 single PCB congeners in plasma from Inuit

women from Nunavik and southern Quebec were highly correlated with those in placenta (Pearson's $r = 0.77–0.97$; $P < 0.001$), and concentrations in Inuit women were on average four times higher than in women from southern Quebec (Pereg *et al.*, 2002). Doucet *et al.* (2009) analysed placenta from Canadian women having elective abortions in 1998–2006 and reported annual average total PCB concentrations ranging from 7 to 70 ng/g lipid, with no clear time trend.

(ii) Europe

Koopman-Esseboom *et al.* (1994) used the concentrations of four congeners (PCB-118, PCB-138, PCB-153, and PCB-180), as measured in umbilical cord blood and in breast milk, as indicators of exposure of the developing fetus and breastfed infant. For these congeners, the correlation coefficients between maternal plasma, cord plasma and human milk were highly significant.

Soechitram *et al.* (2004) analysed PCBs (PCB-118, PCB-138, PCB-146, PCB-153, PCB-156, PCB-180) and hydroxylated metabolites of PCBs (PCB-107, PCB-136, PCB-146, PCB-153, PCB-172, PCB-187) in samples of maternal plasma and corresponding cord blood in the Netherlands. The calculated ratio for cord versus maternal blood was 1.28 ± 0.56 for PCBs and 2.11 ± 1.33 for hydroxylated PCBs, expressed per gram of lipid. A significant correlation between the respective maternal and cord concentrations for both PCBs and hydroxylated PCBs was found. The results indicated that approximately 50% and 30% of hydroxylated PCBs and PCBs, respectively, was transferred across the placenta to the fetus.

(e) Hair

(i) North America

Altshul *et al.* (2004) reported median PCB concentrations (sum of 57 congeners) in hair of 2640 ng/g fat (range, 1180–3620 ng/g fat) in a population of students in Boston, USA. Washing hair with shampoo decreased concentrations of PCBs by 25–33% on average, and up to 62% for less chlorinated congeners. [The Working Group considered that the analytical method was reliable and reproducible.] The concentrations of PCBs in hair were higher than in serum. Correlation between concentrations in hair and blood was moderate for the more persistent PCB congeners, with no or little correlation for the other congeners.

(ii) Europe

Covaci *et al.* (2002b) assessed PCB exposure in hair samples from Greece, Romania, and Belgium. Mean PCB concentrations in samples from Belgium were up to 14 ng/g hair, while concentrations in samples from Greece were about three times lower. Similar ratios of PCB-153 over total PCBs were found for all three countries.

(iii) Asia

One study measured PCB concentrations in the hair of pregnant women in various cities in the Islamic Republic of Iran (see Table 1.29).

1.5 Occupational exposure to PCBs

In 1978, an estimated 12 000 persons in the USA were exposed occupationally to PCBs (Lloyd *et al.*, 1976; NIOSH, 1977). Since the previous IARC evaluations of PCBs (IARC, 1978, 1987), occupational exposures to PCBs have changed, since most industrial countries have banned or partially banned their use. Nevertheless, the earlier references cited previously have been incorporated in the present monograph.

Earlier occupational exposures to PCBs occurred during PCB manufacture, capacitor and transformer manufacture and repair, production of carbonless copy paper, and accidental releases from these processes. More recent occupational exposures to PCB usually occur through PCB emissions via waste incineration, fires, and waste recycling.

1.5.1 PCB manufacture

The few available studies of occupational exposure among PCB-manufacturing workers have been performed in France, Italy, Japan, Poland and the USA. Workers' exposures during PCB manufacture have been mentioned since 1936 (Jones & Alden, 1936); air PCB concentrations ranged from 26 to 163 μg/m³. Evidence of extensive exposures was available from two larger clinical studies in Slovakia in which workers' blood concentrations were measured at 1160–9600 ng/g lipid (n = 242) (Langer *et al.*, 1997), and 4905–6540 ng/g lipid (n = 240) (Langer *et al.*, 2002). These workers had been employed at the plant for at least 5 years, but no information was given regarding the type of activities that they had performed.

1.5.2 Capacitor manufacture

Before PCBs were banned, capacitor manufacturers filled (impregnated) casings with wound paper and foil/plastic with the PCB-containing oil before the top was fastened (crimped, sealed, soldered). PCB exposure (probably via the dermal route) occurred during filling: the capacitors were either flood-filled or manually filled, resulting in spills and worker exposures. The brand of PCB oil used differed geographically (Aroclors were used in the USA, Pyralene/Phenochlor in Sweden and Italy) and temporally (the percentage chlorination was reduced, e.g there was a switch from Aroclor 1254, with 54% chlorination, to Aroclor 1242, with 42% chlorination.

Other chemical exposures in capacitor manufacturing were possible, such as from other impregnation oils (e.g. mineral oils), degreasing agents such as trichloroethylene (Brown & Jones, 1981; Bertazzi *et al.*, 1987), dibenzofurans (Gustavsson *et al.*, 1986), chlorinated naphthalenes, lead solder, epoxies, and methyl ethyl ketone (MEK) (Mallin *et al.*, 2004; Persky *et al.*, 2012). Ageing capacitors can from time to time explode [due to the physical stress of metal ageing], thus further exposing workers.

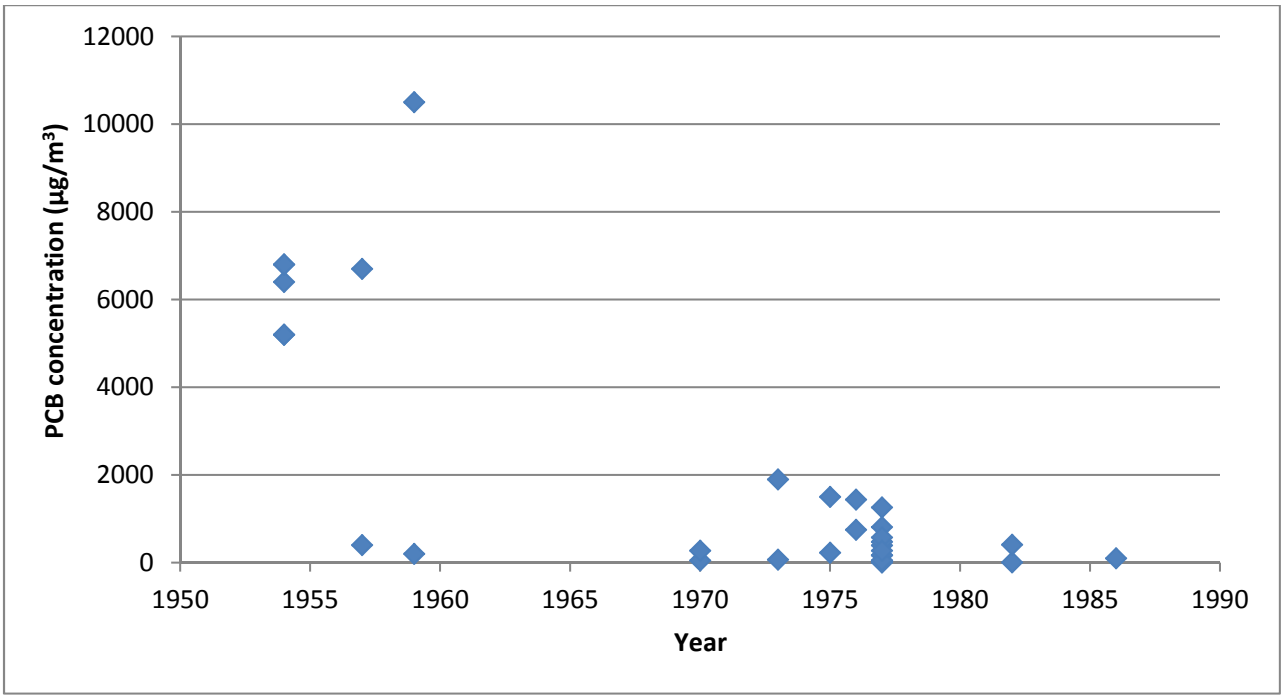
Fig. 1.9 shows ranges of air PCB concentrations in capacitor-manufacturing sites (Ouw *et al.*, 1976; Brown & Jones, 1981; Bertazzi *et al.*, 1982; Fischbein *et al.*, 1982; Lawton *et al.*, 1985; Gustavsson *et al.*, 1986). The earlier concentrations (measured in the 1950s–1960s) were highest (Bertazzi *et al.*, 1982), and decreased in later years.

In the 1950s, maximum PCB concentrations in workroom air in several plants in the USA (Massachusetts) were reported to be 200–10 500 μg/m³ (Elkins, 1959). No details on the number of plants surveyed, nor the number of samples collected, or the work performed in the plants were given, but four different jobs were surveyed: for impregnating with PCBs, average air concentrations ranged from 200 to 5800 μg/m³; for soldering, 800 μg/m³; mixing oil, 600 μg/m³, and regulator filling, 100 μg/m³. No toxic effects were noted at these concentrations; however, it was noted that air PCB concentrations of > 10 000 μg/m³ were "unbearably irritating." This is contrary to a report in a Japanese capacitor-manufacturing plant where a dermatitis outbreak occurred when air PCB concentrations reached 100 μg/m³ (Meigs *et al.*, 1954). Air PCB concentrations between 1953 and 1957 in a Japanese capacitor-manufacturing factory ranged from 400 to 6700 μg/m³ (NIEHS, 1976).

Ouw *et al.* (1976) reported that workers in the electrical industry in Australia were exposed to Aroclor 1242 at air concentrations of 320–2220 μg/m³, with a mean of 1270 μg/m³; and were found to have PCB blood concentrations of approximately 0.4 g/kg bw. Contact with PCBs was primarily via the skin.

Brown & Jones (1981) measured air concentrations of Aroclor 1016 in two plants in the USA (New York and Massachusetts) plants in 1977. The time-weighted averages (TWA) were different for the two plants, with air concentrations at the New York plant being lower than at the Massachusetts

Fig. 1.9 Air PCB concentrations in capacitor manufacturing plants (µg/m³) by year



Compiled by the Working Group using data from Ouw *et al.* (1976), Brown & Jones (1981), Bertazzi *et al.* (1982), Fischbein *et al.* (1982), Lawton *et al.*(1985) and Gustavsson *et al.*(1986)
PCB, polychlorinated biphenyl

plant. For the New York plant, PCB concentrations in personal air samples ranged from 24 to 393 µg/m³ (n = 28), and in area air samples from 3 to 476 µg/m³ (n = 19). For the Massachusetts plant, PCB concentrations in personal air samples ranged from 170 to 1260 µg/m³ (n = 29), and in area air samples from 50 to 810 µg/m³ (n = 25). Air PCB concentrations (TWA) were extremely high during capacitor impregnation (New York: 160 µg/m³, Massachusetts: 850 µg/m³), degreasing (Massachusetts; 1260 µg/m³), and sealing/soldering (New York: 393 µg/m³, Massachusetts: 720 and 1060 µg/m³). Capacitors that failed were sent for repair where they were re-opened and manually drained. Repair workers' PCB exposures were measured as 298 µg/m³ (recovery), and 50 µg/m³ (repair) in the New York plant. [These workers would also have had extensive dermal exposures, which were not assessed.]

Eight studies have reported PCB concentrations in workers' blood in Australia, Finland, Italy, Germany, and the USA (Karppanen & Kolho, 1972; Ouw *et al.*, 1976; Maroni *et al.*, 1981a; Bertazzi *et al.*, 1982; Acquavella *et al.*, 1986; Wolff *et al.*, 1992; Kannan *et al.*, 1994; Seegal *et al.*, 2011; Persky *et al.*, 2012). The reporting of PCB blood concentrations was not uniform, which hindered comparison across studies.

Karppanen & Kolho (1972) compared blood PCB concentrations in workers in a capacitor factory in Finland where Aroclor 1242 had been used as the impregnating fluid: the groups comprised laboratory workers handling PCBs (n = 6), impregnation workers (n = 11) employed for 4 years, and a control group (n = 9) that had never been professionally exposed to PCBs. Blood PCB concentrations were approximately 50 times greater in impregnation workers (0.07–1.9 µg/g)

than in the control group (0.003–0.012 μg/g), and were also higher in laboratory workers (0.036–0.062 μg/g) than in the controls. The pattern of PCB congeners in the exposed workers differed markedly from that of the PCBs actually used. More highly chlorinated PCBs persisted in the blood, while the less chlorinated PCBs contained in Aroclor 1242 had been eliminated from the body. [Consequently, the total PCB intake must have been higher than that reflected by the levels detected in blood.]

Total serum PCB concentrations have been reported to be 18.2 ppb (Acquavella *et al.*, 1986) in a clinical survey among 205 workers at a capacitor-manufacturing plant in the USA. This mean value represented workers (n = 205) with (39%) and without (61%) potential for PCB exposure in their jobs. [The Working Group noted that PCB concentrations were not reported separately for workers with and without occupational PCB exposure.] Log serum PCB concentrations were found to be significantly correlated with duration of employment, age, cumulative occupational exposure, and fish and wine consumption, as confirmed by multiple linear regression.

In a study of mortality in Italy (Bertazzi *et al.*, 1982), workers in the autoclave room were exposed to air PCB concentrations of 5200–6800 μg/m^3 in 1954 (n = 3), and 48–275 μg/m^3 in 1977 (n = 9). Eighteen workplace surface-wipe samples showed extensive PCB contamination (0.2–159 μg/cm^2), as did nine hand-wipe samples (0.3–9.2 μg/cm^2). Workers' serum PCB concentrations were reported by type of PCBs: for highly chlorinated PCBs (54% chlorination) (n = 67), the mean was 230.5 ppb (SD, 174.5), while for less chlorinated PCBs (42% chlorination) (n = 67) the mean was 114.1 ppb (SD, 79.6). In a later study (Bertazzi *et al.*, 1987), the corresponding values were 202.8 ppb (SD, 111.7; n = 37) and 42.9 ppb (SD, 34.7; n = 37), respectively.

Wolff *et al.* (1992) studied PCB blood concentrations in capacitor workers in 1976 and 1979 in the USA. For the first sampling year, the mean concentration of the less chlorinated PCBs (di-, tri-, and tetrachlorobiphenyls) was 55 ng/mL (range, 6–2257 ng/mL), and for the latter year was 41 ng/mL (range, 6–350 ng/mL). Mean concentrations of highly chlorinated PCBs were 10 ng/mL (range, 1–308 ng/mL) in 1976, and 13 ng/mL (range, 2–350 ng/mL) in 1979. These capacitor workers (n = 60) were also surveyed by the National Institute for Occupational Health and Safety in 1977 (NIOSH, 1977), when the following blood PCB concentrations were reported as follows: less chlorinated PCBs (quantified as Aroclor 1242), 2–3300 ppb (ng/mL), and more highly chlorinated PCBs (quantified as Aroclor 1254), 5–250 ng/mL.

About 30 years later (in 2003–2006), Seegal *et al.* (2011) measured blood concentrations of individual PCB congeners in some of these former capacitor workers, and found that concentrations had dropped statistically significantly: mean concentration of less chlorinated PCBs (PCBs 28, 56, 66, 74, 99, 101) was 2.84 ng/g or 0.45 μg/g lipid in men, and 2.29 ng/g or 0.34 μg/g in women; mean concentration of highly chlorinated PCBs (PCBs 105, 118, 138, 146, 153, 156, 167, 170, 172, 174, 177, 178, 180, 183, 187, 199, 203) was 4.09 ng/g or 0.65 μg/g lipid in men, and 3.21 ng/g or 0.47 μg/g lipid in women; and total PCB concentration was 7.47 ng/g or 1.19 μg/g lipid in men, and 5.81 ng/g or 0.86 μg/g lipid in women.

Maroni *et al.* (1981a) carried out a study in two Italian electrical-capacitor manufacturing plants using PCBs as a dielectric fluid. Plant A produced electric capacitors filled with a mixture of mineral oils and PCBs. PCBs with 54% chlorination were used from 1949 to 1965, and subsequently replaced with Pyralene 3010 with 42% chlorination. The power-capacitor casings were filled with PCBs in autoclaves, and were manually removed when cooled from 70 °C to 40 °C before they were welded, tested, and finished externally. Electric "filters" (small capacitor systems used in electrical household appliances)

were impregnated with PCBs. Plant B performed short-circuit testing of high-power capacitors filled with Apirolio, a PCB mixture with 42% chlorination. Stress-testing the capacitors often included explosions. Airborne PCBs were mainly trichlorobiphenyls and concentrations ranged from 48 μg/m^3 (filter operations) to 275 μg/m^3 (power-capacitor manufacturing). Surface-wipe samples showed both tri- and pentachlorobiphenyl mixtures, with the highest amounts being found on the capacitor basket rolling carrier: trichlorobiphenyls, 127 mg; and pentachlorobiphenyls, 15 mg. Plant A employed 67 workers (40 women and 27 men): 48 were currently employed in the capacitor-manufacturing departments, 16 had been employed there for at least 6 months before the beginning of the study, and 3 had always been employed in other non-manufacturing departments without direct exposure to PCBs. PCB recovery from the palms of the hands of power-capacitor workers (plant A) showed total PCB (tri- and pentachlorinated biphenyls) skin-surface concentrations to be 4–28 μg/cm^2. Mean (± SD) blood PCB concentrations differed between current (377 ± 190 μg/kg) and past exposed workers (292 ± 161 μg/kg); workers with occasional exposure had the lowest mean total PCB exposures (110 ± 31 μg/kg). Blood PCB concentrations by job performed were highest for welders (1259 μg/kg), followed by impregnation workers (556 ± 337 μg/kg), assembly of capacitors (406 ± 173 μg/kg), and finally assembly of filters (246 ± 130 μg/kg). The blood PCB concentrations were not correlated with duration of exposure, but with the percentage ratio of hours per year spent with direct exposure to PCBs. Plant B included 13 workers (all men) exposed to PCBs during handling of the capacitors contaminated with Apirolio, dispersed from explosions sometimes caused by stress-testing. Blood PCB concentrations in currently exposed workers in plant B (200 ± 146 μg/kg) were between occasionally exposed (110 ± 31 μg/kg) and past exposed workers (292 ± 161 μg/kg) in plant A. Although the PCB mixture used in both plants had a chlorine content of 42%, the workers differed in their ratio of penta- to trichlorobiphenyls; plant A workers had higher concentrations of pentachlorobiphenyls than of trichlorobiphenyls, while the reverse was true in plant B workers. This difference was attributed to the heavy past exposure to highly chlorinated PCBs used until 1965 in plant A. Workers with abnormal liver findings (n = 16) had twice the concentrations of tri- (215 ± 95 μg/kg) and pentachlorobiphenyls (308 ± 306 μg/kg) compared with workers (n = 64) without abnormal liver findings (tri- and pentachlorobiphenyl concentrations were 92 ± 64 and 176 ± 108 μg/kg, respectively) (Maroni *et al.*, 1981b). Duration of exposure did not explain this observed difference.

One German capacitor-manufacturing worker was reported to have a blood PCB-169 concentration of 11 ng/g (Kannan *et al.*, 1994).

After a capacitor explosion at a Finnish paper mill, workers' (n = 15) blood PCB concentrations were 3.5–48.3 μg/L (Luotamo *et al.*, 1984). [These levels were much lower than during capacitor manufacturing itself.]

In a recent cross-sectional study, Persky *et al.* (2012) reported blood PCB concentrations separately for diseased (having diabetes) and non-diseased (without diabetes) workers. In diseased workers, the concentrations were: DL-PCBs, 2.5 ng/g; NDL-PCBs, 17.0 ng/g; estrogenic PCBs [PCB-52, 99, 101, 110, 153], 3.6 ng/g; anti-estrogenic PCBs [PCB-105, PCB-156], 3.6 ng/g; and PCB-74, 4.9; PCB-99, 1.0 ng/g; PCB-118, 1.4 ng/g; PCB-138, 2.5 ng/g; PCB-146, 0.4 ng/g; PCB-153, 2.8 ng/g; PCB-156, 0.6 ng/g; PCB-170, 0.7 ng/g; PCB-180, 1.1 ng/g; PCB-187, 0.3 ng/g; PCB-194, 0.2 ng/g; PCB-201, 0.2 ng/g; PCB-203, 0.2 ng/g; and PCB-206, 0.1 ng/g. In non-diseased workers, the concentrations were: DL-PCBs, 0.4 ng/g; NDL-PCBs, 4.3 ng/g; estrogenic PCBs, 1.0 ng/g; anti-estrogenic PCBs, 0.1 ng/g; PCB-74, ng/g; 0.8, PCB-99; 0.3 ng/g; PCB-118, 0.2 ng/g; PCB-138, 0.6 ng/g; PCB-146, 0.1 ng/g; PCB-153,

0.8 ng/g; PCB-156, 0.1 ng/g; PCB-170, 0.2 ng/g; PCB-180, 0.4 ng/g; PCB-187, 0.1 ng/g; PCB-194, 0.1 ng/g; PCB-201, 0.1 ng/g; PCB-203, 0.1 ng/g; and PCB-206, 0.04 ng/g.

1.5.3 Transformer manufacture and repair

Transformer manufacture was very similar to capacitor manufacture. Transformers were filled with PCBs, but the impregnation fluid was usually diluted with other chlorinated solvents (e.g. trichlorobenzene; Greenland *et al.*, 1994), and sold under different names such as Askarel (Inerteen), Pyranol, Chlophen, Apirolio, and Derol (Kerns, 1975; Lees *et al.*, 1987; Emmett *et al.*, 1988; Kalina *et al.*, 1991; Greenland *et al.*, 1994; Yassi *et al.*, 1994; Altenkirch *et al.*, 1996; Loomis *et al.*, 1997; Caironi *et al.*, 2005).

Although air concentrations from transformer manufacture were not available, two studies reported air PCB concentrations during transformer repair in two different USA plants (Lees *et al.*, 1987; Emmett *et al.*, 1988). Work activities were sampling and testing transformer fluids for dielectric properties, topping up transformers when oil levels were low, clean-up of any spills or leaks, repair of transformers by drainage of transformer oil to replace parts, and periodic filtering of the transformer oil to upgrade its dielectric properties. Ranges of air PCB concentrations for several job tasks were reported: repair and clean-up (n = 3), 43.1–60.0 μg/m^3 and TWA, 16.7–24.0 μg/m^3; clean-up of PCB leakage (n = 3), 0.1–3.1 μg/m^3 and TWA, 0.01–0.4 μg/m^3; and secondary oil leak repair and clean-up (n = 15), 2.1–17.1 and TWA, 0.7–12.4 μg/m^3 (Emmett *et al.*, 1988). Other job tasks for which concentrations were reported were draining and pumping transformer oil (n = 9), 1.1 μg/m^3; transformer repair (n = 15), 1.2 μg/m^3; network repair (n = 6), 0.5 μg/m^3; topping-up transformer oil (n = 3), 0.5 μg/m^3; explosion spill clean-up (n = 16), 1.7 μg/m^3; and filtering transformer oil (n = 6), 6.1 μg/m^3 (Lees *et al.*, 1987). Transformer-repair activities included handling transformer parts that were wet with transformer fluid without protective gloves, resulting in extensive dermal exposure. In one case, a maintenance transformer worker involved in cleaning up transformer fluid spills daily had a plasma PCB concentration of 250 μg/L (Tröster *et al.*, 1991). [This value is comparable to highly exposed capacitor-manufacturing workers.]

1.5.4 Waste incineration of PCB materials

Ten studies from seven countries (USA, Germany, Spain, Japan, the Republic of Korea, Belgium, and Poland) reported PCB exposures during waste incineration of PCB materials (Colucci *et al.*, 1973; Angerer *et al.*, 1992; Wrbitzky *et al.*, 1995; Gonzalez *et al.*, 2000; Kitamura *et al.*, 2000; Domingo *et al.*, 2001; Raemdonck *et al.*, 2006; Mari *et al.*, 2009; Park *et al.*, 2009). The PCB congeners frequently reported in this industry were PCB-28, PCB-138, PCB-153, and PCB-180. The distribution of PCB congeners in plasma depended on the type of waste material, the furnace (age and type), and the workers' activities. During burning of waste in a waste-incinerating plant, heat from combustion gases is recuperated in a cauldron to produce electricity. PCBs are, together with dioxins, produced by synthesis from organic substances and chorine during this and subsequent cooling-down processes. PCBs (with dioxins) precipitate onto particulate matter (fly ash) and are trapped in the filter (Raemdonck *et al.*, 2006).

Exposed refuse workers (n = 37) in the USA had a median plasma PCB concentration of 2.6 ppb (maximum, 14.1 ppb) (Colucci *et al.*, 1973). [No methods were reported.] Hazardous-waste workers (n = 53) in Germany had a mean plasma PCB concentration of 6.33 μg/L calculated as the sum of PCB congeners PCB-138 (1.86 μg/L) + PCB-153 (2.83 μg/L) + PCB-180 (1.65 μg/L), which was not significantly different

from controls (6.22 µg/L) in the same study (Angerer *et al.*, 1992).

Another study in Germany (Wrbitzky *et al.*, 1995) reported mean plasma PCB concentrations in waste-incineration workers (total PCBs, 3.10 µg/L; range, 1.59–6.89 µg/L) that were approximately half those in the previously described study (Angerer *et al.*, 1992). The total PCBs were the sum of the same PCB congeners as previously reported: PCB-138, 0.95 µg/L (range, 0.49–2.60 µg/L); PCB-153, 1.38 µg/L (range, 0.97–3.10 µg/L); PCB-180, 0.79 µg/L (range, 0.32–1.63 µg/L). Concentrations of PCB-28, PCB-52, and PCB-101 were below the limit of detection (< 0.2 µg/L). These workers operated the incinerator, control panels, electronics, waste gas and transfer stations, and maintained and cleaned boilers and furnaces. Workers employed in the central laboratory, incoming control and sampling, chemical-sorting station, waste-water purification, and mechanical workshop among other periphery jobs had blood PCB concentrations similar to those of workers in management. Concentrations in exposed workers were: total PCBs, 2.82 µg/L; range, 1.21–7.03 µg/L, and this was the sum of PCB-138 (0.87 µg/L; range, 0.24–2.35 µg/L), PCB-153 (1.22 µg/L; range, 0.27–2.83 µg/L), and PCB-180 (0.72 µg/L; range, 0.32–3.48 µg/L). Concentrations in workers in management were: total PCBs, 3.19 µg/L (1.59–7.53 µg/L); PCB-138, 0.98 µg/L (0.49–1.98 µg/L); PCB-153, 1.42 µg/L (0.67–3.37 µg/L); PCB-180, 0.80 µg/L (0.43–2.18 µg/L). [Of the six PCB congeners analysed, only these three were detected.]

Waste-incinerator workers in a plant in Spain were reported to have mean total PCB concentrations of 1.47 µg/L: as in the German study, this was the sum of congeners PCB-138 (0.36 µg/L) + PCB-153 (0.49 µg/L) + PCB-180 (0.57 µg/L) (Gonzalez *et al.*, 2000). Congeners PCB-28 and PCB-52 were not detected, and the concentration of PCB-101 was very low (0.02 µg/L). In another study in Spain, congener-specific concentrations were reported as means (and geometric means): PCB-28, 18.5 (12.9) µg/kg lipid; PCB-52, 10.4 (7.5) µg/kg lipid; PCB-101, 9.0 (7.1) µg/kg lipid; PCB-138, 151 (129) µg/kg lipid; PCB-153, 213 (182) µg/kg lipid; and PCB-180, 209 (158) µg/kg lipid (Domingo *et al.*, 2001). [Although the distribution of congeners differed between the two studies, the PCB concentrations could not be directly compared as the latter values were lipid-adjusted.]

Kitamura *et al.* (2000) reported blood PCB concentrations in Japanese waste workers ($n = 94$) for other PCB congeners: mean (median) PCB-77, 148.59 (149.07) pg/g lipid; PCB-126, 131.81 (98.60) pg/g lipid; and PCB-169, 104.55 (90.45) pg/g lipid. [None of these congeners were measured in the other studies.]

Workers ($n = 15$) employed as operators for incinerators, boiler-maintenance, furnace maintenance, control panel, and waste-gas washing had a mean concentration of total PCBs of 115.7 µg/kg lipid (PCB-28, 0.7 µg/kg lipid; PCB-138, 17.5 µg/kg lipid; PCB-153, 45.5 µg/kg lipid; PCB-180, 52 µg/kg lipid) (Mari *et al.*, 2009). The sum of congeners PCB-138 + PCB-153 + PCB-180 in this study resulted in a total concentration of 115 µg/kg lipid, which was five times lower than that reported in the workers in Spain (573 µg/kg lipid) (Domingo *et al.*, 2001).

In 26 waste-incineration workers from the Republic of Korea, Park *et al.* (2009) found a mean concentration of total PCBs of 214.93 ng/g lipid (median, 161.13 ng/g lipid), of which hexachloro- and heptachloro-congeners accounted for 70% (congeners measured, PCB-77, PCB-81, PCB-105, PCB-114, PCB-118, PCB-123, PCB-126, PCB-156, PCB-157, PCB-167, PCB-169, and PCB-189). [Co-exposures to dioxins, furans, and other combustible products found in fly-ash are common for waste-incineration workers.] The waste-incinerator workers did not have statistically significantly higher PCB concentrations than control subjects ($n = 7$) (mean PCB concentration, 19.13 ng/g lipid; median, 94.63 ng/g lipid).

1.5.5 Electronic-waste recycling and scrap-metal dealers

One study reported PCB exposures of workers in e-waste recycling in China (Wen *et al.*, 2008). However, they did not report air or serum PCB concentrations, but PCB concentrations in hair samples collected from 94 workers. The PCB concentration range was 55.4–7200 ng/g.

In 17 scrap-metal dealers in two plants in the USA, mean serum PCB concentrations were 7.5 ppb (range, 1–65.3 ppb) (Malkin, 1995). Serum PCB concentrations were significantly related to eating lunch outside the lunchroom [suggesting hand-to-mouth contact as a source of exposure]. The gas-chromatography peak pattern resembled that of Aroclor 1260. [Both waste recycling and scrap handling result in coexposures to dioxins and metals.]

1.5.6 Locomotive-repair workers

Locomotive-repair workers (n = 120) in the USA were found to have elevated serum concentrations of PCBs, which was attributable to exposure to transformer fluids (Pyranol, Inerteen, Aroclor) (Chase *et al.*, 1982). Workers were divided into three exposure groups: "exposed" workers who had frequent opportunity for direct contact with PCB-containing transformer fluids; "nominally exposed" workers in the facility did not have opportunity for contact with PCBs; and "non-exposed" workers whose work environment did not involve any PCB fluids. Workers' plasma PCB concentrations were: exposed workers, 33.4 ppm (10–312 ppm); nominally exposed workers, 14.2 ppm (10–30 ppm); and non-exposed workers, 12.0 ppm (10–27 ppm).

1.5.7 Miscellaneous use of PCB oil

PCBs can be emitted by several other sources, including light ballasts and microscopic immersion oil, which contains 30–45% PCBs. Fluorescent light ballasts emit PCBs during burnout (IARC, 1978) and air concentrations depend on the distance from the source. Since the previous *IARC Monograph* on PCBs (IARC, 1978), no new studies regarding PCB exposures during work with carbonless copy paper, microscopic immersion oil, or after a fluorescent light ballast burnout have been published.

One study reported a PCB air measurement from a carbonless copy paper stockroom of 0.07 mg/m^3 (Tatsukawa, 1976). Hasegawa *et al.* (1973) reported that blood PCB concentrations in workers in carbonless paper producing plants (0.01–0.02 µg/g) 2 years after exposure were 10% those found during the period when the PCBs were used. No air or biological monitoring data have been published to assess the extent of PCB exposures during the use of microscope oil (Bennett & Albro, 1973). Four and a half hours after burn-out of a ballast, the concentration of PCBs was the highest (166 µg/m^3) 1 m below the burned-out ballast, while the lowest concentration (12 µg/m^3) was found at a distance of 4.5 m from the fixture (Staiff *et al.*, 1974).

In 1958–1978 in Canada, areas around transformers mounted outdoors were treated with phenoxy herbicides (2,4-D and 2,4,5-D) to reduce foliage (Hay & Tarrel, 1997). To increase adherence of the herbicides to the plant leaves, herbicide sprayers (n = 225) would mix 4 pounds [1.8 kg] of phenoxy herbicide with 10 gallons [37.9 L] of used transformer fluid and 90 gallons [340.7 L] of water before spraying. PCB exposures were not measured during this operation.

Use of Aroclor 1254 was reported in a petrochemical plant in the USA during the 1950s, where 31 men had been "heavily exposed" (Bahn *et al.*, 1976). No information regarding how PCB was used was given [but could have been PCBs used as fluids for hydraulic and heat-transfer systems]. No air or blood concentrations of PCBs were reported.

United States navy vessels built between 1946 and 1977 commonly contained PCBs in insulation material, electrical cable, and ventilation

gaskets (Still *et al.*, 2003). In nuclear submarines, PCBs were also used in soundproofing material, missile-launch tubes, electrical cables, banding and sheet rubber, heat-resistant paints, hull coatings, and electrical transformers. Activities associated with PCB exposure during dismantling of these vessels were transformer clean-up and removal; cutting/crushing of PCB-contaminated steel, steel-shot blasting of PCB-contaminated surfaces, chiselling/hand-chipping of PCB-contaminated surfaces, and shoveling/sweeping of PCB-contaminated debris. Surface-wipe sampling showed PCB amounts ranging from non-detects to 11 000 µg/100 cm^2. [Information for PCB exposure in the military is scarce.]

Cumulative lifetime exposure to PCBs among Mohawk men at Akwesasne (a Native American community of more than 10 000 persons located along the St Lawrence River in New York, Ontario, and Quebec) who had been occupationally exposed to PCBs was positively associated with serum total PCB concentration ($P = 0.03$) (other non-occupational sources such as fish consumption and living close to hazardous waste sites discussed in the article are not referenced here). The congener profile was most similar to that of Aroclor 1248, the commercial mixture used at local industrial facilities (Fitzgerald *et al.*, 2007) as a hydraulic fluid in a foundry's die-casting machines from 1959 to 1974, and as a component in aluminium-processing heat-transfer equipment. The occupational exposure of Mohawk men was independently assessed by two occupational hygienists as the probability of exposure to PCBs for all jobs of more than 6 months duration with the following qualitative ratings: (1) definitely not exposed; (2) possibly exposed; (3) probably exposed; and (4) definitely exposed. These ratings were assigned weights of zero, 0.25, 0.5, and 1.0, respectively. The weights for each job were then multiplied by duration of employment in that job, and the results were summed over all jobs to estimate cumulative lifetime occupational exposure to PCBs for each man. [The Working Group noted that this population was also exposed environmentally (see Section 1.4.1(a)).]

A qualitative PCB exposure assessment among welders in Sri Lanka was performed recently (Lankatilake *et al.*, 2012). PCB oil extracted from discarded transformers was widely used as coolant oil in small-scale welding facilities in Sri Lanka to facilitate heat transmission and thereby assist in the cooling process. Exposure to coolant oil occurs during replacement of the coolant and while repairing machinery. The amount of coolant oil used in a welding machine depends on the type of machine, but on average is about 5 L. During repairs, there is a high risk of exposure to PCBs in the transformer oil.

1.5.8 Occupations with exposure to PCB by-products

PCBs have also been reported as a by-product in an electric arc furnace steelmaking plant in the United Kingdom (Aries *et al.*, 2008). Air PCB concentrations in decreasing order by department were: melting shop, 586 pg/m^3 (range, 144–1313 pg/m^3); casting area, 187 pg/m^3 (range, 73–272 pg/m^3); control cabin, 99 pg/m^3 (range, 57–129 pg/m^3). The most prominent congeners were PCB-118 (100–500 pg/m^3), PCB-105 (10–80 pg/m^3), and PCB-77 (5–35 pg/m^3).

Using static high-volume samplers (0.2 m^3/min for 12 hours or 24 hours) in a basic oxygen steelmaking (BOS) and iron ore sintering plant, Jackson *et al.* (2012) calculated mean TEQ pg/m^3 for the by-products PCDD/F and PCBs. The BOS process involves the transfer, desulfurization, and refining of hot metal in a steel converter, and secondary steelmaking treatments. Sintering is a process for blending and fusing iron-ore fines, fluxes, coke, and recycled materials (grit and dusts from other processes). Air concentration ranges were: sinter plant, 0.19–3.72 TEQ pg/m^3 ($n = 12$); and BOS plant, 0.08–0.71 TEQ pg/m^3 ($n = 24$). In

all instances, concentrations of PCBs were much higher than of PCDD/Fs. PCB-126 contributed significantly to the total TEQ (5–20%).

PCBs have been reported as a by-product in penta- and trichlorophenol wood-preservation pesticide manufacturing with PCDFs and PCDDs (Hryhorczuk *et al.*, 1998; Collins *et al.*, 2008). Serum PCB concentrations (sum of PCB-77, PCB-81, PCB-126, and PCB-169) were measured by the company in these workers (Collins *et al.*, 2008): for pentachlorophenol workers only (n = 26; period exposed, 1944–1980) 73.6 pg/g lipid; trichlorophenol workers only (n = 12; period exposed, 1954–1979): 75.9 pg/g lipid; pentachlorophenol and trichlorophenol workers (n = 14; period exposed, 1961–1980): 86.3 pg/g lipid; tradesmen (n = 10): 121.1 pg/g lipid. These PCB concentrations were not much different from those of a selected reference population (n = 36; 75.0 pg/g lipid).

1.5.9 Removal of PCB-containing sealants

PCB-containing sealants were used in building construction before PCBs were banned in that country. For example, sealant used in Sweden contained 4.7–8.1% Clophen A40 (Sundahl *et al.*, 1999). Air PCB concentrations of 10–120 µg/m^3 were reported after removal of the sealant by a variety of methods: cutting the elastic sealant with an oscillating knife; grinding the concrete with a mechanical machine; sawing the concrete with a mechanical saw; or cutting the concrete with a mechanical chisel. The removal methods were changed by equipping the tool with suction, which reduced air PCB concentrations to non-detects to 3.1 µg/m^3 (Kontsas *et al.*, 2004). Serum PCB concentrations in sealant-remover workers were 0.6–17.8 µg/L (mean, 3.9 µg/L; and median, 1.9 µg/L). For highly chlorinated PCBs, the mean was 3.5 µg/L (median, 1.6 µg/L), and for less chlorinated PCBs, the mean was 0.4 µg/L (median, 0.2 µg/L). Correlation between concentrations in air and serum was only noted for PCB-28 and PCB-52.

During sealant removal in Finland, total PCB concentration in dust samples was 0.026 mg/m^3 (Priha *et al.*, 2005). Congeners determined in the sealant were: PCB-28, 82 mg/kg; PCB-52, 3030 mg/kg; PCB-77, 37 mg/kg; PCB-101, 10 325 mg/kg; PCB-118, 6145 mg/kg; PCB-126, 42 mg/kg; PCB-138, 11 765 mg/kg; PCB-153, 11 185 mg/kg; PCB-169, 32 mg/kg; and PCB-180, 7254 mg/kg (Priha *et al.*, 2005).

Swedish construction workers removing PCB-containing sealants had serum PCB concentrations (sum of 19 congeners) of 575 mg/g lipid, while controls (construction workers not involved in PCB abatement work) had levels of 267 mg/g lipid (Seldén *et al.*, 2008; Wingfors *et al.*, 2006). Concentrations of PCB-180 were not significantly different between groups, while concentrations of many less chlorinated PCBs (especially PCB-66 and PCB-56/PCB-60, but also PCB-28, PCB-44, PCB-52, PCB-74, PCB-101, and PCB-105) were much higher in the exposed workers than in the controls.

1.5.10 People working in contaminated buildings

People working in contaminated buildings (office workers, teachers) are exposed to PCBs (Wiesner *et al.*, 2000); PCB concentrations have been surveyed in workers' air (Gabrio *et al.*, 2000; Schwenk *et al.*, 2002; Peper *et al.*, 2005; Schettgen *et al.*, 2012) and blood (Gabrio *et al.*, 2000; Schwenk *et al.*, 2002; Peper *et al.*, 2005; Herrick *et al.*, 2011; Schettgen *et al.*, 2012).

Mean indoor air concentrations of PCBs in three contaminated schools in Germany were reported to be between 77 and 10 125 ng/m^3; 90% of the total PCBs were either PCB-28 or PCB-52 (Gabrio *et al.*, 2000). These congeners were also reported to be found at high concentrations (> 4000 ng/m^3) in other studies in Germany (Schwenk *et al.*, 2002; Peper *et al.*, 2005; Schettgen *et al.*, 2012). The teachers (n = 96) working in the three contaminated buildings had mean

blood PCB-28 concentrations that differed by school (0.045 µg/L, 0.057 µg/L, and 0.098 µg/L, respectively), and that were significantly elevated compared with teachers (n = 55) not working in contaminated schools (range, not detected to 0.035 µg/L) (Gabrio *et al.*, 2000).

Median indoor air concentrations were measured over 2 years in schools in Germany for congeners PCB-28 (33 ng/m³), PCB-52 (293 ng/m³), and PCB-101 (66 ng/m³) (Liebl *et al.*, 2004). Concentrations of more highly chlorinated indicator congeners (PCB-153, PCB-138, and PCB-180) were all below 80 ng/m³. The median sum of indicator congeners was 2.04 µg/m³. Biomonitoring of teachers (n = 9) and cleaning personnel (n = 1) in schools in Germany showed that median blood PCB concentrations exceeded the German reference values after adjusting for age in 8 out of 10 workers for PCB-138, 7 out of 10 for PCB-153, and 8 out of 10 for PCB-180 (Neisel *et al.*, 1999).

In teachers in the USA, the relative contribution of lighter congeners (PCBs 6–74) (mean total serum PCB concentration, 1.86 ng/g; n = 18) was higher than in controls (Herrick *et al.*, 2011). This was also observed in other studies: mean concentration of PCB-28, 0.28 µg/L; PCB-101, 0.07 µg/L; PCB-138, 1.29 µg/L; PCB-153, 1.68 µg/L; and PCB-180, 1.14 µg/L in Peper *et al.*, (2005); median concentration of PCB-28, 0.087 µg/L; PCB-52, 0.024 µg/L; and PCB-101, 0.012 µg/L in Schettgen *et al.*, (2012); and mean concentration of PCB-28, 0.24 µg/L; PCB-52, 0.07 µg/L; PCB-101, 0.02 µg/L; PCB-153, 0.96 µg/L; PCB-138, 0.70 µg/L; and PCB-180, 0.62 µg/L in Schwenk *et al.*, (2002).

People working inside contaminated buildings other than schools may also be exposed to PCBs. In Germany, air PCB concentrations in contaminated commercial buildings were 1280 ng/m³ (PCB-28, 110 ng/m³; PCB-52, 125 ng/m³; PCB-101, 11 ng/m³; PCB-138, < 2 ng/m³; PCB-153, < 2 ng/m³; PCB-180, < 2 ng/m³) (Broding *et al.*, 2007). The PCB contamination originated from insulation material and elastic sealing compounds. Serum PCB concentrations were determined in 2002 for 583 persons who had worked between 1 and 40 years in the contaminated commercial building. The median serum total PCB concentration was 2.32 µg/L (PCB-28, 0.09 µg/L; PCB-52, 0.01 µg/L; PCB-138, 0.55 µg/L; PCB-153, 0.9 µg/L; and PCB-180, 0.7 µg/L). People not working in the contaminated building (n = 205) had significantly lower serum concentrations of PCB-28 and PCB-52 (0.023 µg/L and 0.004 µg/L, respectively) (Broding *et al.*, 2008).

1.5.11 Clean-up of hazardous waste

Occupational exposure to PCBs has also been measured in workers who perform clean-up of hazardous waste. After an explosion and fire of unlabelled chemical waste drums at the former site of Chemical Control Corporation in Elizabeth, New Jersey, USA, the mean air PCB concentration was 0.11 µg/m³ (n = 3) (Costello & King, 1982). In workers (n = 32) removing hazardous waste, including transformers, in the USA, plasma PCB mean concentration was 205 ng/g lipid (range, limit of detection to 527 ng/g lipid) (Horii *et al.*, 2010). Hexa and heptachlorinated biphenyls accounted for 60% of the PCB concentrations.

1.5.12 Firefighters and rescue workers

Firefighters and rescue workers have also been surveyed for PCB exposure in several recent studies, demonstrating a wide variability in serum PCB concentrations (Table 1.33; Kelly *et al.*, 2002; Schecter *et al.*, 2002; Dahlgren *et al.*, 2007; Chernyak *et al.*, 2009, 2012).

1.6 Exposure assessment of epidemiological studies

1.6.1 Studies of occupational exposure

Many epidemiological studies of occupational PCB exposure and cancer have been performed; the majority are among workers in capacitor-manufacture and transformer manufacture and repair. Duration of employment was used to assess exposure in most of these studies (Brown & Jones, 1981; Bertazzi *et al.*, 1982, 1987; Cammarano *et al.*, 1984; Brown, 1987; Nicholson & Selikoff, 1987; De Guire *et al.*, 1988; Taylor *et al.*, 1988; Liss, 1989; Petruska & Engelhard, 1991; Greenland *et al.*, 1994; Tynes *et al.*, 1994; Yassi *et al.*, 1994, 2003; Gustavsson *et al.*, 1986; Savitz & Loomis, 1995; Tironi *et al.*, 1996; Gustavsson & Hogstedt, 1997; Hay & Tarrel, 1997; Kimbrough *et al.*, 1999, 2003; Loomis *et al.*, 1997; Charles *et al.*, 2003; Mallin *et al.*, 2004; Caironi *et al.*, 2005; Prince *et al.*, 2006a, b; Ruder *et al.*, 2006; Ahrens *et al.*, 2007; Hopf *et al.*, 2009b, 2010, 2014; Silver *et al.*, 2009; Pesatori *et al.*, 2013). In the remaining studies, exposure to PCBs was assessed using a variety of approaches, including job-exposure matrices (JEM), development of worker's exposure zones, and measurement of serum PCB concentrations.

JEMs were used in several studies (Greenland *et al.*, 1994; Loomis *et al.*, 1997; Prince *et al.*, 2006a, b; Ruder *et al.*, 2006; Silver *et al.*, 2009).

Greenland *et al.* (1994) developed a JEM in a case–control study of cancer mortality at a transformer-assembly facility. Pyranol was used as the transformer oil from 1936 to 1976. Pyranol was composed of 50% PCBs (mainly hexachlorobiphenyls) and 50% trichlorobenzene, but the PCB content could vary from 45% to 80%. A combination of 1000 job titles in 50 departments in 100 buildings resulted in more than 5500 entries in the JEM. Each entry was rated for seven selected exposures from 1901 to 1984. A four-point categorical rating scale was used to rate the jobs. Former employees and experienced industrial hygienists rated each entry. For pyranol, benzene, and solvents, the analysis categories were: 0, no exposure; 1, indirect exposure, meaning that the chemical was found in the work area, but the worker did not perform tasks using it; 2, direct exposure. Cumulative exposures were calculated using these scores and individual job histories.

A cancer mortality study among electric-utility workers in five companies exposed to PCBs used job categories to estimate weekly exposures in hours for each job (Loomis *et al.*, 1997). PCBs were used in capacitors, transformers and switches. Capacitor fluids were 100% PCBs, while transformer fluids contained 70% PCBs and 30% chlorinated benzene solvents. Exposure assessments were performed by expert panels for each company. The panel members (industrial hygienists, safety personnel, managers, and long-term workers) recorded their individual exposure assessments for PCBs, and other exposures, which were later discussed to resolve differences. For each occupational category and decade, the frequency in times per week and duration in hours of exposure to insulating fluids during the average working week was indicated. This was used to construct company and calendar time-specific JEMs Industrial hygiene surveys of the plants were used to interpret the panel's exposure assessment. Each occupational category (in total, 28) was classified according to workers' potential exposure to PCBs.

Three plant-specific semiquantitative JEMs were used in a study of cancer of the breast in former capacitor-manufacturing workers (women) in Indiana, Massachusetts, and New York, USA (Silver *et al.*, 2009). Two of these JEMs had been used previously in a mortality study (Prince *et al.*, 2006a, b) of former workers at the Indiana and Massachusetts plants, and one in a mortality study of former workers at the Indiana plant (Ruder *et al.*, 2006). Two of the three JEMs have been described in detail in separate publications (Hopf *et al.*, 2009b,

Table 1.33 Serum PCB concentrations in firefighters

Country	Population	Activity	PCB congeners measured	Mean serum PCB concentration	Reference
USA	Firefighters (n = 58)	Extinguishing a transformer fire	NA	2.96 ppb (range, 1.9–9.6 ppb)	Kelly *et al.* (2002)
	Rescue workers (n = 7)	Working during the collapse of the World Trade Center, New York, September 2001	Non-*ortho* PCBs	43–328 pg/g lipid	Dahlgren *et al.* (2007)
			Mono-*ortho* PCBs	19–404 ng/g lipid	
			ΣDL-PCBs[a]	19–405 ng/g lipid	
Russian Federation	Firefighters, symptomatic (n = 8)	Participated in extinguishing a fire at a cable-manufacturing plant (no SCBA)	ΣDL-PCBs[a]	198.6 pg/g lipid	Chernyak *et al.* (2009, 2012), Schecter *et al.* (2002)
	Firefighters, asymptomatic (n = 5)	Participated in extinguishing a fire at a cable-manufacturing plant (no SCBA)	ΣDL-PCBs[a]	198.9 pg/g lipid	
	Firefighters (n = 7)	Other fires	ΣDL-PCBs[a]	231.7 pg/g lipid	
			Congener 77, 126, 169	Symptomatic firefighters > than the other groups	Schecter *et al.* (2002)

[a] DL-PCBs, dioxin-like PCBs, i.e. PCBs 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189

NA, not available; PCB, polychlorinated biphenyl; SCBA, self-contained breathing apparatus

2010). Exposure determinants or factors that influenced PCB exposures for each plant were assessed for all jobs listed in the work histories. Jobs with similar rating of the exposure determinants were grouped into exposure categories. Each job-exposure category, exposure intensity (high, medium, low, background) and frequency (continuous, intermittent) were qualitatively rated separately for inhalation and dermal exposure. The plant-specific JEMs used available air PCB concentrations (the same as in Sinks *et al.* (1992) for the Bloomington plant) to assign inhalation weightings. The product of intensity and frequency (fraction of day exposed) was calculated for each job-exposure category. Finally, the JEM was modified for eras with different conditions of PCB exposure (change in Aroclor use, ventilation-system improvements, lay-out changes etc).

[These historical reconstructions are better than using duration of employment alone in the epidemiological studies, since duration does not distinguish between jobs with higher or lower potential for PCB exposure. Most of these retrospective studies involved manufacturing plants that used limited amounts of other chemicals, or at least when other chemicals were used, these jobs were often indicated and could be excluded from the epidemiological analysis. Creating cohorts of today's working environment would include a very diverse industry with multitude of job activities, including an array of different chemicals. Therefore it would be difficult to draw definitive statements on the causations of a possible observed mortality excesses.]

In their retrospective study of mortality, Sinks *et al.* (1992) developed workplace exposure zones to classify worker exposure. The capacitor-manufacturing plant studied was divided into five zones of exposure by drawing consecutive circles (radius, approximately 69 m) centred upon the heaviest source of PCB exposure. The production area was thus divided into three zones by proximity to PCB source. Two other zones were

defined: maintenance and office workers. Air sampling was conducted in these five zones, and means were assigned as the weight (1–5) of PCB exposure for the zone.

Serum PCB concentrations were used in one case–control study (Laden *et al.*, 2001b), and in a recent cross-sectional study (Persky *et al.*, 2012) (see Section 1.5.2).

1.6.2 Studies of environmental exposure

Cohort studies of environmental exposure have used many approaches to assess exposure to PCBs. Exposure approaches include interview, questionnaires, cumulative PCB exposure, dietary intake of fatty fish, PCB concentrations in biological media such as blood, adipose tissue, and breast milk, and in the environment such as carpet dust, or any combinations of these. Biological measures of body burden have been used extensively (see Table 1.34).

1.7 Regulations and guidelines

1.7.1 Global

For Parties to the Stockholm Convention on Persistent Organic Pollutants (POPs) (UNEP, 2001), presently 179 Member States, the production of PCBs is totally prohibited, although the presence of PCBs in equipment is allowed to continue until 2025. The environmentally sound management of waste containing or contaminated with PCBs at a content above 0.005% must be achieved by 2028.

Annex I of the Basel Convention on the Transboundary Movements of Hazardous Wastes and Their Disposal (UNEP, 2011) defines a category of hazardous waste specific to PCBs: "Y10 waste substances and articles containing or contaminated with PCBs and/or polychlorinated terphenyls (PCTs) and/or polybrominated biphenyls (PBBs)." Additionally, Annex VIII defines as "hazardous" any electrical waste containing or contaminated with PCBs at a concentration greater than 50 mg/kg. The Basel Convention is legally binding for 179 countries (status in 2013).

The Codex Alimentarius Commission, recognizing the importance of prevention of human exposure through source-directed measures (i.e. strict control of industrial and agricultural processes that may generate and release PCDDs, PCDFs, and PCBs), adopted the Code of Practice Concerning Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49–2001) (Codex Alimentarius, 2001) and the Code of Practice for the Prevention and Reduction of Dioxin and Dioxin-like PCB Contamination in Foods and Feeds (CAC/RCP 62–2006) (Codex Alimentarius, 2006). No limits in foodstuffs were included, but management options were recommended.

(a) Provisional tolerable monthly intake

In 2002, the Joint Food and Agriculture Organization of the United Nations (FAO)/WHO Expert Committee on Food Additives (JECFA) established a provisional tolerable intake of 70 pg/kg bw per month for PCDDs, PCDFs, and DL-PCBs expressed as TEFs, based on reproductive end-points (JEFCA, 2002). The value was expressed "per month" to reflect that exposure is cumulative and chronic rather than acute.

(b) Drinking-water

No water quality guidelines have been set for these substances because of their low solubility in water.

(c) Air

Air quality guidelines for PCBs have not been established, because exposure by direct inhalation generally constitutes only a small proportion of total exposure, in the order of 1–2% of the daily intake from food. Although this air concentration is only a minor contributor to direct human exposure, it is a major contributor to contamination of the food-chain (WHO, 2000).

Table 1.34 Common measures of exposure to PCBs and design of the exposure assessment in epidemiological studies in non-occupational settings

Exposure measure	Exposure assessment	Examples of exposure categories reported
Cumulative PCB exposure	Regular jobs held	• Job-exposure schemes • Industry classifications • Potential exposure to PCBs as assessed by an occupational hygienist
Dietary intake of fatty fish containing PCBs	Standardized questionnaires or interviews	• Number of fish meals per day
High-level dietary intake of contaminated rice oil (mass poisoning)	Admission to hospital	• Area of residence
Environmental PCB concentrations	PCB concentrations in carpet dust PCBs in soil	• Amount of PCBs in dust • Amount of PCBs in soil
PCB concentrations in biological samples	Serum PCB concentration, non-lipid adjusted	• Sum of PCB congeners • High or low PCB body burden: 'high' exposure (higher than the median based on the control group) vs. 'low' exposure (lower than the median based on the control group)
	Serum PCB concentration, lipid adjusted	• Sum of PCB congeners measured • Single PCB congeners • Potentially estrogenic PCBs (PCB-44, PCB-54) and PCB-101, PCB-187 • Potentially anti-estrogenic, immunologic, dioxin-like, non-*ortho* substitution, mono-*ortho* substitution, moderately persistent (PCB-66, PCB-77, PCB-105, PCB-118, PCB-126) • Immunotoxic PCBs (PCB-66, PCB-74, PCB-105, PCB-118, PCB-138, PCB-153, PCB-156, PCB-167, PCB-180) • Di-*ortho* substitution, limited DL-PCBs and persistent PCBs (PCB-128, PCB-138, PCB-170) • Biologically persistent inducers of CYP1A and CYP2B • Environmentally relevant PCBs (PCB-195, PCB-206, PCB-209) • Neurotoxic PCBs (PCB-18, PCB-28) • Non-dioxin-like PCBs (PCB-74, PCB-99, PCB-118, PCBs 138–158, PCB-146) • Sum of DL-PCBs (PCB-105, PCB-118, PCB-156) • Sum of NDL-PCBs (PCB-28, PCB-99, PCB-138, PCB-153, PCB-170, PCB-183, PCB-187) • *BRCA1* inhibiting PCBs (PCB-101, PCB-138) • Pseudo-estrogen PCBs (PCB-28, PCB-52, PCB-153) • Phenobarbital inducers (PCB-101, PCB-153, PCB-180, PCB-194) • Most-represented congeners (PCB-118, PCB-138, PCB-153, PCB-180)
	Plasma PCB concentration	Sum of the four most prevalent PCB congeners (PCB-118, PCB-153, PCB-138, PCB-180)
	Adipose tissue PCB concentrations	Sum of 18 PCBs Sum of dioxin-like PCBs (PCB-77, PCB-126, PCB-169)
PCB concentrations in biological samples (cont.)	Tumour tissue PCB concentrations	Sum of PCB congeners (PCB-28, PCB-31, PCB-49, PCB-52, PCB-101, PCB-105, PCB-118, PCB-138, PCB-153, PCB-170, PCB-180), measured at the time of diagnosis

DL-PCB, dioxin-like polychlorinated biphenyl; NDL-PCB, non-dioxin-like polychlorinated biphenyl

1.7.2 Environmental regulations

(a) European Union and Member States

The Member States of the European Union have taken actions to eliminate the production, use, and release of PCBs since 1985. In 2004, to implement the Stockholm Convention on POPs, by regulation EC/850/2004 (EU 850/2004), the production, placing on the market, and use of PCBs were prohibited. Low POPs concentration limits were adopted through Council Regulation (EC) No. 1195/2006 (EU 1195/2006) amending Annex IV to Regulation (EC) No 850/2004. Within the European Union of 26 Member States, several measures have been adopted to reduce the presence of PCDDs, PCDDs, PCDFs, and PCBs in the environment, in food and in feed. These include:

- Commission Regulation (EC) No. 1883/2006 of 19 December 2006 laid down methods of sampling and analysis for the official control of levels of dioxins and DL-PCBs in certain foodstuffs;
- Commission Recommendation 2006/88/EC of 6 February 2006 concerning the reduction of the presence of dioxins, furans and PCBs in feedingstuffs and foodstuffs;
- Commission Recommendation 2006/794/EC of 16 November 2006 on the monitoring of background levels of dioxins, DL-PCBs and NDL-PCBs in foodstuffs.
- The most recent Commission Regulation (EU) No. 1259/2011 amended Regulation EU 1881/2006 as regards maximum levels for DL-PCBs and NDL-PCBs (EC, 2011a); it also changed the formerly used 1998 WHO TEFs to the scheme adopted in 2005 (referred to as WHO_{2005}-TEFs) (Van den Berg *et al.*, 2006) and includes maximum levels for NDL-PCBs in food.

  See Table 1.35

(b) North America

(i) USA

The United States Food and Drug Administration has established tolerance levels in various foods in an attempt to reduce human exposure to PCBs (FDA, 2013). [These limit values were set in 1971 and 1977, before any epidemiological and most experimental studies were conducted, and have not been revised since.] The temporary tolerance levels for PCB residues are as follows:

- 1.5 ppm in milk (fat basis);
- 1.5 ppm in manufactured dairy products (fat basis);
- 3 ppm in poultry (fat basis);
- 0.3 ppm in eggs;
- 0.2 ppm in finished animal feed for food-producing animals (except the following finished animal feeds: feed concentrates, feed supplements, and feed premixes);
- 2 ppm in animal feed components of animal origin, including fishmeal and other by-products of marine origin and in finished animal feed concentrates, supplements, and premixes intended for food-producing animals.
- 2 ppm in fish and shellfish (edible portion). The edible portion of fish excludes head, scales, viscera, and inedible bones;
- 0.2 ppm in infant and junior foods;
- 10 ppm in paper food-packaging material intended for or used with human food, finished animal feed and any components intended for animal feeds. The tolerance does not apply to paper food-packaging material separated from the food therein by a functional barrier that is impermeable to migration of PCB.

The United States Environmental Protection Agency (EPA) has set a maximum contaminant level for PCBs of 0.0005 mg/L (500 ppt) in drinking-water. The EPA requires that spills

Table 1.35 Maximum permitted levels for dioxin-like compounds and indicator PCBs in the European Food and Feed regulation

Foodstuffs	Maximum permitted levels[a]		
	Sum of PCDDs, PCDFs, DL-PCBs[b] (pg WHO_{2005}-TEQ per g fat)	DL-PCBs[b] (pg WHO_{2005}-TEQ per g fat)	Sum of PCB_6[c] (ng/g fat)
Meat and meat products (excluding edible offal) of the following animals):			
Bovine animals and sheep	4.0	1.75	40
Poultry	3.0	0.75	40
Pigs	1.25	0.5	40
Liver of terrestrial animals and derived products thereof	10.0		40
Muscle meat of fish and fishery products and products thereof (with the exemption of wild caught eel and wild-caught fresh water fish, with the exception of *diadromous* fish species caught in fresh water, fish liver and derived products, and marine oils)[a]	6.5 pg/g ww	2.5 pg/g ww	75 ng/g ww
Muscle meat of wild caught fresh water fish, with the exception of *diadromous* fish species caught in fresh water, and products thereof[d]	6.5 pg/g ww		125 ng/g ww
Muscle meat of wild caught eel (*Anguilla anguilla*) and products thereof	10.0 pg/g ww		300 ng/g ww
Fish liver and derived products thereof with the exception of marine oils referred to above	20.0 pg/g ww		200 ng/g ww
Marine oils (fish body oil, fish liver oil and oils of other marine organisms intended for human consumption)	6.0		200
Raw milk and dairy products, including butter fat	5.5	2.0	40
Hen eggs and egg products	5.0	1.75	40
Fat of the following animals:			
Bovine animals and sheep	4.0		40
Poultry	3.0		40
Pigs	1.25		40
Mixed animal fats	2.5	0.75	40
Vegetable oils and fats	1.25		40
Foods for infants and young children	0.2 pg/g ww		1.0 ng/g ww
Fruits, vegetables and cereals		0.1 pg/g ww	

[a] The maximum level expressed on fat is not applicable for foods containing < 2% fat (the maximum level expressed on product basis for foods containing < 2% fat = maximum level expressed on fat for that food × 0.02).

[b] The Commission Recommendation 2011/516/EU (EC, 2011b) replaces regulation 2006/88/EC and sets separate action levels for PCDD/PCDF (expressed as WHO_{2005}-TEQ) and DL-PCB (expressed as WHO_{2005}-TEQ).

[c] PCB_6 comprises PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, and PCB-180

[d] The maximum level for crustaceans applies to muscle meat from appendages and abdomen. In the case of crabs and crab-like crustaceans (*Brachyura* and *Anomura*) it applies to muscle meat from appendages.

DL-PCB, dioxin-like polychlorinated biphenyl; PCDD, polychlorinated dibenzodioxins; PCDF, polychlorinated dibenzofurans; TEQ, toxic equivalent; ww, wet weight

Adapted from EC (2011a) and EC (2011b)

or accidental releases into the environment of 1 pound (0.45 kg) or more of PCBs be reported to the EPA (ATSDR, 1996).

(ii) Canada

The import, manufacture, and sale (for re-use) of PCBs were made illegal in Canada in 1977. Release of PCBs to the environment was made illegal in 1985. However, use of PCB-containing equipment is allowed until the end of its service life. The storage of PCBs has been regulated since 1988. Export has been regulated since 1997. These provisions are maintained in the Chlorobiphenyls Regulations, under the Canadian Environmental Protection Act, 1999 (CEPA, 2011).

The regulation of waste is consistent with the Basel Convention's "Technical guidelines for the environmentally sound management of wastes consisting of, containing, or contaminated with persistent organic pollutants" (Basel Convention, 2007, 2015).

(c) Australia and New Zealand

(i) Australia

The Industrial Chemicals (Notification and Assessment) Act 1989 was amended to give effect to the Stockholm Convention (NICNAS, 1989).

The National Strategy for The Management of Scheduled Waste was endorsed by the Australian and New Zealand Environment and Conservation Council in 2003 (ANZECC, 2003) and provides for the safe management and disposal of organochlorine pesticides, PCBs and hexachlorobenzene. The PCB Management Plan provides treatment provisions for different types of PCB waste including liquid residues and discharges, gaseous emissions, solid residues and disposal (Australian Government, 2006, 2007).

(ii) New Zealand

The Hazardous Substances and New Organisms (HSNO) Act 1996 (as amended by the HSNO [Stockholm Convention] Act Amendment 2003), prohibits the production, use and import of the chemicals listed in Annex A of the Convention, including PCBs. Exempted use of PCBs as per the Toxic Substances Regulations 1983 is permitted, but subject to phase-out no later than December 2016. The HSNO Act 1996 is administered by The New Zealand Environment Risk Management Authority (ERMA) by: assessing new chemicals, pesticides or industrial chemicals currently in use that exhibit POP characteristics (Articles 3.3 and 3.4); permitting the appropriate use of POPs for laboratory-scale research or as a reference standard (Article 3.5); managing the existing exempted use and storage of PCBs (Article 3.6); prohibiting import, manufacture, or use of POPs (Article 3.1 and 3.2). The Imports and Exports (Restrictions) Act 1988, via the Imports and Exports (Restrictions) Prohibition Order (No. 2) 2004, prohibits export of POPs (except as conditionally provided under Article 3.2). Import and export are regulated under The Imports and Exports (Restrictions) Act 1988.

(d) Asia

(i) China

China implements an import and export registration system, included under its Regulations on Environmental Management of Chemicals and the Import and Export of Toxic Chemicals of 1994. In 2005, PCBs were included in the List of Toxic Chemicals Strictly Prohibited from Import and Export, by No. 116 Notice on the List of Goods Prohibited from Import (the Sixth Group). The National Implementation Plan under the Stockholm Convention entered into force for China in 2004, and also applied to the Special Administrative Regions of Hong Kong and Macao (NIP China, 2007). This plan aims to prohibit and prevent the production and import of PCBs, and to achieve the environmentally sound management of currently used equipment containing PCBs. China used to produce PCBs, but production was stopped in the 1970s. The

plan called for establishing a system for the declaration, registration, and environmentally sound management of equipment in use containing PCBs by 2010. Identification of high-risk equipment currently in use across the country is to be achieved by 2015, with uses of PCBs eliminated by 2025.

Furthermore, China has also stipulated special administrative regulations and standards with regard to PCBs. The Notice on the Issues Concerning Prevention of Pollution Caused by Hazardous Polychlorinated Biphenyls was promulgated in 1979 to ban future imports of power equipment containing PCBs. The Notice on Enhancement of the Management over Waste Polychlorinated Biphenyl Power Capacitors was issued in 1990 to forbid trading and dismantling downstream capacitors containing PCBs. The Provisions on the Pollution Caused by Power Installations Containing Polychlorinated Biphenyls and Related Wastes of 1991 addresses the declaration, transfer, transport, import, treatment, disposal, sealing-up and storage of PCB wastes and other sources. The Control Standard on Polychlorinated Biphenyls for Wastes (GB13015–91) was implemented in 1991, in which the value of the control standard on PCBs wastes and the treatment methods for wastes containing PCBs are stipulated (NIP China, 2007).

(ii) Taiwan, China

Importation of PCBs was prohibited in 1980. The Environmental Protection Administration of Taiwan, China, banned the manufacture, sale, and use of PCBs in 1988. An extensive investigation of electrical devices in 1990–1991 indicated that more than 80 000 PCB-containing electrical devices were still in use, mainly capacitors and transformers. A full-scale ban on the use of PCBs, with the exception of experimental, research, and educational purposes, took effect in January 2001. This prohibited use of any electrical devices containing PCBs by the end of 2000, mandating immediate disposal at end of use of capacitors and transformers containing PCBs (Environmental Protection Administration, 1988). Furthermore, PCBs may not be detectable in effluents from business, sewage systems and building sewage-treatment facilities.

(iii) India

According to Schedule VI of the Hazardous Waste (Management, Handling and Transboundary Movement) Rules 2008, the import and export of hazardous wastes, substances and articles containing or consisting of or contaminated with PCBs are prohibited (Ministry of Environment and Forests, 2008).

(e) Africa

United Republic of Tanzania

The Industrial and Consumer Chemicals (Management and Control) Act of 2003 provides for the management and control of PCBs under the list of severely restricted/banned/eliminated chemicals in Schedule 8. The government of the United Republic of Tanzania issued an Environmental Management Act (Government of the United Republic of Tanzania, 2004) that specifically provides for the control and management of current and future POPs, requiring submission of an annual report on implementation.

1.7.3 Occupational exposure limits

(a) USA

The manufacture of PCBs ended in the USA in 1977. Standards for occupational exposures (permissible exposure limits; PELs) in the USA are set by the Occupational Safety and Health Administration (OSHA) (29CFR1910.1000 Table Z-1 Limits for air contaminants). The PELs are 8-hour TWAs unless otherwise noted, and are determined from breathing-zone air samples. The PELs established by OSHA are 1000 μg/m^3 for PCB mixtures containing 42% chlorine, and 500 μg/m^3 for PCB mixtures containing 54%

chlorine (set in 1971 and not revised after this time). Both standards encompass all physical forms of these compounds: aerosols, vapour, mist, sprays, and PCB-laden dust particles. OSHA recognizes that PCBs are absorbed through intact skin; therefore, routes for dermal and inhalation exposure should be evaluated by an industrial hygienist. The National Institute for Occupational Safety and Health (NIOSH) recommends a 10-hour TWA of 1 μg/m^3 based on minimum reliable detectable concentration and the potential carcinogenicity of PCBs. NIOSH also recommends that all workplace exposures be reduced to the lowest feasible level.

(b) Europe

The maximum allowable airborne concentrations for PCBs containing 42% and 54% chlorine in the Federal Republic of Germany [before reunification] were 1.0 and 0.5 mg/m^3, respectively; and in Sweden, 0.5 mg/m^3 (IARC, 1978).

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2. CANCER IN HUMANS

2.1 Cohort studies of occupational exposure

Commercial mixtures of congeners of polychlorinated biphenyls (PCBs) were manufactured starting in the 1920s in Austria, France, Germany, Italy, Japan, Spain, Poland, the Russian Federation, the United Kingdom, and the USA. No published epidemiological studies of cancer among PCB-production workers were available to the Working Group.

2.1.1 Capacitor manufacture

Studies of cancer mortality and incidence among workers exposed to PCBs in the manufacture of capacitors have been conducted in Italy (Bertazzi *et al.*, 1982, 1987; Tironi *et al.*, 1996; Pesatori *et al.*, 2013), Sweden (Gustavsson *et al.*, 1986; Gustavsson & Hogstedt, 1997), and the USA (Brown & Jones, 1981; Brown, 1987; Sinks *et al.*, 1992; Kimbrough *et al.*, 1999, 2003; Mallin *et al.*, 2004; Prince *et al.*, 2006a, b; Ruder *et al.*, 2006; Silver *et al.*, 2009). The details of cohort studies among capacitor-manufacturing workers are presented in Table 2.1.

Bertazzi *et al.* (1982, 1987) studied 544 male and 1556 female former capacitor-production workers exposed between 1946 and 1980 at one capacitor-manufacturing plant in Monza, Italy. Cancer mortality until 1991 was non-statistically significantly increased among men (standardized mortality ratio, SMR, 1.1; 95% CI, 0.7–1.7; 20 deaths) and women (SMR, 1.2; 95% CI, 0.7–1.8; 19 deaths) (Tironi *et al.*, 1996). The most recent update also included 373 male and 97 female workers at a second plant that operated from 1950 to 1982 (Pesatori *et al.*, 2013). There was no excess overall cancer mortality; however, mortality due to cancers of the digestive tract, not otherwise specified, was statistically significantly increased (SMR, 2.5; 95% CI, 1.2–5.3; seven deaths). Deaths due to cancer of the brain (SMR, 1.8; 95% CI, 0.9–3.6; eight deaths) and lymphoma (SMR, 1.9; 95% CI, 1.0–3.3; twelve deaths) were in excess, especially for Hodgkin disease (SMR, 4.0; 95% CI, 1.3–12; three deaths) among women. Men were at increased risk of mortality from cancer of the biliary tract (SMR, 3.9; 95% CI, 1.5–10.4; four deaths) and cancer of the prostate (SMR, 1.7; [95% CI, 0.8–3.5]; seven deaths). [This cohort was notable for the high proportion of women.]

Gustavsson & Hogstedt (1997) studied cancer incidence and mortality until 1991 among 242 male capacitor-manufacturing workers employed for at least 6 months between 1965 and 1978 at a plant in Sweden. Individuals were classified as "high-exposed" if they had ever worked in the impregnation or repair departments. Cancer mortality was not significantly elevated among highly exposed workers (SMR, 1.9; 95% CI, 0.8–3.9; seven deaths). Two cases of cancer of the liver and bile duct were diagnosed (SMR, 6.7; 95% CI, 0.0–37 for highly exposed workers). Mortality from non-Hodgkin lymphoma (NHL) was increased among highly exposed workers based on one case (SMR, 9.1; 95% CI, 0.2–51).

Table 2.1 Cohort studies in capacitor-manufacturing workers

Reference, location, follow-up period	Total No. of subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Tironi *et al.* (1996), Italy, 1954–1982	1556 women, 544 men	Employment, 1 wk, 1946–82	All cancers (140–209)	All women	19	SMR, 1.2 (0.7–1.8)	Update of cohort studied by Bertazzi *et al.* (1982, 1987)
				All men	20	SMR, 1.1 (0.7–1.7)	
			Digestive organs (150–159)	All women	2	SMR, 0.9 (0.1–3.3)	
				All men	10	SMR, 2.0 (0.9–3.6)	
			Lymphatic & haematopoietic (200–209)	All women	5	SMR, 1.4 (0.5–3.3)	
				All men	3	SMR, 2.0 (0.4–5.9)	
			Lymphoma (200–202)	Women	4	SMR, 1.8 (0.5–4.5)	
Pesatori *et al.* (2013), Italy, 1946–1978 (plant 1)	1551 women and 544 men (plant 1); 97 women and 373 men (plant 2)	Employment > 1 wk 1946–1978 (plant 1), all workers employed 1950–1982 (plant 2); PCBs used until 1980	All cancers	All workers	183	SMR, 1.0 (0.9–1.0)	
			Lymphoma (200–202)		12	SMR, 1.9 (1.1–1.3)	
			Digestive NOS (159)		7	SMR, 2.5 (1.2–5.3)	
			Brain		8	SMR, 1.8 (0.9–3.6)	
			Breast	All women	16	SMR, 0.8 (0.5–1.3)	
			Prostate	All men	7	SMR, 1.7 (0.8–3.5)	
Gustavsson & Hogstedt (1997); Gustavsson *et al.* (1986), Sweden, 1965–1991	242 men	Employed > 6 mo, 1965–1978; low, medium, or high exposure to PCBs	All cancers (140–209)	High-exposed	7	SMR, 1.9 (0.8–3.9)	Age, calendar period, country of origin
			Liver (155)	High-exposed	1	SMR, 6.7 (0.02–37)	
			Lung (162)	High-exposed	2	SMR, 2.2 (0.3–8.0)	
			Prostate (185)	High-exposed	1	SMR, 2.2 (0.1–12)	
			Lymphatic & haematopoietic (200–209)	High-exposed	1	SMR, 3.3 (0.1–19)	
			Lymphoma (200–202)	High-exposed	1	SMR, 9.1 (0.2–51)	

Table 2.1 (continued)

Reference, location, follow-up period	Total No. of subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Mallin *et al.* (2004), Illinois, USA, 1944–2000	2885 white (25 non-white workers excluded)		All cancers (140–208)	All	347	[SMR, 1.1 (1.0–1.2)]	Sex, age, race, calendar period
			Stomach (151)	All	17	[SMR, 1.9 (1.1–3.1)]	Workers also exposed to trichloroethylene, 1,1,1-trichloroethane, lead solder, mineral oil, lacquer, paint thinner, epoxies, methyl ethyl ketone
			Intestine excluding rectum (152–153)	All	39	[SMR, 1.3 (0.9–1.7)]	
			Biliary passages, liver, & gallbladder (155–156)	All	14	[SMR, 2.4 (1.3–4.1)]	
			Thyroid (193)	Men	3	SMR, 15.2 (3.1–45)	No deaths from thyroid cancer among women
			Rectum (154)	All	7	[SMR, 1.1 (0.5–2.4)]	
			Prostate (185)	Men	9	SMR, 1.1 (0.5–2.0)	
			Breast (174–175)	Men	49	SMR, 1.2 (0.9–1.6)	
			NHL (200, 202)	Women:			No NHL deaths among those who worked 5–9 years. Data not reported for men
				Worked < 1 yr	7	SMR, 2.1 (0.8–4.3)	
				Worked 1–4 yr	4	SMR, 1.6 (0.4–4.1)	
				Worked ≥ 10 yr	2	SMR, 1.9 (0.2–6.8)	
			Oral cavity & pharyn (140–149)	Men only	3	SMR, 1.1 (0.2–3.3)	No deaths among women
Ruder *et al.* (2006), Indiana, USA, 1957–1998	3569	JEM based on department, job, tasks, monitored exposure levels, estimated cumulative exposure for each worker		*Cumulative exposure*			Sex, age, race, calendar period
			All cancers	Lowest tertile (< 11 000 unit-days)	56	SMR, 0.9 (0.7–1.2)	
				Middle tertile (11 000–89 999 unit days)	62	SMR, 0.9 (0.7–1.2)	
				Highest tertile (≥ 90 000 unit-days)	52	SMR, 0.8 (0.6–1.1)	*P* for trend = 0.48
			Melanoma	Lowest tertile	5	SMR, 3.7 (1.2–8.7)	
				Middle tertile	2	SMR, 1.5 (0.2–5.4)	
				Highest tertile	9	SMR, 2.4 (1.1–4.6)	*P* for trend = 0.72
			Brain	Lowest tertile	3	SMR, 1.4 (0.3–4.0)	
				Middle tertile	4	SMR, 1.8 (0.5–4.6)	
				Highest tertile	5	SMR, 2.7 (0.9–6.3)	*P* for trend = 0.016

Table 2.1 (continued)

Reference, location, follow-up period	Total No. of subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Ruder et al. (2006), Indiana, USA, 1957–1998 (cont.)			Breast	Lowest tertile	4	SMR, 1.0 (0.3–2.7)	
				Middle tertile	3	SMR, 0.9 (0.2–2.7)	
				Highest tertile	0	–	
			Prostate	Lowest tertile	1	SMR, 0.5 (0.0–2.7)	
				Middle tertile	2	SMR, 0.8 (0.1–2.7)	
				Highest tertile	1	SMR, 0.3 (0.0–1.8)	
			NHL (200, 202)	Lowest tertile	1	SMR, 0.4 (0.0–2.3)	
				Middle tertile	5	SMR, 1.9 (0.6–4.5)	
				Highest tertile	3	SMR, 1.3 (0.3–3.8)	
			Oral cavity & pharyn	Lowest tertile	2	SMR, 2.0 (0.2–7.1)	
				Middle tertile	0	–	
				Highest tertile	1	SMR, 0.9 (0.0–4.9)	
Prince *et al.* (2006b), Hopf *et al.* (2010), Massachusetts & New York, USA, 1939–1998	14 458	JEM for each plant based on department, job, tasks, monitored exposure levels, estimated cumulative exposure for each worker		Cumulative exposure: referent category < 150 unit-yr			Sex, age, race, calendar period The New York plant was also studied by Kimbrough *et al.* (1999, 2003). Results for 0-yr lag
			All cancers	150 to < 620 unit-yr	229	RR, 1.1 (0.9–1.3)	*P* for trend = 0.03
				620 to < 2300 unit-yr	238	RR, 1.3 (1.1–1.5)	
				≥ 2300 unit-yr	240	RR, 1.3 (1.1–1.5)	
			Melanoma	150 to < 620 unit-yr	2	RR, 0.3 (0.1–1.3)	*P* for trend = 0.83
				≥ 620 unit-yr	6	RR, 0.7 (0.2–1.9)	
			Brain	150 to < 620 unit-yr	5	RR, 0.6 (0.2–1.8)	*P* for trend = 0.32
				620 to < 2300 unit-yr	3	RR, 0.4 (0.1–1.6)	
				≥ 2300 unit-yr	3	RR, 0.5 (0.1–1.7)	
			Stomach	150 to < 620 unit-yr	6	RR, 1.5 (0.5–4.9)	
				620 to < 2300 unit-yr	10	RR, 3.2 (1.1–9.3)	
				≥ 2300 unit-yr	8	RR, 2.9 (0.9–9.2)	*P* for trend = 0.12
			Intestine excluding rectum	150 to < 620 unit-yr	26	RR, 1.5 (0.8–2.6)	*P* for trend = 0.55
				620 to < 2300 unit-yr	26	RR, 1.5 (0.8–2.6)	
				≥ 2300 unit-yr	27	RR, 1.4 (0.8–2.6)	

Table 2.1 (continued)

Reference, location, follow-up period	Total No. of subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Prince et al. (2006b), Hopf et al. (2010), Massachusetts & New York, USA, 1939–1998 (cont.)			Rectum	150 to < 620 unit-yr	5	RR, 1.1 (0.3–3.9)	
				620 to < 2300 unit-yr	1	RR, 0.2 (0.0–1.8)	
				≥ 2300 unit-yr	8	RR, 1,4 (0.4–4.3)	*P* for trend = 0.36
			Biliary passages, liver, & gallbladder	150 to < 620 unit-yr	3	RR, 1.7 (0.3–10.0)	
				620 to < 2300 unit-yr	6	RR, 3.1 (0.6–15)	
				≥ 2300 unit-yr	9	RR, 4.2 (0.9–20)	*P* for trend = 0.07
			Breast	150 to < 620 unit-yr	26	RR, 1.1 (0.6–1.9)	
				620 to < 2300 unit-yr	19	RR, 0.8 (0.4–1.4)	
				≥ 2300 unit-yr	27	RR, 1.3 (0.8–2.3)	*P* for trend = 0.26
			Prostate	150 to < 620 unit-yr	5	RR, 1.5 (0.4–5.6)	
				620 to < 2300 unit-yr	7	RR, 2.8 (0.8–9.6)	
				≥ 2300 unit-yr	18	RR, 6.1 (2.0–18)	*P* for trend < 0.01
			NHL (200, 202)	150 to < 620 unit-yr	13	RR, 1.6 (0.7–3.6)	
				620 to < 2300 unit-yr	3	RR, 0.5 (0.1–1.7)	
				≥ 2300 unit-yr	7	RR, 1.2 (0.4–3.3)	*P* for trend = 0.99
			Myeloma (203)	150 to < 620 unit-yr	6	RR, 1.5 (0.5–4.9)	
				620 to < 2300 unit-yr	9	RR, 2.4 (0.8–7.3)	
				≥ 2300 unit-yr	8	RR, 1.9 (0.6–5.9)	*P* for trend = 0.48
Kimbrough *et al.* (2003), New York, USA, 1946–1998	7075	Duration of employment, whether hourly or salaried	All cancers (140–208)	Hourly workers	381	[SMR, 1.0 (0.9–1.2)]	Sex, age, race, calendar period The plant was also studied by Prince *et al.* (2006b) and Silver *et al.* (2009)
				Salaried workers	111	[SMR, 0.8 (0.7–1.0)]	
			Prostate	Hourly workers	17	SMR, 1.3 (0.7–1.8)	
				Salaried workers	4	SMR, 0.5 (0.1–1.4)	
			Brain	Hourly workers	5	[SMR, 0.5 (0.2–1.2)]	
				Salaried workers	6	[SMR, 1.5 (0.6–3.4)]	
			Breast	Hourly workers	32	SMR, 0.9 (0.6–1.3)	
				Salaried workers	6	SMR, 0.9 (0.3–1.9)	
			Skin, including melanoma	Hourly workers	9	[SMR, 1.2 (0.6–2.4)]	
				Salaried workers	6	[SMR, 2.1 (0.8–4.7)]	
			Biliary passages, liver, & gallbladder	Hourly workers	6	[SMR, 0.97 (0.4–2.1)]	
				Salaried workers	1	[SMR, 0.3 (0.0–2.6)]	

Table 2.1 (continued)

Reference, location, follow-up period	Total No. of subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Kimbrough et al. (2003), New York, USA, 1946–1998 (cont.)			Intestine excluding rectum	Hourly workers	41	[SMR, 1.3 (0.9–1.7)]	
				Salaried workers	11	[SMR, 0.9 (0.5–1.7)]	
			Rectum	Hourly workers	8	[SMR, 1.2 (0.5–2.4)]	
				Salaried workers	4	[SMR, 1.6 (0.4–4.5)]	
			Oral cavity	Hourly workers	4	[SMR, 2.0 (0.6–5.2)]	
				Salaried workers	1	[SMR, 1.1 (2.9–6.4)]	
Silver *et al.* (2009), Indiana, Massachusetts & New York, USA, 1940–1998	5752 women	JEMs (see Ruder *et al.*, 2006 and Prince *et al.*, 2006b for description) Questionnaire for non-occupational risk factors	Breast	All	257	SIR, 0.8 (0.7–0.9)	Sex, age, race, calendar period. Results for subcohort with questionnaire data (n = 3141). Exposure lagged 10 yr
				Cumulative exposure per 1000 unit-yr:			Age, race, calendar period, ever smoking, parity, age at first live birth, breast cancer in first-degree female relative, age began hormone use
				All women	145	HR, 1.0 (1.0–1.1)	
				White women	131	HR, 1.0 (1.0–1.0)	
				Non-white women	14	HR, 1.3 (1.1–1.6)	

HR, hazard ratio; JEM, job-exposure matrix; mo, month; NHL, non-Hodgkin lymphoma; NOS, not otherwise specified; RR, rate ratio; SIR, standardized incidence ratio; SMR, standardized mortality ratio; SRR, standardized rate ratio; wk, week; yr, year

[Findings based on this small cohort were difficult to interpret because of limited precision.]

A cohort of 2885 white workers employed between 1944 and 1977 at a capacitor-manufacturing facility in Illinois, USA, who were exposed to PCBs (1952–1977), chlorinated naphthalenes (1944–1981), and other chemicals, was followed until 2000 (Mallin *et al.*, 2004). Plant records were incomplete and short-term workers (less than 1 year employment) were least likely (83%) to have been traced. There was excess mortality from cancers of the stomach [SMR, 1.9; 95% CI, 1.1–3.1], liver and biliary tract [SMR, 2.4; 95% CI, 1.3–4.1] and breast [SMR, 1.2; 95% CI, 0.9–1.6]. Women with 5 or more years employment during the period of PCB use had significantly elevated mortality from cancers of the liver and biliary tract (SMR, 5.6; 95% CI, 1.5–14; four deaths) and intestine (SMR, 2.3; 95% CI, 1.0–4.3; nine deaths). Men had excess mortality from cancer of the thyroid (SMR, 15.2; 95% CI, 3.1–45; three deaths), while women had excess mortality from NHL, which was not related to the duration of employment (SMRs, 1.6–2.1). Data on NHL were not reported for men. [Exposure assessment was limited and workers were exposed to multiple chemicals, which hampered attribution of cancer outcomes to PCB exposure.]

The United States National Institute for Occupational Safety and Health (NIOSH) cohort (Ruder *et al.*, 2014) included 25 000 workers at facilities in three states, originally studied separately, in Indiana (Sinks *et al.*, 1992; Ruder *et al.*, 2006) and Massachusetts and New York (Brown & Jones, 1981; Brown, 1987; Prince *et al.*, 2006a, b), and combined for an analysis of cancer of the breast (Silver *et al.*, 2009). Separate job-exposure matrices were developed for each of the plants, based on department, job title, era, company records, information about job tasks, and sampling data (Nilsen *et al.*, 2004; Hopf *et al.*, 2009, 2010), with each worker receiving an estimated cumulative exposure score, so that cancer outcomes could be analysed by level of relative exposure.

Updating vital status until 1998 for the Indiana subcohort (which comprised 3569 workers exposed to PCBs between 1957 and 1977) confirmed the earlier findings of excess melanoma and cancer of the brain (Sinks *et al.*, 1992). Melanoma remained in excess (SMR, 2.4; 95% CI, 1.1–4.6), particularly in the lowest tertile of estimated cumulative exposure (SMR, 3.7; 95% CI, 1.2–8.7; five deaths). Mortality from cancer of the brain (SMR, 1.9; 95% CI, 1.0–3.3) increased with exposure, with a standardized mortality ratio of 2.7 (95% CI, 0.9–6.3; five deaths) in the highest quartile and a significant exposure–response trend in the standardized rate ratio (SRR) ($P = 0.02$). Among those having worked ≥ 90 days, both melanoma (SMR, 2.7; 95% CI, 1.1–5.2) and cancer of the brain (SMR, 2.1; 95% CI, 1.1–3.8) were elevated, especially for women (melanoma: SMR, 6.0; 95% CI, 1.2–17.5; three deaths; cancer of the brain: SMR, 2.9; 95% CI, 0.6–8.4; three deaths). The standardized mortality ratio for mortality from NHL was 1.2 (95% CI, 0.6–2.3) (Ruder *et al.*, 2006).

The original studies in the Massachusetts-New York subcohorts (Brown & Jones, 1981; Brown, 1987) included only 2567 workers considered to be highly exposed to PCBs during 1938–1977 (Massachusetts) or 1946–1977 (New York). The update until 1998 expanded the study population to include 14 458 workers with at least 90 days of potential exposure to PCBs (Prince *et al.*, 2006b). Cancer of the liver, leukaemia and aleukaemia [aplastic anaemia], and NHL were not in excess overall, but mortality from multiple myeloma was (SMR, 1.85; 95% CI, 1.23–2.67). In the New York subcohort, mortality from melanoma was elevated (SMR, 1.79; 95% CI, 0.98–3.0). Mortality from cancer of the stomach was elevated among men (SMR, 1.53; 95% CI, 0.98–2.28) and increased with cumulative exposure (trend, $P = 0.039$). Mortality from cancer of the prostate was not elevated overall (SMR, 1.0; 95% CI, 0.72–1.45),

but increased with cumulative exposure (trend, $P < 0.001$). Mortality from intestinal cancer was elevated among women (SMR, 1.31; 95% CI, 1.02–1.66), especially in categories with higher cumulative exposure, but did not show a clear trend.

[The NIOSH studies were originally reported in multiple, overlapping publications based on several plants, but were subsequently merged into a single cohort. The Working Group regarded the quality of the NIOSH studies as high, and noted that they represented considerable effort to enumerate, expand and update the cohorts and assess exposure using objective job-exposure matrices.]

In addition to the NIOSH studies, separate analyses were conducted independently for the New York plant (Kimbrough *et al.*, 1999, 2003). These studies, which used duration of employment and whether hourly or salaried as surrogates for exposure, reported on virtually the same workers as in the NIOSH New York subcohort (mortality until 1998, employed at least 90 days, 7075 workers versus the 6941 studied by NIOSH), but found no significant excess mortality for any cancers (Kimbrough *et al.*, 1999, 2003). [The Working Group noted that the analyses by Kimbrough included 134 more workers than did Prince et al. but was not able to determine the reason for the discrepancy. In addition, Kimbrough et al. presented results only in subgroups defined by sex and pay grade, limiting the power of the analyses.]

The NIOSH study of cancer of the breast (Silver *et al.*, 2009) included 5752 women employed for at least 1 year in any one of the three capacitor-manufacturing facilities studied previously by NIOSH. Exposure to PCBs was estimated semiquantitatively using job-exposure matrices and information about incident cancer of the breast, parity, age at first live birth, breast cancer in a first-degree female relative, hormone use, and smoking was used in analyses for 3952 women who completed questionnaires. Cancer registries and death certificates up to 1998 were used to identify 281 incident cases. The overall standardized incidence ratio (SIR) for cancer of the breast was 0.8 (95% CI, 0.7–0.9), with little effect of employment duration or cumulative exposure. However, for the 282 women of race identified by questionnaire as "other than white," there was a positive, statistically significant association with cumulative exposure, with a hazard ratio for cancer of the breast of 1.3 (95% CI, 1.1–1.6) per 1000 unit-years of estimated cumulative exposure, while no association was observed in "white" women.

2.1.2 Transformer manufacture and repair

Studies of cancer mortality and incidence among workers exposed to PCBs in the manufacture or repair of transformers have been conducted in Canada (Yassi *et al.*, 1994, 2003), Italy (Caironi *et al.*, 2005), and the USA (Greenland *et al.*, 1994; Table 2.2).

Cancer mortality among a subset of deceased former workers at a transformer-manufacturing plant in Massachusetts, USA, was evaluated for (ever having had) exposure to PCBs (Pyranol) (Greenland *et al.*, 1994). There were positive associations with cancer of the liver and biliary tract (odds ratio, OR, 2.4; 95% CI, 0.6–9.7) and lymphoma (OR, 3.3; 95% CI, 1.1–9.3). In an analysis adjusted for age at death, year of death, and year of hire, the adjusted odds ratio was 2.2 (95% CI, 0.8–6.5) for cancer of the liver and biliary tract and 1.5 (95% CI, 0.55–4.3) for lymphoma. [The Working Group noted that numbers of deaths by site associated with exposure to PCBs were not reported, and job histories were unavailable for 34% of the study population.]

Cancer incidence and mortality until 1995 were studied in a cohort of 2222 men working between 1946 and 1975 at a transformer-manufacturing plant in Manitoba, Canada, where PCBs (Askarels) were used from 1956 to fill large transformers (mineral oils were used in other

Table 2.2 Cohort studies in transformer-manufacturing and transformer-repair workers

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Greenland *et al.* (1994), Massachusetts, USA, 1969–1984	1821 deceased white male workers, aged 21–90 yr, vested in company pension plan	Expert assessment	Oral cavity, larynx, pharynx	Pyranol exposure, ever	NR	OR, 1.1 (0.4–3.4)	Age at death, yr of hire, yr of death. Job history unavailable for 34% of deceased former workers; non-white men and women excluded; workers with > 50% work history unrated for PCBs excluded; deceased < 1969 or not vested (10–15 yr work) excluded. No. of exposed deaths, NR. Pyranol contained about 50% PCB. Other exposures included solvents, machining fluids, asbestos, resins
			Oesophagus		NR	OR, 0.9 (0.2–4.1)	
			Stomach		NR	OR, 0.9 (0.3–3.1)	
			Colon excluding rectum		NR	OR, 0.6 (0.3–1.4)	
			Rectum		NR	OR, 0.9 (0.3–2.3)	
			Pancreas		NR	OR, 1.1 (0.4–2.6)	
			Biliary passages, liver, and gallbladder		NR	OR, 2.4 (0.6–9.7)	
				Pyranol exposure at 97th percentile of control exposure	NR	OR, 2.2 (0.8–6.5)	
			Trachea, bronchus, & lung	Pyranol exposure, ever	NR	OR, 1.0 (0.6–1.6)	
			Prostate		NR	OR, 0.8 (0.4–1.7)	
			Bladder		NR	OR, 0.5 (0.1–2.3)	
			Kidney		NR	OR, 0.4 (0.1–3.4)	
			Lymphoma (200–203)	Pyranol exposure at 97th percentile of control exposure	NR	OR, 3.3 (1.1–9.3)	
					NR	OR, 1.5 (0.6–4.3)	
			Leukaemia (204–208)	Pyranol exposure, ever	NR	OR, 0.5 (0.1–2.1)	
			Brain		NR	OR, 1.1 (0.3–3.9)	

Table 2.2 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Yassi *et al.*(1994, 2003), Manitoba, Canada, 1946–1995; 1950–1995 (mortality); 1969–1995 (cancer incidence)	2222 men			Employment:			
			All cancers	> 1 mo	NR	SMR, 1.2 (1.0–1.5)	13% excluded from original mortality study because of missing identifiers. Total of 261 deaths in cohort until 1995
				> 6 mo	NR	SMR, 1.2 (0.9–1.6)	Total of 104 deaths in subcohort until 1995
				Transformer assembly	NR	SMR, 1.6 (0.9–2.8)	Total of 31 deaths in transformer-assembly department until 1995
			Digestive organs (150–159)	> 1 mo	NR	SMR, 1.3 (0.9–1.9)	
				> 6 mo	NR	SMR, 1.3 (0.6–2.3)	
				Transformer assembly	NR	SMR, 2.7 (1.0–5.9)	
			Stomach	> 1 mo	NR	SMR, 0.8 (0.2–2.3)	
				> 6 mo	NR	SMR, 1.8 (0.4–5.2)	
				Transformer assembly	NR	SMR, 5.1 (9.6–18)	
			Pancreas	> 1 mo	NR	SMR, 3.6 (1.9–6.1)	
				> 6 mo	NR	SMR, 4.8 (2.1–9.5)	
				Transformer assembly	NR	SMR, 7.5 (1.5–2.2)	
			Melanoma	> 6 mo	8	SMR, 1.8 (0.2–6.4)	
			All cancers	> 1 mo	NR	SIR, 1.2 (1.0–1.4)	Total diagnoses, 168
				> 6 mo	NR	SIR, 1.0 (0.8–1.3)	Total diagnoses, 65
				Transformer assembly	NR	SIR, 1.1 (0.6–1.7)	Total diagnoses, 18
			Digestive organs (150–159)	> 1 mo	NR	SIR, 1.4 (1.1–1.9)	
				> 6 mo	NR	SIR, 1.1 (0.6–1.8)	
				Transformer assembly	NR	SIR, 1.6 (0.6–3.4)	
			Stomach	> 1 mo	NR	SIR, 1.3 (0.5–2.7)	
				> 6 mo	NR	SIR, 0.4 (0.0–2.4)	
				Transformer assembly	NR	SIR, 1.7 (0.0–9.5)	

Table 2.2 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Yassi et al.(1994, 2003), (cont.)			Pancreas	> 1 mo	NR	SIR, 2.7 (1.3–4.9)	
				> 6 mo	NR	SIR, 4.3 (1.7–8.8)	
				Transformer assembly	NR	SIR, 7.2 (1.5–21.1)	
			Gall bladder	> 1 mo	NR	SIR, 5.1 (1.4–13)	
				> 6 mo	NR	SIR, 2.9 (0.0–16)	
				Transformer assembly	NR	0	
			Melanoma	> 1 mo	10	SIR, 2.2 (1.1–4.0)	
Caironi *et al.* (2005), Bergamo, Italy, 1950–early 1990s; 1950–2002	471 (372 men, 99 women)		Stomach	All exposed	7	SMR, 1.6 (0.6–2.5)	No. of deaths, but not SMRs reported for other cancers (oral cavity, 4; oesophagus, 1; pancreas, 1; larynx, 2; lung, 18; breast, 3; prostate, 3; bladder, 2; lymphoma, 3; other cancers, 4)
			Intestine excluding rectum (153–4, 159)	All exposed	11	SMR, 2.6 (1.6–3.5)	
			Liver	All exposed	3	SMR, 0.3 (0.0–1.1)	
			Leukaemia (204–208)	All exposed	2	SMR, 1.8 (0.0–3.6)	

HR, hazard ratio; JEM, job-exposure matrix; mo, month; NHL, non-Hodgkin lymphoma; NR, not reported; RR, rate ratio; SIR, standardized incidence ratio; SMR, standardized mortality ratio

transformers) (Yassi *et al.*, 2003). The mortality study showed an increased risk of mortality for cancer of the digestive tract, particularly cancers of the stomach and pancreas, among workers in the transformer-assembly department. The incidence study included ten cases of malignant melanoma in the full cohort (SIR, 2.2; 95% CI, 1.1–4.0). Increased risk of cancers of the gall bladder and pancreas was also observed among all workers, and an excess of cancer of the pancreas was reported among workers in the transformer-assembly department (SIR, 7.2; 95% CI, 1.5–21.1) (Yassi *et al.*, 2003). [The Working Group noted that the authors did not assess individual exposure to PCBs, which makes it difficult to attribute effects specifically to PCBs.]

In a study in Bergamo, Italy, among 471 workers who built transformers between 1950 and 1988, using PCBs until 1980 and mineral oils thereafter, and who repaired transformers from 1988 until the early 1990s, mortality from cancer of the intestine was significantly elevated (SMR, 2.6; 95% CI, 1.6–3.5; 11 deaths), but mortality from cancer of the stomach or liver, or leukaemia, was not (Caironi *et al.*, 2005). [This was a small study, but it focused on transformer-repair workers who would have had substantial dermal exposure to PCBs.]

2.1.3 Electric power and telecommunications

Studies of cancer mortality and incidence among workers exposed to PCBs in the electric-power and telecommunications industries have been conducted in Canada (De Guire *et al.*, 1988; Hay & Tarrel, 1997), Italy (Cammarano *et al.*, 1984, 1986), Norway (Tynes *et al.*, 1994), and the USA (Savitz & Loomis, 1995; Loomis *et al.*, 1997; Charles *et al.*, 2003; Table 2.3).

De Guire and coworkers found increased incidence of and mortality from malignant melanoma among 9590 employees of a telecommunications company in Montreal, Canada, who had been employed for 6 months or more between 1976 and 1983 (De Guire *et al.*, 1988, 1992). Three deaths were identified among men (SMR, 3.0; 95% CI, 0.6–8.8), with a stronger association for those with < 20 years latency (SMR, 9.4; 95% CI, 1.1–34; two deaths) than for those with ≥ 20 years latency (one death; SMR, 1.3; 95% CI, 0.0–7.1). Only one case occurred among women (SMR, 4.8; 95% CI, 0.1–27). [This was a reasonably large cohort, but the number of incident cases was small. Exposure to PCBs may have occurred, but was not assessed.]

Cancer incidence among 5088 workers in the hydroelectric-power industry in Norway employed for at least 1 year between 1920 and 1991 was examined in relation to magnetic fields or electric sparks, and to exposure to PCBs (Tynes *et al.*, 1994). Workers were classified as ever or never exposed to PCBs, based on work histories. The incidence of malignant melanoma was increased in the full cohort (SIR, 1.1; 95% CI, 0.7–1.8) and among power-supply electricians (SIR, 2.1; [95% CI, 1.0–3.7]). Significantly increased incidence was also reported among workers ever exposed to PCBs and to > 15 μT-years of magnetic fields (SIR, 2.7; [95% CI, 1.2–5.2]). [This study investigated exposure to PCBs and to electric and magnetic fields. Exposures to PCBs and to electric and magnetic fields may be correlated through associations with certain jobs, but exposure is unlikely to confound the association with PCBs, as such exposure is not known to be associated with melanoma.]

Loomis and colleagues assessed risk of cancer in relation to PCB exposure among 138 905 male employees of five utility companies in California, North Carolina, Pennsylvania, Tennessee, and Virginia, USA, who were employed for at least 6 months between 1950 and 1986 (Savitz & Loomis, 1995; Loomis *et al.*, 1997). Exposures were assessed jointly by representatives of employees and management and by industrial hygienists. Mortality from melanoma increased with increasing exposure to PCBs, from 1.2 (95% CI, 0.6–2.5) for those with < 2000 hours cumulative

Table 2.3 Cohort studies in electric-power and telecommunications workers

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
De Guire *et al.* (1988, 1992), Montreal, Canada, 1976–1983	9590	Working on 1 January 1976 or up to 31 December 1963, ≥ 6 mo employment. Exposed to polyvinyl chloride, soldering fumes, and PCBs	All cancers	Men	67	SMR, 0.6 (0.5–0.7)	
			Oral cavity, larynx, pharynx	Men	1	SMR, 0.2 (0.0–1.0)	
				Women	17	SMR, 0.9 (0.5–1.4)	
			Digestive organs (150–159)	Men	22	SMR, 0.7 (0.4–1-1)	
				Women	5	SMR, 1.2 (0.4–2.9)	
			Trachea, bronchus, lung	Men	26	SMR, 0.6 (0.4–0.8)	
				Women	4	SMR, 1.5 (0.4–4.0)	
			Melanoma	Men	3	SMR, 3.0 (0.6–8.8)	
				Women	1	SMR, 4.8 (0.1–27)	
				Men, < 20 yr latency	2	SMR, 9.4 (1.1–34)	
				Men, ≥ 20 yr latency	1	SMR, 1.3 (0.0–7.1)	
				Women, < 20 yr latency	1	SMR, 12.1 (0.0–67)	
			Eye, brain	Men	2	SMR, 0.5 (0.1–1.7)	
			Lymphatic and haematopoietic (200–208)	Men	7	SMR, 0.7 (0.3–1.5)	
			Bone, breast (170–171, 173–178)	Women	5	SMR, 0.9 (0.3–2.0)	
Tynes *et al.* (1994), Norway, 1920–1991; 1953–1991	5088 men	Worked ≥ 1 yr at any of eight hydroelectric-power companies	Rectum	Employment ≥ 1 yr	27	SIR, 1.1 (0.7–1.6)	
			Lung		68	SIR, 1.1 (0.9–1.4)	
			Breast		1	SIR, 1.1 (0.0–76)	
			Prostate		90	SIR, 1.1 (0.9–1.3)	
			Bladder		27	SIR, 0.8 (0.5–1.2)	
			Melanoma		19	SIR, 1.1 (0.7–1.8)	
			Brain		13	SIR, 0.9 (0.5–1.5)	
			Lymphoma		12	SIR, 0.7 (0.4–1.2)	
			Leukaemia		11	SIR, 0.9 (0.5–1.6)	
			Melanoma	Ever exposed to PCBs	9	SIR, 1.8 [0.8–3.4]	Incidence of other cancers not analysed in association with PCB exposure
				Ever exposed to PCBs, 0–15 μT-yr	0		
				Ever exposed to PCBs, > 15 μT-yr	9	SIR, 2.7 [1.2–5.2]	

Table 2.3 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Loomis *et al.* (1997), California, North Carolina, Pennsylvania, Tennessee, Virginia, USA, 1950–1988	138 905 men	Employed > 6 mo, 1950–1986, exposures assessed by panels of workers, hygienists, managers; calculated cumulative exposure to insulating fluids containing PCBs	All cancers	Potential PCB exposure:			Age, calendar time, race, social class, active work status.
				> 0 to < 5 year	916	RR, 2.2 (0.9–1.2)	
				5 to < 10 year	454	RR, 1.0 (0.9–1.2)	
				10 to < 20 year	601	RR, 1.1 (1.0–1.2)	
				≥ 20 year	656	RR, 1.1 (1.0–1.2)	
				Cumulative PCB exposure (h), 20-yr lag:			
				> 0–2000	2605	RR, 1.0 (1.0–1.1)	
				> 2000–10 000	331	RR, 1.2 (1.1–1.3)	
				> 10 000	81	RR, 1.0 (0.8–1.3)	
			Brain	Potential PCB exposure:			Age, calendar time, race, social class, active work status, magnetic fields, solvents
				0 to < 5 yr	32	RR, 1.3 (0.8–2.2)	
				5 to < 10 yr	15	RR, 1.4 (0.7–2.6)	
				10 to < 20 yr	17	RR, 1.3 (0.7–2.4)	
				≥ 20 yr	12	RR, 1.1 (0.6–2.2)	
				Cumulative PCB exposure (h), 20-yr lag:			
				> 0–2000	66	RR, 1.0 (0.7–1.6)	
				> 2000–10 000	5	RR, 0.7 (0.3–1.9)	
				> 10 000	0	RR, 0.0 (0.0–2.6)	
			Liver (155)	Potential PCB exposure:			Age, calendar time, race, social class, active work status, solvents
				0 to < 5 yr	13	RR, 1.1 (0.5–2.3)	
				5 to < 10 yr	5	RR, 0.8 (0.3–2.2)	
				10 to < 20 yr	13	RR, 1.8 (0.9–3.6)	
				≥ 20 yr	5	RR, 0.7 (0.3–1.9)	
				Cumulative PCB exposure (h), 20-yr lag:			
				> 0 to 2000	29	RR, 0.5 (0.3–0.5)	
				> 2000–10 000	3	RR, 0.4 (0.1–1.4)	
				> 10 000	1	RR, 0.4 (0.1–3.0)	

Table 2.3 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Loomis et al. (1997), (cont.)			Melanoma	Potential PCB exposure:			Age, calendar time, race, social class, active work status, occupational sunlight, wood preservatives
				0 to < 5 yr	25	RR, 1.3 (0.6–2.6)	
				5 to < 10 yr	9	RR, 1.1 (0.5–2.7)	
				10 to < 20 yr	11	RR, 1.4 (0.6–3.3)	
				≥ 20 yr	8	RR, 1.6 (0.6–4.2)	
				Cumulative PCB exposure (h), 0-yr lag:			
				> 0–2000	73	RR, 1.2 (0.6–2.5)	
				> 2000–10 000	12	RR, 1.7 (0.7–7.1)	
				> 10 000	3	RR, 1.9 (0.5–7.1)	
				Cumulative PCB exposure (h), 20-yr lag:			
				> 0 to 2000	42	RR, 1.3 (0.8–2.2)	
				> 2000–10 000	8	RR, 2.6 (1.1–6.0)	
				> 10 000	1	RR, 4.8 (1.5–15)	
				RR per 2000 h cumulative PCB exposure (continuous variable):			
				0-yr lag	-	RR, 1.02 (0.99–1.05)	
				20-yr lag	-	RR, 1.05 (1.01–1.09)	
Charles *et al.* (2003), California, North Carolina, Pennsylvania, Tennessee, Virginia, USA, 1950–1988	387 cases of prostate cancer and 1935 controls matched on age at risk	See Loomis *et al.* (1997)	Prostate (185)	Cumulative PCB exposure (h):			Age-matched and adjusted for race Same cohort studied by Loomis *et al.* (1997)
				< 1.9	94	OR, 1.0	
				1.9 to < 12.6	85	OR, 0.9 (0.6–1.2)	
				12.6 to < 620.1	105	OR, 1.1 (0.8–1.5)	
				620.1 to < 2821.4	55	OR, 0.8 (0.6–1.2)	
				≥ 2821.4	48	OR, 1.2 (0.8–1.7)	

Table 2.3 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Charles et al. (2003) (cont.)				Cumulative PCB exposure (h), 5-yr lag:			
				< 1.6	91	OR, 1.0	
				1.6 to < 12.1	87	OR, 0.9 (0.6–1.2)	
				12.1 to < 597.9	104	OR, 1.1 (0.8–1.5)	
				597.9 to < 2763.2	58	OR, 0.9 (0.6–1.3)	
				≥ 2763.2	47	OR, 1.1 (0.8–1.7)	
				Cumulative PCB exposure ≥ 2763.2 h and EMF ≥ 4.4 μT-yr	35	OR, 1.5 (1.0–2.2)	Equivalent results for total cumulative exposure
Hay & Tarrel (1997), New Brunswick, Canada, 1950–1966; 1950–1992	225 men		All cancers	First sprayed 1950–1958	18	SMR, 1.5 (0.9–2.3)	Sprayed vegetation under power lines with 2,4-D and 2,4,5-T; 1958–66, waste transformer oil with PCBs added to herbicides
				First sprayed 1959–1966	3	SMR, 1.1 (0.2–3.2)	
Cammarano *et al.* (1984, 1986), Milano, Italy, 1960–1969; 1969–1985	270 men	Working on 1 January 1960 or up to 31 December 1969, ≥ 6 mo employment		Exposure:			Exposed to PAHs, asbestos, hydrazine, chromium, nickel, beryllium, and PCBs SMRs from Cammarano *et al.* (1986) All other cancer sites had one or zero death
			All cancers	≥ 10 yr	18	[SMR, 2.2 (1.3–3.4)]	
			Stomach	≥ 10 yr	3	[SMR, 3.0 (0.6–8.7)]	
			Trachea, bronchus, lung	≥ 10 yr	5	[SMR, 1.8 (0.6–4.1)]	
			Bladder	≥ 10 yr	2	[SMR, 7.4 (0.9–26)]	

EMF, electromagnetic fields; mo, month; OR, odds ratio; PAH, polycyclic aromatic hydrocarbon; PCB, polychlorinated biphenyl; RR, rate ratio; SIR, standardized incidence ratio; SMR, standardized mortality ratio; wk, week; yr, year

exposure to PCBs, to 1.7 (95% CI, 0.7–7.1) among those with 2000–10 000 hours cumulative exposure, to 1.9 (95% CI, 0.5–7.1) for those with > 10 000 hours of cumulative exposure over their career. When exposure was lagged by 20 years, the respective relative risks were 1.3 (95% CI, 0.8–2.2), 2.6 (95% CI, 1.1–6.0), and 4.8 (95% CI, 1.5–15.0). When the risk of melanoma was modelled with a continuous variable for cumulative exposure to PCBs, the relative risk per 2000 hours of exposure was 1.05 (95% CI, 1.01–1.09) with a 20-year lag. There was no association with cancer of the liver, and the association with cancer of the brain was less strong: the relative risk was 1.6 (95% CI, 0.9–3.0) among those with < 2000 hours cumulative exposure and 1.8 (95% CI, 0.8–4.0) among those with 2000–10 000 hours cumulative exposure, but there were no deaths from cancer of the brain among those with > 10 000 hours cumulative exposure (Loomis *et al.*, 1997).

A nested case–control study within this utility-worker cohort investigated mortality from cancer of the prostate relative to exposure to electromagnetic fields and PCBs (Charles *et al.*, 2003). Cases were 387 prostate-cancer decedents; 1935 controls (5 per case) were randomly selected from the risk sets of the cases. The odds ratio for cumulative exposure to PCBs for ≥ 2821.4 hours and mortality from cancer of the prostate, adjusted for age and race, was 1.2 (95% CI, 0.8–1.7). For workers with ≥ 2763.2 hours of exposure to PCBs and ≥ 4.4 μT years of exposure to magnetic fields, the adjusted odds ratio was 1.5 (95% CI, 1.0–2.2).

[The Working Group considered that, because of the size of the cohort and the efforts to assess exposure, the Loomis–Charles studies were the strongest in this group, especially the results showing an exposure–response effect. The lagged analysis of melanoma mortality was informative about exposure-time windows.]

Some information about cancer risk among electrical workers with exposure to PCBs was reported in two smaller studies. Hay & Tarrel (1997) investigated mortality in 1958–1991 among power-company workers in Canada who applied mixtures of the pesticides 2,4-D (2,4-dichlorophenoxyacetic acid) and 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) and waste transformer oil that contained up to 10% PCBs. All-cancer mortality was increased among workers who first sprayed in 1958 or earlier (SMR, 1.5; 95% CI, 0.9–2.3; 18 deaths), but not among those first exposed in 1959 or later, when used transformer oil was added to the pesticide mix (SMR, 1.1; 95% CI, 0.2–3.2; three deaths). [The Working Group noted that the results were not presented by cancer site and concluded that exposures to PCBs were likely to have been negligible.]

Mortality until 1985 was investigated among 270 men who had worked for at least 6 months in a thermoelectric power plant in Italy and who were exposed to PCBs, chromium, nickel, beryllium, polycyclic aromatic hydrocarbons (PAHs), asbestos, and hydrazine (Cammarano *et al.*, 1984, 1986). Among workers with > 10 years exposure, 18 cancer deaths occurred [SMR, 2.2; 95% CI, 1.3–3.4] (Cammarano *et al.*, 1986). [The Working Group noted that workers were exposed to several human carcinogens and that the study was very small, with only one death for most cancer sites, making it difficult to interpret site-specific mortality.]

2.1.4 Miscellaneous industries

As PCBs have been used in many applications, workers in many industries have been exposed, and as structures and equipment that contain PCBs are repaired, demolished, or replaced, workers involved in these operations and/or in waste recycling and disposal may be exposed. There have been many reports of PCB exposure levels and existing or potential health effects associated with exposure to materials containing PCBs, but studies of cancer are very limited.

Robinson *et al.* (1999) conducted a proportionate mortality study of 31 068 deceased,

unionized, electrical workers employed in the construction industry, who might have been exposed to PCBs (and other agents) during their working lives. Excess mortality occurred for melanoma (proportionate mortality ratio, PMR, 1.23; 95% CI, 1.02–1.47) and cancer of the prostate (PMR, 1.07; 95% CI, 1.00–1.14). [Although this very large death-certificate study found an excess risk for cancers that have been associated with exposure to PCBs in other PCB-exposed cohorts, exposure in this cohort could not be confirmed.]

Unspecified industrial uses of PCBs have been associated with an increased risk of cancer. Bahn and colleagues reported two cases of malignant melanoma among 31 workers in research and development and refinery industries in New Jersey, USA, who were exposed to PCB mixtures, where 0.04 cases would be expected [SIR, 50.0; 95% CI, 5.6–217] (Bahn *et al.*, 1976).

2.2 Cohort studies of environmental exposure

2.2.1 Accidental exposure to PCBs

(a) Cancer mortality in Yusho patients, Japan

The first evaluation by IARC of the possible carcinogenic risk of human exposure to PCBs reported the accidental exposure to PCBs through ingestion of rice oil contaminated by Kanechlor 400 in 1968 in western Japan (see Section 1). In an early analysis of deaths occurring up to 5.5 years after exposure among 1200 Yusho patients, nine deaths from malignant neoplasms were reported, including three tumours of the stomach, two tumours of the lung, one cancer of the liver, one of the breast, and two lymphomas (Urabe, 1974; Kuratsune, 1976). A first update considered mortality among 1761 Yusho patients followed up until 1983 (Kuratsune *et al.*, 1988). Among men, there was a statistically significant increase in mortality from all neoplasms (SMR, 2.13; 95% CI, 1.5–3.0), and particularly cancer of the liver (SMR, 5.6; 95% CI, 2.6–10.7), and lung (SMR, 3.2; 95% CI, 1.4–6.3). No statistically significant increase in tumours was reported among the women.

After these early reports, two other mortality analyses of this cohort have been published with follow-up periods up to 1990 and up to 2007 respectively (see Table 2.4). The first report (Ikeda & Yoshimura, 1996) analysed the mortality of 1815 patients (916 men and 899 women), with an average follow-up of 17 years. In the 40-year follow-up of the total of 1918 patients registered as of 31 December 2007 (Onozuka *et al.*, 2009), 254 cases who had not been diagnosed as Yusho from the beginning of the incident were excluded, leaving 1664 cases for analysis (860 men and 804 women). Of the 269 deaths among men, there was a significant excess mortality from all cancers (SMR, 1.37; 95% CI, 1.11–1.66), and from cancers of the lung (SMR, 1.75; 95% CI, 1.14–2.57) and liver (SMR, 1.82; 95% CI, 1.06–2.91). For women, mortality for cancer of the liver was in excess, although not significantly so (SMR, 1.95; 95% CI, 0.78–4.01). Analysis of different periods since the incident showed that the increased risk for all malignancies, and for cancers of the lung and liver tended to decrease over time.

A more recent analysis that did not exclude the 254 patients diagnosed after 1977 (Yoshimura, 2012) reported essentially the same pattern of mortality, with slightly weaker standardized mortality ratios for cancers of the lung and liver (Table 2.4).

Finally, another analysis of mortality of Yusho patients followed up until 2007 was restricted to the area of Tamamoura in the Goto Archipelago (Nagasaki prefecture), because it was the most severely affected (Kashima *et al.*, 2011). Standardized mortality ratios for all cancers, lung cancer, and liver cancer were estimated using the rates of Nagasaki prefecture as the reference and compared for the years 1968–77 and 1978–2002. A slight excess cancer of the

Table 2.4 Cohort studies of cancer associated with poisoning from rice oil contaminated with PCBs in Japan and Taiwan, China

Reference, location follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases/ deaths	SMR (95% CI)	Covariates Comments
Yusho patients							
Onozuka *et al.* (2009) Fukuoka and Nagasaki, Japan 1968–2007	1664 Yusho patients	Mass poisoning by contaminated rice oil		Overall, compared with national death rates	100 men, 33 women		Age, sex Total number of Yusho patients was 1918, but 254 subjects registered after 1977 (not diagnosed as Yusho from the beginning of the incident) were excluded in this analysis
		Men	All cancers		100	1.37 (1.11–1.66)	
			Liver		17	1.82 (1.06–2.91)	
			Lung		26	1.75 (1.14–2.57)	
			Stomach		20	1.17 (0.72–1.81)	
			Rectum		2	0.65 (0.08–2.36)	
			Pancreas		6	1.49 (0.55–3.24)	
			Leukaemia		2	1.19 (0.14–4.29)	
		Women	All cancers		33	0.75 (0.51–1.05)	
			Liver		7	1.95 (0.78–4.01)	
			Lung		4	0.82 (0.22–2.11)	
			Stomach		2	0.22 (0.03–0.81)	
			Rectum		1	0.56 (0.01–3.10)	
			Pancreas		3	1.02 (0.21–2.98)	
			Leukaemia (204–206)		0	0.00 (0.00–3.25)	
			Breast		3	0.93 (0.19–2.72)	
			Uterus		3	1.14 (0.24–3.33)	
Yoshimura (2012) Fukuoka and Nagasaki, Japan 1968–2007	1918 Yusho patients	Mass poisoning by contaminated rice oil		Overall, compared with national death rates			Age, sex As for Onozuka *et al.* (2009), including the 254 subjects registered after 1977
		Men	All cancers		106	1.26 (1.03–1.53)	
			Liver		18	1.67 (0.99–2.63)	
			Lung		27	1.56 (1.03–2.27)	
			Stomach		21	1.09 (0.68–1.67)	
		Women	All cancers		46	0.89 (0.65–1.17)	
			Liver		8	1.87 (0.81–3.69)	
			Lung		5	0.86 (0.28–2.01)	
			Stomach		4	0.39 (0.11–0.99)	

Table 2.4 (continued)

Reference, location follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases/ deaths	SMR (95% CI)	Covariates Comments
Kashima *et al.* (2011) Fukuoka and Nagasaki, Japan 1968–2002	533 Yusho patients from Tamamoura area	Mass poisoning by contaminated rice oil		Rates from Tamamoura, compared with Nagasaki prefecture	329 (total)		Age, sex As for Onozuka *et al.* (2009) for both sexes combined, using different reference population; Tamanoura was the most affected area Liver cancer was not mentioned in the analysis of the period 1968–77
		1968–77	All cancers		86	1.13 (0.92–1.40)	
			Lung		11	1.37 (0.76–2.48)	
		1978–2002	All cancers		243	1.03 (0.91–1.17)	
			Liver		21	0.77 (0.50–1.18)	
			Lung		37	0.87 (0.63–1.20)	
Yucheng patients							
Tsai *et al.* (2007) Three counties in central Taiwan, China 1980–2003	1823 Yucheng patients	Mass poisoning by contaminated rice oil		Overall, compared with national death rates	215 deaths (129 men, 86 women)		Age, sex There was also a significant association for mortality by chronic liver disease and cirrhosis (ICD-9 571)
		Men (n = 841)	All cancers		29	0.9 (0.6–1.3)	
			Nasopharynx		3	2.3 (0.5–6.8)	
			Liver & intrahepatic bile ducts		4	0.5 (0.1–1.2)	
			Lung		7	1.1 (0.4–2.2)	
			Lymphatic & haematopoietic (200–208)		4	2.3 (0.6–6.0)	
		Women (n = 987)	All cancers		12	0.7 (0.3–1.1)	
			Nasopharynx		0	–	
			Liver & intrahepatic bile ducts		4	1.6 (0.4–4.1)	
			Lung		1	0.3 (0.0–1.9)	
			Lymphatic & haematopoietic		0	–	
		Both sexes	All cancers		41	0.8 (0.6–1.1)	
			Nasopharynx		3	1.6 (0.3–4.7)	
			Liver & intrahepatic bile ducts		8	0.7 (0.3–1.4)	
			Lung		8	0.8 (0.4–1.6)	
			Lymphatic & haematopoietic (200–208)		4	1.3 (0.4–3.4)	

Table 2.4 (continued)

Reference, location follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases/ deaths	SMR (95% CI)	Covariates Comments
Li *et al.* (2013) Three counties in central Taiwan, China 1980–2008	1803 Yucheng patients and 5170 referents (neighbours)	Mass poisoning by contaminated rice oil		Overall, compared with neighbourhood referents	295 deaths (178 men, 117 women)		Age, sex, community Significant association for mortality from chronic liver disease and cirrhosis (ICD-9 571)
		Men (*n* = 830)	All neoplasms (148–239)		46	1.3 (0.9–1.7)	
			Liver & intrahepatic bile ducts (155)		4	0.4 (0.1–1.1)	
			Trachea, bronchus & lung (162)		10	1.5 (0.8–2.7)	
			Stomach (151)		7	3.5 (1.5–7.0)	
			Lymphatic & haematopoietic (200–208)		5	3.0 (1.1–6.6)	
			Thyroid gland (193)		0	–	
		Women (*n* = 973)	All neoplasms (148–239)		21	0.8 (0.5–1.2)	
			Liver & intrahepatic bile ducts (155)		6	2.1 (0.9–4.5)	
			Trachea, bronchus & lung (162)		1	0.4 (0.0–1.7)	
			Stomach (151)		1	0.5 (0.0–2.5)	
			Lymphatic & haematopoietic (200–208)		0	–	
			Thyroid gland (193)		2	2.0 (0.3–6.7)	
			Breast, female (174)		4	1.1 (0.4–2.7)	
		Both sexes	All neoplasms (148–239)		67	1.1 (0.8–1.4)	
			Liver & intrahepatic bile ducts (155)		10	0.9 (0.4–1.5)	
			Trachea, bronchus & lung (162)		11	1.1 (0.6–1.9)	
			Stomach (151)		8	2.0 (0.9–3.8)	
			Lymphatic & haematopoietic (200–208)		5	1.5 (0.6–3.4)	
			Thyroid gland (193)		2	2.2 (0.4–7.2)	

PCB, polychlorinated biphenyl; SMR, standardized mortality ratio

lung was observed in Tamamoura in 1968–77 (SMR, 1.37; 95% CI, 0.76–2.48) [data for cancer of the liver not reported for that period] and no increase in mortality was seen during the later period (Table 2.4). However, significant excess mortality for all cancers, and for cancers of the lung or liver, were observed for the rest of the Goto Archipelago (excluding Tamamoura) in 1978–2002.

[The Working Group noted that excess cancer mortality was largely restricted to men. In addition the excesses of cancers of the lung and liver were observed in the full population of Yusho patients in analyses using national references rates, but not in the subset from the Tamamoura area analysed using local reference rates. Important confounders such as tobacco smoking for cancer of the lung, or viral hepatitis for cancer of the liver could not be taken into account directly, although they may have been partially controlled for by using local reference rates, if the distribution of such confounders in the local reference population were similar to that of the study population. Yusho patients were also exposed to PCDFs. The possibility of confounding by other exposures therefore could not be completely ruled out.]

(b) Cancer mortality in Yucheng patients, Taiwan, China

In 1979, about 10 years after the incident in western Japan, a similar food poisoning incident occurred in three counties (Taichung, Changhua, and Miaoli) of central Taiwan, China (see Section 1). About 2000 residents from these counties had ingested rice oil contaminated with PCBs, and showed clinical manifestations similar to those described for Yusho (skin eruptions and pigmentation, ocular hypersecretion, and peripheral neuropathy); the syndrome was named 'Yucheng' ('oil disease' in Chinese) (see Section 4). Two mortality analyses have been carried out on this exposed cohort, after 12 and 24 years of follow-up, and are summarized in Table 2.4. The first study cohort was based upon 2038 cases registered until 1979; after excluding 99 cases for which vital status could not be assessed, 1940 [sic] Yucheng patients (929 men, 1011 women) remained for analysis of mortality (Hsieh *et al.*, 1996). During 1980–91, 11 deaths from malignancies were observed (8 men, 3 women); overall and sex-specific mortality was non-significantly lower than among the general population, using either local or national reference rates. Data for specific tumour sites were sparse, and included one death from Hodgkin lymphoma and two deaths from cancer of the liver (one man and one woman). Another analysis of the same study population was conducted with the same follow-up (1980–91), but further exclusions, leaving 1837 patients for analysis and 10 observed deaths from cancer (Yu *et al.*, 1997). Although the standardized mortality ratio for all cancers differed substantially from that in the previous analysis, it was not significantly different from that expected based on national rates (SMR, 1.2; 95% CI, 0.6–2.3). Data for specific cancer sites were not reported. [The Working Group noticed the discrepancy between estimates of standardized mortality ratio based upon apparently very similar data sets.]

Data for updated analyses of Yucheng patients are shown in Table 2.4. Tsai *et al.* (2007) extended the follow-up to 2003. From a list of 2061 registered patients, 70 exposed in utero and 168 who could not be traced were excluded, leaving 1823 patients. Forty-one deaths by cancer were observed between 1980 and 2003. Mortality from all neoplasms was not statistically different from that in the population in Taiwan, China, overall or by sex; mortality from cancers at several sites, including liver, lung, and the lymphatic and haematopoietic system, was also similar to that of the national population. As in a previous study, mortality from chronic liver disease and cirrhosis was significantly increased. [The Working Group noted that chronic liver disease and cirrhosis are important risk factors for cancer of the liver,

together with infection with hepatitis B and C viruses, and tobacco smoking.]

A second updated analysis of Yucheng patients extended the follow-up to 2008 (Li *et al.*, 2013). As referents for comparison, the authors used subjects from the registry set up in 1979, residents of the same community, of the same sex and age (within 3 years) as the Yucheng patients, but who did not meet the criteria to be considered as Yucheng patients. After exclusions because of missing or inconsistent data, a total of 1803 Yucheng subjects and 5170 neighbourhood referents were considered for analysis; a total of 67 Yucheng patients died from cancer during 1980–2008. No significant association with all cancer mortality was found overall or among women. Among men, increased mortality was reported for cancer of the stomach (SMR, 3.5; 95% CI, 1.5–7.0, seven deaths) and neoplasms of lymphatic and haematopoietic tissue (SMR, 3.0; 95% CI, 1.1–6.6, five deaths). Mortality from cancer of the liver was elevated among women (SMR, 2.1; 95% CI, 0.9–4.5, six deaths), but not among men (SMR, 0.4; 95% CI, 0.1–1.1, four deaths). [The neighbourhood referent population used in this study may also have been exposed, which would lead to underestimation of relative risks.]

[The Working Group noted that the excess mortality from all cancers and tumours of the liver observed in Yusho patients was not present in Yucheng patients. The composition of PCDF isomers differed markedly between the two incidents: the main PCDF isomer in Yusho patients was 2,3,4,7,8-pentachlorinated dibenzofuran, which has a higher toxic equivalency factor than the main isomer affecting Yucheng patients, 1,2,3,4,7,8-hexachlorinated dibenzofuran (Onozuka *et al.*, 2009). On the other hand, no excess mortality for cancers of the stomach or lymphatic and haematopoietic tissue was observed in the Yusho patients. The same other limitations mentioned for the Yusho cohort applied to the Yucheng studies: residual confounding, or chance due to multiple comparison made in these analyses could not be discounted.]

2.2.2 Dietary exposure to PCBs

See Table 2.5

Apart from incidental contamination, chronic exposure to PCBs may occur through a diet rich in foods with a high content of PCBs; such exposure has been observed in northern Europe in populations with a high consumption of fish.

Cohorts of fishermen from the east coast and west coasts of Sweden were established in 1968 and 1965 respectively (Rylander & Hagmar, 1995; Svensson *et al.*, 1995). Women who were, or had been, married to these fishermen were identified from national and local population registries. After exclusion because of death, divorce, or emigration, the respective cohorts of fishermen's wives included 1986 women on the east coast and 6605 women on the west coast (Rylander & Hagmar, 1995). Information on vital status and cancer incidence up to 1989 was gathered from Swedish statistics and the Swedish cancer registry. Cancer incidence was compared directly between the cohorts on the east coast (contaminated) and west coast (control), with adjustment for age and calendar year. The incidence rate ratio (IRR) for all cancers was 1.19 (95% CI, 1.00–1.41). Among specific cancer sites, risk was increased for cancer of the breast (IRR, 1.35; 95% CI, 0.98–1.86), cervix (IRR, 1.93; 95% CI, 0.83–4.50) and corpus uteri (IRR, 1.16; 95% CI, 0.61–2.20). All cancer mortality was also significantly more elevated in the east-coast cohort when compared with the regional rates (SIR, 1.17; 95% CI, 1.00–1.36). Dietary information showed modest differences in consumption of fatty fish between the east and west coasts. In a recent update extending the follow-up until 2002 (Mikoczy & Rylander, 2009) expected mortality and cancer incidence were based on national

Table 2.5 Cohort studies of risk of cancer associated with high dietary intake of PCBs

Reference, location, follow-up period	Total subjects	Exposure assessment/ population	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Mikoczy & Rylander (2009) Sweden 1968–2002 (east coast) 1965–2002 (west coast)	2042 (east coast) and 6674 (west coast) fishermen's wives	Dietary intake of fatty fish from Baltic Sea (east coast)		Comparison with national rates		*SIR (95% CI)*	Age Possible coexposure to PCDDs and PCDFs
		West coast	All sites		1201	0.92 (0.87–0.98)	
			Stomach		39	0.86 (0.61–1.18)	
			Colon		103	0.97 (0.79–1.18)	
			Rectum		52	1.00 (0.75–1.31)	
			Liver, bile ducts		39	0.99 (0.70–1.36)	
			Lung		33	0.61 (0.42–0.86)	
			Breast		305	0.90 (0.81–1.01)	
			Melanoma		38	1.03 (0.73–1.41)	
			Skin		60	1.43 (1.09–1.84)	
			Brain		41	1.05 (0.75–1.42)	
			Soft tissue sarcoma		3	0.38 (0.08–1.10)	
			Lymphohaematopoietic (200–207)		75	0.92 (0.73–1.16)	
			Hodgkin lymphoma (201)		3	0.63 (0.13–1.83)	
			Multiple myeloma (203)		19	1.12 (0.68–1.76)	
			NHL (200, 202)		35	1.03 (0.71–1.43)	
		East coast	All sites (140–209)		345	1.09 (0.98–1.21)	
			Stomach		12	1.39 (0.72–2.43)	
			Colon		38	1.61 (1.14–2.21)	
			Rectum		13	1.09 (0.58–1.86)	
			Liver, bile ducts		11	1.35 (0.67–2.42)	
			Lung		12	0.83 (0.43–1.46)	
			Breast		92	1.03 (0.83–1.27)	
			Melanoma		8	0.76 (0.33–1.49)	
			Skin		9	0.95 (0.43–1.80)	
			Brain		14	1.37 (0.75–2.30)	
			Soft tissue sarcoma		1	0.51 (0.01–2.84)	
			Lymphohaematopoietic (200–207)		18	0.94 (0.56–1.48)	
			Hodgkin lymphoma (201)		1	0.93 (0.02–5.19)	
			Multiple myeloma (203)		6	1.58 (0.58–3.43)	
			NHL (200, 202)		6	0.71 (0.26–1.55)	

Table 2.5 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment/ population	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Turunen *et al.* (2008) Finland 1980–2005	4260 fishermen's wives	Dietary intake of fatty fish from Baltic Sea		Overall, compared with national death rates		*SMR (95% CI)*	Age
			All malignant neoplasms		115	0.97 (0.80–1.15)	
			Colon		10	1.30 (0.62–2.39)	
			Rectum & anus		8	2.13 (0.92–4.19)	
			Stomach		2	0.30 (0.04–1.08)	
			Breast		18	0.80 (0.47–1.25)	
			Larynx, trachea & lung		8	0.70 (0.30–1.38)	
			Lymphoid, haematopoietic, & related tissue		10	0.83 (0.40–1.53)	
Helmfrid *et al.* (2012) Gusum, Sweden 1960–2003	Residents in contaminated area (number not given)	Consumption of foods with high PCB content from contaminated local river	(ICD-7)	Overall, compared with national death rates		*SIR (95% CI)*	Age, time period Possible coexposure to metals because of industrial activities
		Men	All sites		346	0.91 (0.78–1.05)	
			Stomach		25	1.00 (0.65–1.83)	
			Colon		21	0.76 (0.46–1.16)	
			Rectum		10	0.54 (0.25–0.99)	
			Liver/bile ducts		8	0.88 (0.37–1.73)	
			Pancreas		14	1.17 (0.63–1.97)	
			Bronchus & lung		22	0.64 (0.40–0.97)	
			Breast		1	NR	
			Prostate		100	1.06 (0.86–1.29)	
			Testis		7	2.46 (0.99–5.08)	
			Malignant melanoma of skin		15	1.56 (0.87–3.94)	
			Other skin		15	0.81 (0.45–1.34)	
			Brain		3	0.31 (0.06–0.91)	
			Lymphoma (200–202)		22	1.60 (1.00–2.42)	
			Multiple myeloma (203)		7	1.25 (0.50–2.42)	
			Leukaemia (204)		5	0.88 (0.28–3.57)	
			Lymphatic & haematopoietic tissues (200–207)		38	1.20 (0.84–1.65)	

Table 2.5 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment/ population	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Helmfrid et al. (2012) Gusum, Sweden 1960–2003 (cont.)		Women	All sites		295	0.91 (0.77–1.07)	
			Stomach		15	1.11 (0.62–1.88)	
			Colon		17	0.65 (0.37–1.04)	
			Rectum		12	0.95 (0.49–1.66)	
			Liver/bile ducts		9	0.91 (0.41–1.73)	
			Pancreas		6	0.62 (0.22–1.34)	
			Bronchus & lung		6	0.49 (0.18–1.08)	
			Breast		80	0.97 (0.77–1.21)	
			Malignant melanoma of skin		11	1.22 (0.60–2.19)	
			Other skin		7	0.70 (0.28–1.44)	
			Brain		13	1.37 (0.72–2.34)	
			Lymphoma (200–202)		8	0.82 (0.35–1.63)	
			Multiple myeloma (203)		2	0.49 (0.05–1.77)	
			Leukaemia (204)		4	1.25 (0.34–3.21)	
			Lymphatic & haematopoietic tissues (200–207)				
Tomasallo *et al.* (2010) Great Lakes area, USA 1995–2006	3757 subjects (2275 fish consumers, 1482 non-consumers)	Dietary intake of Great Lakes sport fish				*SMR (95% CI)*	
			All cancers	Fish consumers	83	0.92 (0.74–1.13)	
			Pancreas		6	1.24 (0.45–2.44)	
			Brain		5	1.91 (0.60–3.96)	
			Breast, ovary, & uterus		6	1.47 (0.46–3.04)	
			All cancers	Non-consumers	47	0.87 (0.64–1.13)	
			Pancreas		2	0.72 (0.07–2.07)	
			Brain		1	0.70 (0.0–2.76)	
			Breast, ovary, & uterus		1	0.44 (0.0–1.73)	

NHL, non-Hodgkin lymphoma; NR, not reported; OR, odds ratio; PCDDs, polychlorinated dibenzodioxins; PCDFs, polychlorinated dibenzofurans; SIR, standardized incidence ratio; SMR, standardized mortality ratio

rates, and no direct comparison between east- and west-coast cohorts were reported. Standardized mortality ratios for all cancers combined were 0.98 (0.91–1.06) for the west-coast cohort and 1.15 (95% CI, 0.98–1.34) for the east-coast cohort. Statistically significant excess incidence was reported for cancer of the colon in the east-coast cohort (SIR, 1.61; 95% CI, 1.14–2.21) and non-melanoma cancer of the skin in the west-coast cohort (SIR, 1.43; 95% CI, 1.09–1.84). [The Working Group noted that the excess of cancer incidence observed using regional rates as reference became nonsignificant when national rates were used. Because of the lack of specific exposure information, the possibility of confounding cannot be ruled out.]

In Finland, a cohort of Baltic Sea fishermen was identified from the Professional Fishermen Register, and their wives were identified from the Population Register (Turunen *et al.*, 2008). A cohort of 4260 women was linked with Statistics Finland's national cause-of-death data from 1980 to 2005, and expected deaths were calculated according to national rates. Furthermore, a cross-sectional substudy was conducted among 94 cohort participants who undertook a health examination in 2004–2005, including a food-frequency questionnaire and fasting-blood collection; data from a population-based survey were used for comparison. No statistically significant standardized mortality ratios were found for all cancers, or for any specific tumour site.

After an accidental spill of oil contaminated with PCBs from the brass works industry in Gusum, Sweden, in 1972, elevated levels of PCBs were measured in local fish in 2006. Among the population of the contaminated area, 641 cases of cancer were identified in 1960–2003, which was not above the expected number based on national rates for the same period (Helmfrid *et al.*, 2012). Among men, 22 lymphomas were observed, with a statistically significant increased standardized incidence ratio (SIR) of 1.60 (95% CI, 1.00–2.42). There was also an increased risk of cancer of the testis (SIR, 2.46; 95% CI, 0.99–5.08; seven cases) and malignant melanoma of the skin (SIR, 1.56; 95% CI, 0.87–3.94) in the contaminated area when compared with the general population, while the risk for cancer of the prostate was near unity (SIR, 1.06; 95% CI, 0.86–1.29). In addition to the cohort analysis, a case–control study based upon a dietary questionnaire was carried out on 67 cases of cancer, including cancers of the colorectum, skin (including melanoma), cervix, breast, prostate, and lymphoma, and 326 controls resident in the same area. The case–control analysis reported an increased risk of cancer of the female breast associated with consumption of fish more than twice per month, but with only two cases. Excess risks of lymphoma (five cases, including men and women) were also observed with consumption of fish more than twice per month. Consumption of locally produced foods was also analysed, but no other statistically significant increased risks associated with potential sources of exposure to PCBs were reported in the case–control analysis. [The Working Group noted that subjects from this area could have also been exposed to other contaminants, such as metals. The case–control analysis was based upon a very small number of subjects, and there was poor assessment of dietary exposure and control for potential confounders.]

Regular consumption of predatory fish constitutes a large source of exposure to several persistent pollutants, including PCBs, for residents of the Great Lakes Basin (Falk *et al.*, 1999). A cohort of regular consumers of sport fish from the Great Lakes, and residents in the same communities who consumed no sport fish from the Great Lakes (referents), were recruited in 1993–94 (Tomasallo *et al.*, 2010). A total of 3757 subjects (2275 fish consumers and 1482 referents) were followed from 1995 to 2006, and mortality was compared with national death rates. Information about fish consumption and other lifestyle characteristics was obtained by telephone interview, and a blood sample for measurement of PCBs

was collected for a subgroup of 610 individuals. During the 12-year follow-up period, 342 deaths were recorded, including 134 deaths from cancer. Cancer mortality rates did not differ from those of the general population for fish consumers or referents: SMRs for all cancers were 0.92 (95% CI, 0.74–1.13) and 0.87 (95% CI, 0.64–1.13), respectively. However, fish consumers had non-statistically significant excesses of cancers of the pancreas, brain and combined breast, uterus and ovary. Although blood PCB levels were positively associated with fish consumption among fish consumers ($P < 0.001$ for comparison of mean PCB concentrations according to three levels of fish consumption), there was no association between fish consumption and cancer mortality. [The Working Group regarded this study as informative because it included information about PCB exposures, as well as fish consumption. However, the possibility of confounding from concurrent exposure to other contaminants could not be ruled out.]

[Compared with cohorts of Yusho or Yucheng patients, who consumed food contaminated with a high level of PCBs for a short period, potential exposure to PCBs through diet is a long-term, low-level exposure. Fish or local vegetables contaminated by PCBs are often also contaminated by other compounds such as DDT, PCDFs, PCDDs, or heavy metals. Furthermore, as detailed information on other risk factors for the tumours analysed (i.e. lymphoma, breast, colon, skin) was lacking, residual confounding could not be ruled out as a potential explanation for the associations found in these studies.]

2.2.3 Nested case–control studies of PCB concentrations in blood or adipose tissue

Since the 1980s, several cohort studies have addressed the potential relationship between risk of cancer and internal measurements of exposure to PCBs. The most commonly used marker of past exposure to PCBs is the serum or plasma concentration of a set of PCB congeners, although a few studies have measured PCB concentrations in adipose tissue. Most studies used a case–control design nested within a cohort as an efficient method for analysis: PCB concentrations were measured in all incident cases diagnosed within a defined follow-up period, and in a sample of at-risk subjects (controls) selected within the same cohort. A few studies have used a case–cohort approach, in which the referent group is formed by a random sample of the whole cohort selected at baseline. Various sets of PCB congeners were measured; PCB-118, PCB-138, PCB-153, and PCB-180 were reported more often because they were frequently analysed and prevalent in human biological samples (see Section 1.2 for more information on analytical methods). In some instances results for individual congeners were provided but, unless otherwise specified, the summary estimate refers to the sum of all measured PCBs.

(a) Cancer of the breast

See Table 2.6

(i) USA

The New York University Women's Health Study (NYUWHS) enrolled 14 290 women from New York City between 1985 and 1991; these women donated a 30 mL blood sample while attending a mammography screening clinic (Wolff *et al.*, 1993). During this period, women who were diagnosed with cancer of the breast 1–6 months after entry into the study were defined as cases. Controls were selected at random from all cohort members who were alive and free of cancer at the time of the cancer diagnosis in a case patient, matched on menopausal status, age at entry and day of menstrual cycle at the time of blood collection. Concentrations of PCBs were measured without correction for serum lipids. [Since cases were diagnosed only 1–6 months subsequent to entry, the disease

Table 2.6 Nested case-control studies on risk of cancer of the breast and measured serum or adipose concentrations of PCBs

Reference, location, follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
USA							
Wolff *et al.* (2000a) New York, USA 1985–1991 until 1994	14 275 women; 148 cases and 295 controls	Serum, GC, lipid-corrected concentrations (Akins method)		Quartiles of PCB concentration (ng/g lipid)			Age, menopausal status, date of blood collection (matching), age at menarche, number of pregnancies, age at first pregnancy, family history of breast cancer, lactation, height, BMI No. and list of PCB congeners not provided; LOD, < 1 ng/mL
				478–638	30	1.55 (0.59–4.12)	
				639–876	26	1.23 (0.49–5.08)	
				> 876	33	2.02 (0.76–5.37)	
						P for trend = 0.23	
Krieger *et al.* (1994) Northern California, USA 1964–1969 until 1990	57 040 women; 150 case–control pairs (50 each white, black, Asian)	Serum, GC/ECD, no lipid adjustment		Tertiles of PCB concentration (ng/mL)			Race, age, date of entry, duration of follow-up (matching), BMI, age at menarche, menopausal status, ever pregnant No. and list of PCB congeners not provided; LOD, 2 ng/mL
			All women	3.5–5.0		1.17 (0.66–2.10)	
				5.1–20.6		0.94 (0.48–1.84)	*P* for trend = 0.88
			White	2.94–3.96		0 21 (0.05–0.88)	
				3.97–10.01		0 17 (0.03–0.89)	*P* for trend = 0.039
			Black	3.51–4.98		1.74 (0.56–5.43)	
				4.99–20.55		2 13 (0.70–6.50)	*P* for trend = 0.18
			Asian	4.16–5.76		1.56 (0.47–5.17)	
				5.77–14.62		1.06 (0.32–3.52)	*P* for trend = 0.93
Hunter *et al.* (1997), Laden *et al.* (2001a) 11 states, USA (Nurses' Health Study cohort) 1989–1994	32 826 women; 370 case–control pairs	Serum PCB levels measured by GC/ECD, no lipid adjustment		Quintiles of PCB concentration (µg/g lipid)			Age, menopausal status, month of blood collection, fasting status at blood sampling (matching), BMI, breast cancer in first-degree relatives, history of benign breast disease, age at menarche, first full term pregnancy, parity, lactation LOD, < 1 ng/mL; sum of 16 penta-, hexa-, and heptachlorobiphenyls; congeners 118, 138, 153 and 180 accounted for 64% of total
			Sum of PCBs	0.406–0.491	65	0.73 (0.44–1.21)	Continuous (log-concentration) *P* = 0.56
				0.491–0.596	65	0.75 (0.44–1.28)	
				0.602–0.763	80	0.85 (0.49–1.47)	
				0.766–1.986	74	0.84 (0.47–1.52)	

Table 2.6 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Hunter et al. (1997), Laden et al. (2001a) (cont.)			PCB-118	0.045–0.060	62	0.68 (0.39–1.17)	Continuous (log-concentration) $P = 0.67$
				0.061–0.074	61	0.62 (0.36–1.06)	
				0.074–0.101	90	1.02 (0.59–1.77)	
				0.101–0.313	69	0.69 (0.39–1.22)	
			PCB-138	0.066–0.087	69	0.82 (0.49–1.37)	Continuous (log-concentration) $P = 0.21$
				0.087–0.108	75	0.90 (0.53–1.50)	
				0.109–0.142	65	0.71 (0.41–1.20)	
				0.143–0.402	78	0.87 (0.50–1.50)	
			PCB-153	0.078–0.094	58	0.67 (0.39–1.14)	Continuous (log-concentration) $P = 0.26$
				0.095–0.121	75	0.69 (0.41–1.15)	
				0.121–0.159	69	0.77 (0.45–1.31)	
				0.159–0.447	79	0.83 (0.47–1.48)	
			PCB-180	0.055–0.068	65	0.70 (0.41–1.20)	Continuous (log-concentration) $P = 0.67$
				0.069–0.082	62	0.65 (0.37–1.11)	
				0.082–0.103	63	0.70 (0.41–1.19)	
				0.103–0.467	91	0.98 (0.55–1.75)	
				Tertiles of PCB concentration			
			BMI ≥ 30	Tertile 2	19/21	0.40 (0.15–1.05)	Continuous (log-concentration) $P = 0.02$
				Tertile 3	11/19	0.26 (0.09–0.76)	
			Nulliparous	Tertile 2	5/14	0.81 (0.18–3.68)	Continuous (log-concentration) $P = 0.02$
				Tertile 3	12/6	5.30 (1.06–26.6)	
Laden *et al.* (2002) 1989–1994	367 pairs	Serum PCB levels measured by GC/ECD, no lipid adjustment	*CYP1A1* exon 7 genotype	Tertiles of PCB concentration			Same data set as study by Hunter *et al.* (1997), Laden *et al.* (2001a)
			Wildtype	0.13–0.46	113	1.00	
			Variant	0.13–0.46	12	0.54 (0.24–1.22)	
				0.46–0.65	18	0.76 (0.35–1.63)	
				0.65–1.99	21	1.36 (0.60–3.12)	Interaction ($P = 0.19$)
			Postmenopausal women				
			Wildtype	0.13–0.47	84	1.00	
			Variant	0.13–0.47	16	0.52 (0.20–1.36)	
				0.47–0.67	12	1.29 (0.51–3.21)	
				0.67–1.99	7	2.78 (0.99–7.82)	Interaction ($P = 0.05$)

Table 2.6 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Dorgan *et al.* (1999) Missouri, USA 1977–1987 until 1989	7224 women; 105 cases and 208 matched controls	Serum, GC/ECD, lipid-corrected concentrations		Quartiles of PCB concentration (ng/g lipid)			Age, benign breast disease, mo/year blood collection (matching), height, weight, BMI, parity, age at menarche, menopause, estrogen use, history of breast cancer in first-degree relatives, smoking, education 70% lost to follow-up after 1983; LOD, 0.25–0.97ng/g; 27 PCB congeners measured[a]
			Sum of PCBs	258–369	21	0.7 (0.3–1.4)	Continuous (log-concentration) $P = 0.79$
				370–563	33	1.1 (0.6–2.2)	
				564–2682	21	0.7 (0.3–1.5)	
			PCB-118	50–74	25	1.1 (0.6–2.3)	Continuous (log-concentration) $P = 0.77$
				75–109	34	1.6 (0.8–3.2)	
				110–533	23	1.0 (0.5–2.2)	
			PCB-138	70–93	29	1.3 (0.6–2.5)	Continuous (log-concentration) $P = 0.82$
				94–124	26	1.2 (0.6–2.3)	
				125–359	26	1.2 (0.6–2.4	
Helzlsouer *et al.* (1999) Maryland, USA 1974–1994 or 1989–1994	20 305 recruited in 1974; 25 080 recruited in 1989; 340 cases and matched controls	Serum, GC/ECD, lipid-corrected concentrations		Sum of PCBs (ng/g lipid)			Age, race, menopausal status, date of blood collection
			Recruited in 1974	< 394.47	42	1.00	Approx. 70% participation; no association for specific congeners (data not reported); no effect modification by menopausal status, ER status, polymorphisms in *GSTM1*, *GSTT1*, *GSTP1*, *COMT* and *CYP17*; LOD, NR; 27 PCB congeners measured
				394.48–558.72	59	1.41 (0.79–2.50)	
				558.73–669.46	41	0.94 (0.49–1.77)	
				669.47–852.22	45	1.08 (0.59–2.01)	
				852.23–6460.04	48	1.12 (0.59–2.15)	P for trend = 0.44
			Recruited in 1989	13.6–191.8	40	1.00	
				191.9–333.5	32	0.78 (0.41–1.47)	
				333.6–2007.9	33	0.76 (0.38–1.51)	P for trend = 0.60

Table 2.6 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Cohn *et al.* (2012) Oakland, California, USA 1959–1967 until 1998 (average follow-up, 17 years)	Women in the CHDS who gave birth in 1959–1967 [number of participants not given]; 112 case–control pairs (cases all aged < 50 yr)	Serum samples collected during early post-partum, GC/ECD		Quartiles of PCB concentration (mmol/L)			Age (matching), blood lipids (total cholesterol, total triglycerides), parity, year of blood draw, BMI, breast-feeding after current pregnancy 10 congeners measured[b] No associations with total PCBs or with Wolff's groups (data not shown)
			PCB-167	Quartile 2	NR	1.09 (0.48–2.47)	*P* for trend < 0.04
				Quartile 3	NR	0.70 (0.27–1.78)	
				Quartile 4	NR	0.24 (0.07–0.79)	
			PCB-187	Quartile 2	NR	0.94 (0.41–2.17)	*P* for trend < 0.02
				Quartile 3	NR	0.92 (0.36–2.38)	
				Quartile 4	NR	0.35 (0.11–1.14)	
			PCB-203	Quartile 2	NR	1.21 (0.46–3.18)	*P* for trend < 0.001
				Quartile 3	NR	2.89 (0.98–8.55)	
				Quartile 4	NR	6.34 (1.85–21.7)	
Northern Europe							
Høyer *et al.* (1998, 2000) Copenhagen, Denmark (CCHS cohort) 1979–1993	5838 women with two examinations (1976–78 and 1981–83); 155 cases, 274 controls	Serum, GC/ECD, lipid-corrected concentrations		Quartiles of PCB concentration [unit not given]			Age, date of examination, weight changes between two examinations, parity, HRT Response rate 75% (first exam), 78% (second exam); LOD, 0.66–0.20 ng/mL; No. and list of PCB congeners not provided
			Sum of PCBs	Quartile 2	NR	0.8 (0.4–1.9)	
				Quartile 3	NR	0.8 (0.4–1.7)	
				Quartile 4	NR	1.6 (0.8–3.3)	*P* for trend = 0.17
			PCB-118	Quartile 2	NR	0.8 (0.4–1.9)	
				Quartile 3	NR	1.1 (0.5–2.4)	
				Quartile 4	NR	1.9 (0.9–3.9)	*P* for trend = 0.07

Table 2.6 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Høyer et al. (1998, 2000) (cont.)			PCB-138	Quartile 2	NR	0.9 (0.4–1.9)	
				Quartile 3	NR	1.0 (0.5–2.1)	
				Quartile 4	NR	2.1 (1.0–4.4)	*P* for trend = 0.04
			PCB-153	Quartile 2	NR	0.7 (0.3–1.4)	
				Quartile 3	NR	0.8 (0.4–1.8)	
				Quartile 4	NR	1.3 (0.2–2.6)	
			PCB-180	Quartile 2	NR	1.2 (0.6–2.5)	
				Quartile 3	NR	1.1 (0.5–2.2)	
				Quartile 4	NR	0.9 (0.4–2.2)	
Høyer *et al.* (2001)	161 cases, 318 controls		ER status	Quartiles of PCB concentration [unit not given]			Age, weight, parity, HRT See Høyer *et al.* (2000) for details
			ER+	811–1076.04	24/56	1.1 (0.6–1.7)	
				1076.04–1405.73	20/57	0.7 (0.4–1.2)	
				< 1405.73	36/56	1.3 (0.8–2.2)	
			ER–	811–1076.04	11/23	1.0 (0.4–2.7)	
				1076.04–1405.73	11/23	1.3 (0.4–3.9)	
				< 1405.73	8/23	0.8 (0.3–2.6)	
Høyer *et al.* (2002)	162 cases, 316 controls		*p53* mutations in tumour	Quartiles of PCB concentration [unit not given]			Age, weight, parity, HRT See Høyer *et al.* (2000) for details
			Wildtype	Quartile 2	24	0.53 (0.28–1.04)	
				Quartile 3	20	0.52 (0.26–1.05)	
				Quartile 4	34	0.96 (0.50–1.83)	
			≥ 1 p53 mutations	Quartile 2	9	1.78 (0.43–7.41)	
				Quartile 3	11	3.82 (0.85–17.4)	
				Quartile 4	10	3.00 (0.66–13.6)	

Table 2.6 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Ward *et al.* (2000) Norway (Janus cohort) 1973–1991	25 431 women working outside home and resident on a farm; 150 case–control pairs	Serum, HRGC/ID-HRMS, lipid-corrected concentrations		Quartiles of PCB concentration (ng/g lipid)		[95% CI not given]	Age (matching), occupation, age at first birth, parity, residence All cases ≥ 2 years from blood collection to diagnosis; sum of 36 congeners: 26 with > 90% samples > LOD Groups according to Wolff's classification (Wolff *et al.*, 1997)
			Sum of PCBs	Quartile 2		0.6	*P* = 0.47 (paired t-test)
				Quartile 3		0.8	
				Quartile 4		0.5	
			Group 1B	Quartile 2		0.6	*P* = 0.56 (paired t-test)
				Quartile 3		0.6	
				Quartile 4		0.5	
			Group 2A	Quartile 2		0.8	*P* = 0.50 (paired t-test)
				Quartile 3		0.6	
				Quartile 4		0.6	
			Group 2B	Quartile 2		0.4	*P* = 0.32 (paired t-test)
				Quartile 3		1.0	
				Quartile 4		0.5	
			Group 3	Quartile 2		0.7	
				Quartile 3		0.8	
				Quartile 4		0.6	*P* = 0.18 (paired t-test)
Raaschou-Nielsen *et al.* (2005) Copenhagen and Aarhus, Denmark (DCH cohort) 1993–1997 until 2000	29 875 women; 220–365 pairs, depending on congener	Adipose tissue, GC/MS, lipid-corrected concentrations		Quartiles of PCB concentration (ng/g lipid)			Age, use of HRT (matching), benign breast tumour, BMI, alcohol, parity, age at delivery, years of HRT, lactation Response rate, 37%; all cases were postmenopausal women; LOD, 2.8–28.4 ng/g lipids; 18 PCB congeners[c] measured
			All cases (*n* = 365)	671–852	NR	0.9 (0.6–1.4)	
				852–1.024	NR	0.7 (0.5–1.1)	
				1024–4357	NR	1.1 (0.7–1.7)	
				Continuous (log ng/g lipid)	NR	*P* = 0.44	

Table 2.6 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Raaschou-Nielsen et al. (2005) (cont.)			ER+ (n = 261)	671–852	NR	1.1 (0.6–1.8)	
				852–1.024	NR	0.8–0.5–1.4)	
				1024–4357	NR	1.4 (0.8–2.5)	
				Continuous (log ng/g lipid)	NR	P = 0.50	
			ER– (n = 75)	671–852	NR	0.4 (0.1–1.3)	
				852–1.024	NR	0.3 (0.1–0.9)	
				1024–4357	NR	0.3 (0.1–0.9)	
				Continuous (log ng/g lipid)	NR	P = 0.007	

[a] Congeners measured: 28, 52, 56, 66, 74, 90, 101, 105, 110, 118, 138, 146, 153, 156, 170, 172, 178, 180, 183, 187, 189, 193, 194, 195, 201, 203, 206

[b] Congeners measured: 101, 187, 201, 138, 170, 99, 153, 180, 183, 203

[c] Congeners measured: 28, 52, 54, 99, 101, 104, 105, 118, 128, 138, 153, 155, 156, 170, 180, 183, 187, 201

BMI, body mass index; CHDS, Child Health and Development Studies; DCH, Diet, Cancer, and Health; ECD, electron capture detection; ER, estrogen receptor; FTP, full-term pregnancy; GC, gas chromatography; HRGC, high-resolution gas chromatography; HRT, hormone-replacement therapy; ID-HRMS, isotope dilution high-resolution mass spectrometry; LOD, limit of detection; mo, month; NR, not reported; PCB, polychlorinated biphenyl

could have been present when the blood sample was collected, despite negative mammography findings, and could therefore have affected the measured concentration of PCBs.] Additional cases and controls were included in an extended follow-up of this cohort to 1994, giving totals of 148 cases and 295 controls (Wolff *et al.*, 2000a). In this update, only incident cases were considered (thus excluding those with a lag time of 6 months or less). Serum lipids were measured and PCB concentrations were calculated on a lipid basis. The risk estimates were further adjusted for family history of cancer of the breast, reproductive risk factors, height, and body mass index (BMI). Odds ratios increased across quartiles of serum PCB concentrations, reaching 2.02 (95% CI, 0.76–5.37) in the highest quartile; the trend was not statistically significant. [The Working Group noted that this was a well-designed study; however, the follow-up was relatively short and the analysis thus had limited power.]

Krieger *et al.* (1994) performed a nested case–control study among women in Northern California, USA, who were members of the Kaiser Permanente Medical Care Program and who underwent a health examination, including giving a sample of blood, between 1964 and 1969, and were followed up until 1990. Among the 2072 patients identified with cancer of the breast, 150 cases were randomly selected (50 white, 50 black, and 50 Asian) and matched to 150 controls by race, age, date of entry, and date of follow-up. After adjustment for reproductive factors, menopausal status and BMI, no association was seen between risk of cancer of the breast and serum PCB concentrations for all subjects (OR, 0.93; 95% CI, 0.83–1.05 per ppb). In subgroup analyses by ethnic group, there was an inverse association for white women (OR, 0.21; 95% CI, 0.05–0.88; and OR, 0.17; 95% CI, 0.03–0.89 for the second and third tertiles respectively, *P* for trend = 0.04) and a positive association for black women (OR, 1.74; 95% CI, 0.56–5.43 and OR, 2.13; 95% CI, 0.70–6.50, respectively, *P* for trend = 0.18). [This was a well-designed study with adjustment for relevant confounders, with more than 2000 cases of cancer of the breast identified during the follow-up; however, only 150 were selected for measurement of PCBs and thus power was limited, especially for subgroup analyses.]

The Nurses' Health Study was established in 1976 and included more than 120 000 registered nurses in the USA, who were subsequently followed by questionnaire every 2 years and 32 826 women from the cohort provided a blood sample between 1989 to 1990. Results were reported from follow-ups until 1992 (Hunter *et al.*, 1997) and 1994 (Laden *et al.*, 2001a). In the first follow-up, no association was found between cancer of the breast and PCB concentrations after adjustment for family history of cancer of the breast, reproductive factors, BMI, and cholesterol (Hunter *et al.*, 1997). The extended follow-up to 1994 included 370 case–control pairs, and provided results for individual congeners (Laden *et al.*, 2001a). The pattern of risk by quintile did not change and no association was found for PCB-118, PCB-138, PCB-153, or PCB-180. In subgroup analyses, a significant increase in risk was reported for exposure to the sum of 16 PCBs in nulliparous women (OR, 5.30; 95% CI, 1.06–26.6 for the third tertile of PCB serum concentrations when compared with the first tertile, but the overall trend was not significant; $P = 0.11$). An inverse association was found for women with BMI ≥ 30; the odds ratio for the highest versus lowest tertile was 0.26 (95% CI, 0.09–0.76; *P* for trend = 0.01), while elevated odds ratios were found for women in the highest tertile of PCB exposure with BMI of 25–29.9 and < 25. Since PCB exposure induces activity of cytochrome P450 1A1 (CYP1A1), and PCBs themselves can be metabolized to carcinogenic intermediates by this enzyme, it was explored whether the potential effect of PCBs was modified by the CYP1A1 polymorphism using the same data set (Laden *et al.*, 2002). In 367 case–control pairs, *CYP1A1* exon 7 and *MspI* polymorphisms

were determined. The relative risk increased across tertiles of PCB exposure among those with the variant exon 7 genotype, but not among those with the wild-type genotype. When the analysis was restricted to postmenopausal women, the odds ratio was 2.78 (95% CI, 0.99–7.82) for the highest tertile of PCB exposure, with a *P* value for interaction of 0.05. No gene–environment interaction was seen for *MspI* polymorphism. [The Working Group noted that this was a well-designed study with good controls for most relevant confounders, including reproductive factors and family history of cancer of the breast. The sample size was reasonable when compared with previous studies, and estimates for specific PCB congeners were reported. The only statistically significant associations were limited to specific subgroups after several subgroup analyses and multiple comparisons.]

In another study in the USA, 7224 female volunteers were identified through the Breast Cancer Detection and Demonstration Project (BCDDP) and donated blood to the Columbia Missouri Breast Cancer Serum Bank; active follow-up continued until 1989 (Dorgan *et al.*, 1999). Among these women, 105 were diagnosed with histologically confirmed cancer of the breast, and two controls for each were selected, matched on year of age, date of blood sampling, and history of benign breast disease at the time of enrolment. No association was reported between risk of cancer of the breast and lipid-corrected concentrations of total PCBs (sum of 27 PCB congeners measured), or serum concentrations of PCB-118 and PCB-138, after adjustment for the main risk factors for cancer of the breast. [This study had a relatively small number of cases and was therefore of limited power].

A case–control study was conducted among residents of Washington County, Maryland, USA, who had participated in one of two studies conducted in 1974 and 1989 to obtain blood samples for a serum bank (Helzlsouer *et al.*, 1999). Participants were followed up until 1994 by linkage with the Washington County Cancer Registry. Of the 346 cases of cancer of the breast diagnosed, valid measurements of PCBs were available for 340 cases, which were matched to 340 participating women without cancer of the breast by age, menopausal status and date of blood collection. Taking into account relevant confounders including family history of cancer of the breast, reproductive history and BMI, no association was found with total PCB serum concentration or with specific congeners. There were no statistically significant associations after stratifying for menopausal status, estrogen-receptor (ER) status or polymorphism in *GSTM1*, *GSTT1*, *GSTP1*, *COMT*, or *CYP17*. [The Working Group noted that this study, with an analysis adjusting for most relevant confounders, investigated the hormone-receptor status of tumours, and also considered possible effect modification by polymorphisms in several genes with a role in metabolism. Although the sample size was adequate for the main analysis, it was limited for subgroup analyses.]

A nested case–control study compared serum concentrations of 16 PCBs in archived early-postpartum serum samples collected between 1959 and 1967 from 112 cases of cancer of the breast and 112 age-matched controls (Cohn *et al.* 2012). Subjects were residents of Oakland, California, participating in the Child Health and Development Studies. Cases of cancer of the breast were identified by linkage to the California Cancer Registry, and the California Vital Status Records. The median time from blood draw to diagnosis was 17 years, and mean age of cases at diagnosis was 43 years. No associations were reported between risk of cancer of the breast and sum of total PCBs, or with PCB groups (Wolff *et al.*, 1997). [No odds ratios were reported for these analyses]. PCB-167 was associated with a lower risk (OR for highest versus lowest quartile, 0.2; 95% CI, 0.1–0.8), as was PCB-187 (OR for highest versus lowest quartile, 0.4; 95% CI, 0.1–1.1). In contrast, PCB-203 was associated

with an increased risk (OR for highest versus lowest quartile, 6.3; 95% CI, 1.9–21.7). [This was the only nested case–control study to include mostly premenopausal women. The study had limited power.]

(ii) Northern Europe

Serum samples were obtained in 1976 from a cohort of 7712 women aged 20 years or older who participated in the Copenhagen City Heart Study (Denmark) and provided information and a non-fasting blood sample (Høyer *et al.*, 1998). Case ascertainment was achieved by linkage to the Danish Cancer Registry up to 1993. For each case, two women free of breast cancer and alive at the time of diagnosis and matched for age and date of examination were selected from the rest of the cohort. After excluding subjects without a valid serum sample, 240 cases and 447 controls were included in the study. Concentrations of 28 PCB congeners were measured in serum. No association was reported between risk of cancer of the breast and lipid-adjusted concentrations of the sum of PCBs or specific congeners.

Participants in the same cohort study were invited for a second examination 5 years after recruitment; 155 cases and 274 controls from the previous study had a second serum sample available (Høyer *et al.*, 2000). Analyses were carried out in this group for four common PCB congeners. A statistically significant increased risk and trend was found for subjects in the highest quartile of PCB-138 concentration (average of two measurements; OR, 2.1; 95% CI, 1.0–4.4; *P* for trend = 0.04). Elevated odds ratios were reported for the highest quartile of exposure to total PCBs and congeners PCB-118 and PCB-153 (OR, 1.6, 1.9 and 1.3, respectively), but the association was not significant for these congeners or for PCB-180.

Within the same cohort, a total of 161 cases with ER status information and 318 matched controls who were free of breast cancer were included in an analysis according to ER status (Høyer *et al.*, 2001). No association was found between incidence of cancer of the breast and PCB concentrations regardless of ER status. Finally, paraffin embedded tumour-tissue specimens were retrieved for 162 cases and 316 controls and found to be suitable for *p53* analysis (Høyer *et al.*, 2002). A non-significant increased risk of cancer of the breast (OR, 3.00; 95% CI, 0.66–13.62) was observed in the highest level of exposure to PCBs among women with mutant *p53*. [Several analyses were carried out using data from this Danish study, but power was limited, particularly for subgroups.]

The JANUS Serum Bank contains serum samples collected between 1973 and 1991 from almost 300 000 individuals undergoing routine health examinations in Norway. Cases of cancer of the breast were identified among 25 431 women working outside home and resident on a farm who were followed until 1993 through linkage with the Norwegian Cancer Registry (Ward *et al.*, 2000). From the 272 cases diagnosed during this period, 150 women with a blood sample taken 2 or more years before diagnosis were randomly selected; an equal number of controls were matched to cases by date of sample collection and date of birth. The mean lipid-corrected concentration of serum PCBs (sum of 36 congeners) was similar for cases and controls (*P* value, 0.47 for paired *t*-test). No association was found for specific PCB congeners or for PCB groups as defined by Wolff *et al.* (1997). [The Working Group noted that this study was well designed and considered most relevant confounders for cancer of the breast but, similar to other nested case–control studies with serum PCB measurements, had limited power.]

Between 1993 and 1997, 29 875 Danish women aged 50 to 64 years were enrolled in a prospective study of diet and cancer and followed until December 2000 through linkage with Danish Cancer Registry (Raaschou-Nielsen *et al.*, 2005). During this period, 409 women were diagnosed with postmenopausal cancer of the breast; each case was matched to one control by age,

postmenopausal status (known/probable), and use of hormone replacement therapy, and measurements of 18 PCBs in adipose-tissue biopsies were obtained. No association was found between concentrations of PCBs and risk of cancer of the breast in the whole data set. However, an inverse association was observed when the analysis was restricted to the 75 ER-negative (ER–) cases (OR, 0.3; 95% CI, 0.1–0.9). This inverse association for ER– cases was also observed for the congeners PCB-138, PCB-153, PCB-170, PCB-180, PCB-183 and PCB-187. [The Working Group noted that this was the largest nested case–control study of cancer of the breast with PCB measurements, and the only one to measure PCBs in adipose tissue rather than serum. The inverse association of concentrations of total PCB and some PCB congeners among women with ER– tumours does not have a clear interpretation.]

(b) NHL

See Table 2.7

(i) USA

Seventy-four cases of NHL (ICD-8 200 or 202) identified during follow-up from 1975 to 1994 of the cohort from Washington County, Maryland, USA (described in the previous section) and 147 controls matched by race, sex, and age were included in a case–control study (Rothman *et al.*, 1997). PCB concentrations were measured in serum collected before diagnosis and corrected for lipids. There was a significant dose–response relationship between risk of NHL and quartiles of lipid-corrected serum concentrations of PCBs (sum of 28 measured congeners). The odds ratios for the third and fourth quartiles when compared with the first quartile were 2.8 (95% CI, 1.1–7.6) and 4.5 (95% CI, 1.7–12.0) respectively; these estimates were adjusted, in addition to matching variables, for education, cigarette smoking and occupational exposure to suspected risk factors for NHL. There was also an indication that seropositivity for the Epstein-Barr virus early antigen (EBV-EA) potentiated the effects of serum PCBs, with a statistically significant interaction (*P* value = 0.025).

An analysis of the same data set focusing on the effect of specific congeners reported a significant exposure–response relationship between risk of NHL and increasing concentrations of PCB-118, PCB-138, and PCB-153 (*P* for trend < 0.05) (Engel *et al.*, 2007). [The Working Group noted that this was the only nested case–control study on PCB concentrations and NHL that adjusted for occupational exposure to potential risk factors.]

An analysis of the association between NHL and exposure to PCBs conducted within the Nurses' Health Study cohort (described in the previous section) was reported in the same publication (Engel *et al.*, 2007). Thirty women with incident NHL diagnosed between the date of blood collection and May 1994 (median follow-up, 1 year) were included as cases and 78 cohort members selected previously as controls for a study of cancer of the breast served as controls. Plasma samples were analysed for PCB concentrations for cases and for controls at the same time. A statistically significant exposure–response relationship was observed between risk of NHL and increasing concentrations of lipid-corrected PCBs (sum of 21 congeners), with an odds ratio of 4.7 (95% CI, 1.2–18.9) for the third tertile, adjusted for age, BMI, and smoking status. A significant exposure–response relationship was also observed for PCB-118 and PCB-138 (*P* for trend < 0.05), but not for PCB-153.

An extended follow-up of the Nurses' Health Study cohort (median time to diagnosis, 5.8 years) included 145 cases of NHL and selected two controls for each case (n = 290) matched on age, race, month of blood draw, and fasting status (Laden *et al.*, 2010). Women with NHL were identified by annual follow-up questionnaires and confirmed by review of medical records and pathology reports. No association was observed between total serum concentrations of PCBs

Table 2.7 Nested case-control studies on risk of non-Hodgkin lymphoma and measured serum or adipose concentrations of PCBs

Reference, location follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
USA							
Rothman *et al.* (1997) Maryland, USA 1972–1990 until 1994	25 802 adults; 74 cases, 147 controls	Serum, GC/ECD, lipid-corrected concentrations		Quartiles of PCB concentration (ng/g lipid)			Race, sex, age (matching), education, cigarette smoking, potential for occupational exposure
			Sum of PCBs	648–806	13	1.3 (0.5–3.3)	28 congeners measured[a]
				814–1060	21	2.7 (0.9–7.8)	
				1070–2070	30	4.1 (1.7–11.9)	*P* for trend = 0.0008
Engel *et al.* (2007) Maryland, USA				Median of quartiles of PCB concentration (ng/g lipid)			Same population studied by Rothman *et al.* (1997) and Helzlsouer *et al.* (1999)
			Total PCBs	726.0	13	1.6 (0.6–4.3)	
				911.5	21	3.0 (1.1–8.3)	
				1337.5	30	4.6 (1.7–12.7)	
			PCB-118	124.6	23	4.9 (1.6–15.3)	
				164.9	17	3.5 (1.0–11.8)	
				214.7	29	5.4 (1.7–17.1)	*P* for trend < 0.05
			PCB-138	129.1	20	2.5 (0.9–6.5)	
				164.5	19	2.7 (1.0–7.5)	
				242.4	27	4.4 (1.5–12.6)	*P* for trend < 0.05
			PCB-153	122.4	14	1.0 (0.4–2.3)	
				163.2	17	1.4 (0.5–3.5)	
				246.9	27	2.2 (0.9–5.2)	*P* for trend < 0.05

Table 2.7 (continued)

Reference, location follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Laden *et al.* (2010) 11 states, USA (Nurses' Health Study cohort)	145 cases and 290 controls	Serum, GC/ECD, lipid-corrected concentrations		Median of quartiles of PCB concentration (ng/g lipid)			Race, age, date of and fasting status at blood draw (matching), region, BMI, smoking, height, parity, breastfeeding 51 congeners measured[a]
			Total PCB	547.8	41/73	1.25 (0.68–2.28)	Continuous (log-concentration) $P = 0.76$
				678.0	41/73	1.32 (0.71–2.43)	
				945.4	30/72	1.02 (0.53–1.95)	
			PCB-118	42.9	49	1.39 (0.78–2.47)	Continuous (log-concentration) $P = 0.42$
				61.0	31	0.89 (0.48–1.64)	
				104.7	27	0.81 (0.42–1.56)	
			PCB-138	53.2	39	1.33 (0.73–2.40)	Continuous (log-concentration) $P = 0.59$
				75.7	48	1.61 (0.89–2.92)	
				113.3	27	0.95 (0.49–1.83)	
			PCB-153	91.2	33	0.85 (0.47–1.54)	Continuous (log-concentration) $P = 0.55$
				120.3	45	1.38 (0.76–2.51)	
				170.0	30	0.82 (0.43–1.56)	
			PCB-180	63.4	33	1.02 (0.54–1.93)	Continuous (log-concentration) $P = 0.82$
				80.5	44	1.24 (0.66–2.31)	
				109.4	32	1.03 (0.52–2.02)	
			Immunotoxic congeners[b]	111.5	56	1.83 (1.01–3.31)	Continuous (log-concentration) $P = 0.48$
				149.6	30	0.94 (0.51–1.76)	
				228.7	25	0.89 (0.45–1.77)	

Table 2.7 (continued)

Reference, location follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Bertrand *et al.* (2010) USA (Physicians' Health Study cohort) 1982–2003	14 916 men; 205 cases and 409 controls	Serum, GC/ECD, lipid-corrected concentrations		Quintiles of PCB concentration (ng/g lipid)			Age, race, time and fasting status at blood draw (matching), region, height, BMI, alcohol, smoking 51 congeners measured[c]
			Total PCB	163–617	33	1.0	Continuous (log-concentration) $P < 0.01$
				> 617–742	31	0.86 (0.47–1.6)	
				> 742–894	34	0.99 (0.55–1.8)	
				> 894–1121	46	1.3 (0.71–2.3)	
				> 1121–5322	61	1.6 (0.91–2.9)	
			PCB-118	> 42–56	29	0.80 (0.42–1.5)	Continuous (log-concentration) $P = 0.15$
				> 56–77	40	1.1 (0.59–2.0)	
				> 77–105	46	1.2 (0.63–2.2)	
				> 105–734	57	1.4 (0.76–2.5)	
			PCB-138	> 59–76	38	1.3 (0.68–2.3)	Continuous (log-concentration) $P = 0.02$
				> 76–97	38	1.2 (0.64–2.1)	
				> 97–122	37	1.2 (0.64–2.2)	
				> 122–541	63	1.8 (0.98–3.2)	
			PCB-153	> 95–122	37	1.2 (0.67–2.3)	Continuous (log-concentration) $P < 0.01$
				> 121–148	36	1.3 (0.68–2.4)	
				> 148–188	37	1.2 (0.62–2.2)	
				> 188–761	67	2.1 (1.1–3.8)	
			PCB-180	> 68–84	40	1.5 (0.82–2.7)	Continuous (log-concentration) $P < 0.01$
				> 84–102	35	1.4 (0.75–2.7)	
				> 102–126	44	1.8 (0.96–3.3)	
				> 126–528	61	2.4 (1.3–4.5)	
			Immunotoxic congeners[b]	> 113–145	35	0.98 (0.54–1.8)	Continuous (log-concentration) $P = 0.09$
				> 145–189	36	0.99 (0.55–1.8)	
				> 189–245	45	1.2 (0.64–2.1)	
				> 245–1813	57	1.4 (0.80–2.6)	

Table 2.7 (continued)

Reference, location follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Northern Europe (Norway, Denmark)							
Engel *et al.* (2007) Norway (JANUS cohort) 1972–1978 until 1999	87 600; 190 case–control pairs	Serum, HRGC/ID-HRMS, lipid-corrected concentrations		Median of quartiles of PCB concentration (ng/g lipid)			Age, sex, county, date of examination (matching), BMI, smoking status All cases ≥ 2 years from blood collection to diagnosis; 36 congeners measured[d]
			Total PCB	1398.3	48	1.1 (0.7–2.0)	
				1674.9	38	1.0 (0.5–1.9)	
				2148.2	60	1.7 (0.8–3.4)	*P* for trend < 0.05
			PCB-118	80.6	43	1.0 (0.5–2.0)	
				100.0	47	1.2 (0.6–2.3)	
				138.7	58	1.7 (0.9–3.5)	*P* for trend < 0.05
			PCB-138	122.8	29	0.6 (0.3–1.2)	
				153.4	42	0.9 (0.5–1.7)	
				190.0	68	1.7 (0.8–3.2)	*P* for trend < 0.05
			PCB-153	268.1	44	1.2 (0.6–2.3)	
				330.2	43	1.2 (0.7–2.2)	
				417.3	63	2.0 (1.0–3.9)	*P* for trend < 0.05
Bräuner *et al.* (2012) Copenhagen and Aarhus, Denmark (DCH cohort) 1994–97 until 2008	57 053; 239 cases and 245 controls	Adipose tissue, GC/MS, lipid-corrected concentrations		Quintiles of PCB concentration (ng/g lipid)		*IRR (95% CI)*	Age, sex (stratified), adjusted for BMI Lipid content by gravimetric method; 10 PCB congeners measured.[e] Participants with PCB concentrations < LOD were excluded from the analysis Case-content analysis
			Total PCB	770–939	55	0.74 (0.44–1.24)	
				939–1143	57	0.81 (0.48–1.35)	
				1143–1351	42	1.15 (0.63–2.11)	
				1351–2157	23	0.71 (0.34–1.45)	
				Linear estimate per IQR	239	0.99 (0.79–1.25)	
			PCB-118	25–34	63	0.88 (0.50–1.56)	
				34–48	58	0.96 (0.55–1.65)	
				48–62	34	0.67 (0.34–1.31)	
				62–150	25	0.72 (0.36–1.44)	
				Linear estimate per IQR	233	0.88 (0.68–1.14	

Table 2.7 (continued)

Reference, location follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Bräuner et al. (2012) Copenhagen and Aarhus, Denmark (DCH cohort) 1994–97 until 2008 (cont.)			PCB-156	28–34	51	0.59 (0.34–1.02)	
				34–41	54	0.68 (0.40–1.16)	
				41–50	45	0.94 (0.51–1.75)	
				50–88	23	0.66 (0.31–1.37)	
				Linear estimate per IQR	171	1.01 (0.79–1.29)	
			PCB-99	20–27	42	1.60 (0.85–3.01)	
				27–37	53	1.56 (0.84–2.89)	
				37–47	24	1.20 (0.58–2.49)	
				47–110	20	1.42 (0.59–3.40)	
				Linear estimate per IQR	171	1.09 (0.83–1.43)	
			PCB-138	100–140	44	0.66 (0.38–1.14)	
				140–180	74	1.04 (0.62–1.74)	
				180–230	41	1.25 (0.67–2.33)	
				230–380	26	0.68 (0.34–1.36)	
				Linear estimate per IQR	238	0.99 (0.78–1.26)	
			PCB-153	240–300	57	0.88 (0.52–1.50)	
				300–370	56	0.67 (0.40–1.12)	
				370–430	42	1.50 (0.81–2.78)	
				430–730	28	0.85 (0.42–1.73)	
				Linear estimate per IQR	239	0.97 (0.77–1.23)	
			PCB-170	87–100	47	1.19 (0.68–2.09)	
				100–130	69	0.93 (0.54–1.59)	
				130–150	42	1.46 (0.75–2.83)	
				150–230	23	0.80 (0.38–1.69)	
				Linear estimate per IQR	238	0.98 (0.72–1.33)	
			PCB-180	170–200	55	1.03 (0.60–1.77)	
				200–240	61	1.19 (0.69–2.05)	
				240–290	49	1.09 (0.59–2.01)	
				290–480	21	0.69 (0.32–1.46)	
				Linear estimate per IQR	239	0.99 (0.77–1.27)	

Table 2.7 (continued)

Reference, location follow-up period	Total subjects	Exposure assessment	Subgroup analysis	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Bräuner et al. (2012) Copenhagen and Aarhus, Denmark (DCH cohort) 1994–97 until 2008 (cont.)			PCB-183	19–24	35	0.58 (0.32–1.03)	
				24–31	69	0.91 (0.54–1.51)	
				31–39	40	1.03 (0.56–1.90)	
				39–65	23	0.68 (0.34–1.37)	
				Linear estimate per IQR	226	0.88 (0.70–1.10)	
			PCB-187	17–46	61	1.00	
				46–56	49	0.69 (0.40–1.17)	
				56–68	62	0.97 (0.57–1.64)	
				68–84	44	1.30 (0.68–2.47)	
				84–140	22	0.69 (0.33–1.44)	
				Linear estimate per IQR	238	0.92 (0.73–1.15)	
			PCB-201	6–15	43	1.00	
				15–19	62	0.98 (0.56–1.73)	
				19–23	58	1.20 (0.66–2.21)	
				23–28	36	0.82 (0.41–1.67)	
				28–45	25	0.88 (0.38–2.03)	
				Linear estimate per IQR	224	0.93 (0.68–1.28)	

[a] Congeners measured: PCBs 28, 52, 56, 74, 99, 101, 105, 110, 118, 138, 146, 153, 156, 170, 172, 177, 178, 180, 183, 187, 189, 193, 194, 195, 201, 203, and 206

[b] Immunotoxic congeners: PCB-66, PCB-74, PCB-105, PCB-118, PCB-156, and PCB-167

[c] Ninety-nine percent of samples had concentrations greater than the limit of detection for PCB congeners 74, 118, 138, 146, 153, 156, 170, 180, 187, 194, 196, 199, 203, 206, and 209

[d] Congeners measured: PCBs 126, 169, 74, 99, 118, 105, 146, 153, 138, 158, 167, 156, 157, 178, 187, 183, 177, 172, 180, 170, 189, 201, 196, 203, 195, 194, 206, 209; 26 with > 90% samples having concentrations greater than the limit of detection

[e] Congeners measured: PCBs 99, 118, 138, 153, 156, 170, 180, 183, 187, and 201. LOD, 0.10–1.00 ng/g lipid; proportion of subjects with values greater than the limit of detection ranged from 72% (PCB-99) to 100% (PCB-153 and PCB-180)

BMI, body mass index; ECD, electron capture detection; ER, estrogen receptor; FTP, full-term pregnancy; GC, gas chromatography; HRGC, high-resolution gas chromatography; ID-HRMS, isotope dilution high-resolution mass spectrometry; IRR, incidence rate ratio; IQR, interquartile range; LOD, limit of detection; mo, month; NHL, non-Hodgkin lymphoma; NR, not reported; OR, odds ratio; PCB, polychlorinated biphenyl

(sum of 51 congeners measured as lipid-corrected concentrations) or for specific congeners (PCB-118, PCB-138, PCB-153, PCB-180) after adjustment for several confounders. The same pattern of no association was observed in the subgroup analysis by the main subtypes of NHL, diffuse large B-cell lymphoma (DLBCL), follicular lymphoma, and chronic lymphocytic leukaemia/small lymphocytic lymphoma. [This was the only nested case–control study on non-Hodgkin lymphoma to include women only. The Working Group noted that it was a well-designed study. The positive association observed in the initial study was not confirmed in the second, larger study, after adjustment for additional relevant confounders. However, in the second study the time since blood draw was prolonged and different laboratories and laboratory methods were used for analysis.]

The Physicians' Health Study began in 1982 in the USA as a randomized trial for the primary prevention of cardiovascular disease and cancer in male physicians aged 40–84 years at enrolment. A total of 14 916 participants provided a blood sample in 1982–84 (before randomization) and were followed until 2003 using annual questionnaires confirmed by review of medical records to identify newly diagnosed NHL (Bertrand *et al.*, 2010). After excluding those with a diagnosis within 6 months after blood collection, prior diagnosis of cancer, NHL of uncommon subtypes (i.e. mantle cell lymphoma), or lacking sufficient information for subtype classification, 205 cases with available blood samples were included. For each case, two subjects who were at risk of NHL when the case occurred were randomly selected as controls matched by race, age, and date of blood collection. Lipid-corrected concentrations of 51 PCB congeners in serum were determined for cases and controls. The odds ratio for the highest versus lowest quintile of total PCBs adjusted for matching variables was 1.9 (95% CI, 1.1–3.2), which was reduced to 1.6 (95% CI, 0.91–2.9) after adjustment for region, BMI, smoking status, alcohol intake, and height, in addition to matching variables. However, using the natural log of lipid-corrected concentrations of PCBs, the association was statistically significant for the fully adjusted model (P value < 0.01, OR not reported). The association was also significant for the log-concentrations of PCB-138, PCB-153 and PCB-180, as well as for the sum of PCBs -118, -138, -153 and -180. [The Working Group noted that this was a well-designed study with reasonable sample size. The multivariable adjustment weakened the association with total PCBs, but did not substantially change the interpretation.]

(ii) Northern Europe

Within the JANUS cohort, described in the previous section, 194 histologically confirmed cases of NHL were ascertained with follow-up to 1999 (median time to diagnosis, 16.6 years) (Engel *et al.*, 2007). Information, including lipid-corrected concentrations of 36 PCB congeners, was available for 190 case–control pairs matched by age, sex, county, and date of examination. In the analysis further adjustments were made for BMI and smoking status. The odds ratio for the association of NHL with the sum of PCBs was 1.7 (95% CI, 0.8–3.4) when comparing the fourth quartile with the first. A statistically significant increase in risk was reported for the highest to the lowest quartile of PCB-153 concentrations (OR, 2.0; 95% CI, 1.0–3.9), with a significant upward dose–response trend (P < 0.05). Odds ratios of 1.7 in the fourth exposure quartile and significant trends were also reported for PCB-118 and PCB-138. [The Working Group noted that the sample size, and therefore the power of the study, was in the range of that of the remaining nested case–control studies. It was not clear, therefore, why significant associations were found for three congeners, namely PCB-118, PCB-138, and PCB-153, but not for all PCBs combined.]

The association between NHL and PCB concentrations in adipose tissue was also studied among participants in the Danish diet and cancer

study (Raaschou-Nielsen *et al.*, 2005) described in section 2.2.3(a)(ii) (Bräuner *et al.*, 2012). Up to July 2008 (mean follow-up, 9.6 years), 278 initially cancer free cohort members were diagnosed with NHL; a subcohort of 256 participants was randomly selected for analysis using a case–cohort approach. Valid measurements of concentrations of 10 PCB congeners in adipose tissue were available for 239 cases and 245 subcohort members. Age was used as the timescale for the analysis, stratified by sex and adjusted for BMI. No association was observed between lipid-corrected concentrations of total PCBs in adipose tissue and risk of NHL. There was also no consistent association and no significant trend with PCB congeners. However, odds ratios were greater than 1 for all concentrations of PCB-99. [The Working Group noted that this was the largest nested case–control study on NHL and PCB concentrations measured in adipose tissue; estimates were adjusted only for age, sex, and BMI. The study explored the potential effect of all PCBs and a list of 10 specific congeners, with a consistent pattern of no association for all of them.]

(c) Cancer of the male genital tract

See Table 2.8

A nested case–control study on the risk of testicular germ cell tumours was carried out within the Norwegian JANUS cohort, described in Section 2.2.3(a)(ii) (Purdue *et al.*, 2009). Cases and controls were selected from cohort members with baseline blood collection without prior history of cancer. One male control was matched to each case by region, age group (2 years), and year of blood draw. Lipid-corrected measurements of the concentrations of 34 PCBs were available for 49 cases and 51 controls; 34 of the 49 cases were seminomas, 8 were non-seminomas, 5 were of mixed histology, and 2 were of unknown histology. There was no statistically significant association between risk of testicular germ cell tumours and total PCB concentration (OR, 1.3; 95% CI, 0.5–3.8 for the third versus the first quartile); however, there was an increased risk of testicular germ cell tumours for the highest versus the lowest tertile of PCB-99 concentration (OR, 2.2; 95% CI, 0.8–5.9) and of PCB-167 (OR, 4.4; 95% CI, 1.0–19.8). Cases of seminoma had significantly lower concentrations of congeners PCB-44, PCB-49, and PCB-52 and significantly higher concentrations of congeners PCB-99, PCB-138, PCB-153, PCB-167, PCB-183, and PCB-195. Similar patterns of elevated odds ratios were seen for PCB-99 and PCB-167 in this subgroup of cases. [The Working Group noted that this was a well-designed study, but with small sample size and very limited power.]

McGlynn *et al.* (2009) analysed concentrations of 15 PCBs in pre-diagnostic serum samples of 736 incident cases of testicular germ cell tumours and 913 controls matched to the cases on age, race, and serum draw date in a cohort of men in the United States military. The sum of PCB concentrations was significantly associated with decreased risk of all testicular germ cell tumours, and with non-seminoma and seminoma. Statistically significantly decreased risks of all testicular germ cell tumours were also associated with eight specific congeners (PCB-118, PCB-138, PCB-153, PCB-156, PCB-163, PCB-170, PCB-180, and PCB-187). Similar decreases in risk were observed for non-seminoma with the same congeners, while decreased risk of seminoma was associated with PCB-138, PCB-153, PCB-156, PCB-163, and PCB-170. Other congeners and groups of congeners were not associated with testicular germ cell tumours. In another study using data from 568 cases and 698 controls enrolled in the same cohort, Chia *et al.* (2010) examined associations between testicular germ cell tumours and 11 PCB congeners in relation to polymorphisms in hormone-metabolizing genes. A statistically significant reduced risk of testicular germ cell tumour for PCB-118 and PCB-138 was found only among subjects with the major homozygous allele for *HSD17B4*. [These appear

Table 2.8 Nested case-control studies on risk of cancer of the male genital tract and measured serum concentrations of PCBs

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Purdue *et al.* (2009) Norway (JANUS cohort) 1972–1978 until 1999	87 647 men; 49 cases and 51 controls	Serum, HRGC/ID-HRMS, lipid-corrected concentrations	TGC tumour (186)	Tertiles of PCB concentration			Age, county, period of blood draw (matching) 34 congeners measured
				Tertile 1	14	1.0	
				Tertile 2	16	1.1 (0.5–2.7)	
				Tertile 3	19	1.3 (0.5–3.8)	
				Selected PCB congeners: tertile 3, tertile 1 as referent:			
				PCB-44	18	0.6 (0.1–3.8)	
				PCB-49	20	1.2 (0.2–7.6)	
				PCB-52	20	1.0 (0.3–3.5)	
				PCB-99	21	2.2 (0.8–5.9)	
				PCB-138	24	1.8 (0.6–5.1)	
				PCB-153	19	1.2 (0.4–3.4)	
				PCB-167	19	4.4 (1.0–20.0)	
				PCB-183	18	1.3 (0.5–3.5)	
				PCB-195	15	1.7 (0.6–4.6)	
			Seminoma (n = 34)	Selected PCB congeners: tertile 3, tertile 1 as referent			
				PCB-44	12	0.2 (0.01–2.0)	
				PCB-49	14	0.3 (0.02–4.7)	
				PCB-52	14	0.4 (0.07–2.3)	
				PCB-99	17	4.4 (1.0–21)	
				PCB-138	17	2.1 (0.6–7.2)	
				PCB-153	13	1.2 (0.4–4.3)	
				PCB-167	15	6.7 (1.1–43)	
				PCB-183	14	2.9 (0.6–14)	
				PCB-195	13	3.0 (0.8–12)	
				Total PCBs	14	1.2 (0.4–4.1)	

Table 2.8 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
McGlynn *et al.* (2009), USA (STEED Study) 2002–2005	Military men [number not reported]; 736 cases and 913 controls	Blood, GC-MS lipid-adjusted concentrations; questionnaire	TGC tumours	Quartiles of PCB concentration (ng/g lipid)			Age, race/ethnicity, date of serum sample collection, serum DDE level, age at serum draw, BMI, height Quartile 1 as reference
				Total PCBs			
			TGC tumours	(158–250)	171	0.88 (0.67–1.16)	*P* for trend = 0.006
				(251–390)	175	0.73 (0.54 –0.98)	
				(> 390)	162	0.61 (0.43–0.86)	
			Seminoma	(158–250)	60	0.90 (0.6–1.35)	*P* for trend = 0.05
				(251–390)	91	0.89 (0.59–1.34)	
				(> 390)	88	0.64 (0.41–1.02)	
			Non-seminoma	(158–250)	111	0.84 (0.61–1.15)	*P* for trend = 0.007
				(251–390)	84	0.62 (0.43–0.88)	
				(> 390)	73	0.55 (0.37–0.83)	
				PCB-118			
			All TGC tumours	(7.2–10.5)	171	0.71 (0.53–0.94)	
				(10.6–15.6)	151	0.60 (0.45–0.81)	
				(> 15.6)	148	0.55 (0.40–0.76)	*P* for trend = 0.0007
				PCB-138			
				(15.6–24.5)	168	0.65 (0.48–0.88)	
				(24.6–37.7)	162	0.54 (0.39–0.75)	
				(> 37.7)	164	0.46 (0.32–0.66)	*P* for trend = 0.0001
				PCB-153			
				(23.4–37.2)	158	0.61 (0.45–0.82)	
				(37.3–56.3)	166	0.53 (0.38–0.73)	
				(> 56.3)	169	0.45 (0.31–0.66)	*P* for trend = 0.0003
				PCB-156			
				(5.3–6.9)	98	0.66 (0.48–0.90)	
				(7.0–10.0)	120	0.77 (0.56–1.06)	
				(> 10.0)	96	0.57 (0.40–0.81)	*P* for trend = 0.002

Table 2.8 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
McGlynn et al. (2009), USA (STEED Study) 2002–2005 (cont.)				*PCB-163*			
				(5.9–8.1)	128	0.70 (0.52–0.93)	
				(8.2–11.5)	110	0.55 (0.40–0.76)	
				(> 115)	131	0.59 (0.42–0.83)	*P* for trend = 0.001
				PCB-170			
				(6.5–9.7)	145	0.73 (0.55–0.98)	
				(9.8–14.5)	136	0.61 (0.44–0.84)	
				(> 14.5)	144	0.56 (0.39–0.80)	*P* for trend = 0.002
				PCB-180			
				(15.8–25.9)	177	0.83 (0.62–1.12)	
				(26.0–41.8)	176	0.68 (0.49–0.95)	
				(> 41.8)	161	0.56 (0.38–0.82)	*P* for trend = 0.003
				PCB-187			
				(5.8–8.0)	133	0.70 (0.52–0.94)	
				(8.1–11.6)	120	0.58 (0.42–0.81)	
				(> 11.6)	133	0.60 (0.42–0.86)	*P* for trend = 0.004
Chia *et al.* (2010) USA 2002–2005	568 cases and 698 controls	Blood, GC-MS lipid-adjusted concentrations; questionnaire	TGC tumours (186)	*PCB-118*			Age, race, date of serum sample, cryptorchidism, family history of testicular cancer, BMI
				AA genotype			Same cohort studied by McGlynn *et al.* (2009)
				(7.01–10.40)	100	0.66 (0.46–0.96)	AA genotype: AA-homozygous major allele *HSD17B4;* AA/TT genotype: minor allele for *HSD17B4*
							Quartile 1 as reference
				(10.41–15.56)	92	0.59 (0.40–0.87)	
				(> 15.57)	74	0.46 (0.31–0.70)	*P* for trend ≤ 0.001
				AT/TT genotype			
				(7.01–10.40)	38	1.27 (0.66–2.41)	
				(10.41–15.56)	31	1.06 (0.54–2.08)	
				(> 15.57)	43	1.69 (0.85–3.38)	*P* for trend = 0.019

Table 2.8 (continued)

Reference, location, follow-up period	Total subjects	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Chia et al. (2010) USA 2002–2005 (cont.)				*PCB-138*			
				AA genotype			
				(15.85–25.00)	95	0.72 (0.49–1.07)	
				(25.01–38.53)	96	0.57 (0.38–0.85)	
				(> 38.53)	79	0.46 (0.30–0.72)	*P* for trend < 0.001
				AA/TT genotype			
				(15.85–25.00)	27	0.61 (0.31–1.20)	
				(25.01–38.53)	36	1.10 (0.54–2.25)	
				(> 38.53)	43	1.61 (0.76–3.41)	*P* for trend = 0.287
Sawada *et al.* (2010) 10 areas of Japan 1990–1995 until 2005	14 203 men; 201 cases and 402 controls	Serum, HRGC/ID-HRMS; lipid-corrected concentrations	Prostate	Quartiles of PCB concentration (ng/g lipid)			Age, area, date, and fasting hours at blood draw (matching), BMI, smoking, alcohol, marital status, intake of green tea and miso soup Sum of 41 congeners; LOD, 2 pg/g wet weight
				319–447	49	1.06 (0.63–1.79)	
				448–668	41	0.84 (0.49–1.46)	
				≥ 669	44	0.97 (0.51–1.87)	
						P for trend = 0.9	

BMI, body mass index; DDE, dichlorodiphenyldichloroethylene; ECD, electron capture detection; GC, gas chromatography; HRGC, high-resolution gas chromatography; ID-HRMS, isotope dilution high-resolution mass spectrometry; IRR, incidence rate ratio; LOD, limit of detection; NR, not reported; OR, odds ratio; PCB, polychlorinated biphenyl; STEED, US Servicemen's Testicular Tumor Environmental and Endocrine Determinants Study; TGC, testicular germ cell

to have been large, well-designed and well-implemented studies, but the consistent inverse associations of cancer risk with exposure to PCBs could not be explained biologically.]

The Japan Public Health Center-based Prospective Study was initiated in 1990. After excluding subjects from Tokyo for whom cancer information was not available, the cohort consisted of 65 657 men, of whom 14 203 (28%) donated blood between 1990 and 1995 (Sawada *et al.*, 2010). Up to December 2005, 201 newly diagnosed cases of cancer of the prostate were identified using several information sources (97% pathologically confirmed). For each case, two controls were selected from among subjects with no history of cancer of the prostate when the case was diagnosed, matched by age (within 3 years), public health-centre area, residence, date and time of day of blood collection, and duration of fasting. Lipid-corrected plasma concentrations of 41 PCB congeners were measured. Apart from matching variables, comparisons between cases and controls were further adjusted for BMI, smoking, alcohol, marital status, and intakes of green tea and miso soup. No statistically significant association with all cancers of the prostate was seen for total PCBs, for individual PCBs, or for PCBs grouped according to Wolff *et al.* (1997). No statistically significant differences were found for total PCBs according to stage (localized or advanced) at diagnosis of cancer of the prostate. [The Working Group noted that this was a well-designed and -conducted study showing null results; although the sample size was limited, power was reasonable for the main analysis, but limited for subgroup analyses.]

2.3 Case–control studies of occupational and environmental exposure

2.3.1 NHL

See Table 2.9

In a case–control study in Australia (Fritschi *et al.*, 2005), including 694 histologically confirmed cases of NHL, and 694 controls, exposure to PCBs was coded by an expert industrial hygienist based on questionnaire information. After adjusting by age, sex, residence and ethnicity, ever exposure to PCBs was not notably related to increased risk of NHL (OR, 1.10; 95% CI, 0.49–2.44) or to the subgroup of B-cell NHL (OR, 1.18; 95% CI, 0.53–2.62); however, risk was elevated among the subjects probably exposed (OR, 4.54; 95% CI, 0.97–21). Indicators of frequency, intensity, and duration of exposure did not show clear trends in risk. Occupational exposure to PCBs was very rare in this study, with only 25 subjects (13 cases and 12 controls) possibly or probably exposed. [The Working Group noted that this general-population case–control study may have been underpowered to detect associations with PCBs, given the low prevalence of exposure.]

A case–control study was conducted in an area of northern Italy where environmental exposure had resulted from soil contamination, most likely generated by spills from an adjacent factory producing PCBs and organochlorine chemicals. PCB concentration in the soil was used to define four areas with increasing concentrations of exposure. Overall, 495 cases of NHL, including 208 prevalent cases and 287 incident cases, identified in the Cancer Registry of the Brescia Local Health Authority, and 1467 population controls, randomly selected from the resident population, frequency-matched to cases by age and sex, participated in the study. Exposure to PCBs was assigned according to residence in one of three contaminated zones or a control zone, using three metrics: main lifetime residence; residence for at least 10 years in a given area; and duration of residence. Risk of NHL was elevated for subjects having resided 10 or more years in any of the three contaminated areas (OR, 1.4; 95% CI, 1.1–1.8), and particularly in the most polluted (OR, 1.9; 95% CI, 0.9–3.9).

Table 2.9 Case–control studies on risk of non-Hodgkin lymphoma and exposure to PCBs

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Fritschi *et al.* (2005), Australia 2000–01	694 694	Population	NHL (200, 202)	Retrospective expert assessment of occupational exposure to PCBs	Unexposed	681	1.0	Age, sex, state of residence, ethnicity
					Any exposure	13	1.10 (0.12–1.31)	
					Possible exposure	NR	0.40 (0.12–1.31)	
					Probable exposure	NR	4.54 (0.97–21)	
					Low intensity level	NR	1.91 (0.75–4.85)	
					Medium intensity level	NR	0.78 (0.17–3.50)	
					Intensity level			
					≤ 4 days/yr	NR	1.44 (0.49–4.22)	
					> 4 days/yr	NR	1.15 (0.35–3.81)	
					< 5 yr duration	NR	1.04 (0.26–4.19)	
					> 5 yr duration	NR	1.13 (0.43–2.97)	
Maifredi *et al.* (2011), Italy	495 1467	Population	NHL (200, 202)	Residence in PCB contaminated areas in Brescia, Italy; median total PCB soil concentration, 0.55 mg/kg	*Residence 1–9* yr			Age, sex Subjects who changed area of residence were repeatedly considered in each area; substantial overlapping in contamination among the areas; incident and deceased cases were included
					Most polluted area	13	1.4 (0.7–2.8)	
					All contaminated areas	21	0.8 (0.5–1.3)	
					Residence ≥ 10 yr			
					Most polluted area	15	1.8 (0.9–3.9)	
					All contaminated areas	80	1.4 (1.1–1.8)	
					Residence 10–19 yr			
					Most polluted area	10	3.8 (1.5–9.8)	
					All contaminated areas	25	1.7 (1.0–2.8)	
					Residence ≥ 20 yr			
					Most polluted area	5	0.8 (0.3–2.3)	
					All contaminated areas	55	1.3 (0.9–1.8)	
Hardell *et al.* (1996, 1997), Sweden	27 17	Hospital	NHL, B-cell type	Total PCBs in adipose tissue	≤ 1300 ng/g lipid		1.0	Age, sex
					> 1300 ng/g lipid		1.8 (0.4–7.4)	

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Hardell *et al.* (2001), Sweden	82 83	Hospital	NHL (200, 202)	PCBs in adipose tissue or lipid-adjusted serum				Age, sex, BMI, sample (blood or adipose tissue)
				Total PCBs	> 1020 ng/g lipid	51	1.8 (0.9–3.9)	Interaction with EBV-EA immunity assessed; pooled analysis of studies conducted at different times with different specimens 36 congeners measured
				Immunotoxic PCBs	> 1020 ng/g lipid	57	3.2 (1.4–7.4)	
				Total PCBs, EBV EA ≤ 80	> 1018 ng/g lipid	17	1.6 (0.5–5.1)	
				Total PCBs, EBV EA > 80	> 1018 ng/g lipid	22	4.0 (1.2–14)	
				Immunotoxic PCBs, EBV EA ≤ 80	> 348 ng/g lipid	18	3.2 (1.7–11)	
				Immunotoxic PCBs, EBV EA > 80	> 348 ng/g lipid	25	6.4 (1.9–24)	
Hardell *et al.* (2009), Sweden	99 99	Population	NHL (200, 202)	Lipid-adjusted plasma PCB concentrations				Age, sex, BMI, time of sampling
				Total PCBs	> Median	59	2.0 (0.99–3.9)	Both sexes; interaction with EBV-EA immunity assessed.
				Moderately chlorinated		58	1.8 (0.9–3.6)	
				Higher-chlorinated		63	1.7 (0.8–3.4)	
				Immunotoxic		54	1.5 (0.8–3.0)	
			Follicular lymphoma	Total PCBs	> 646 ng/g lipid	15	5.9 (1.9–14)	
				Immunotoxic	> 226 ng/g lipid	13	3.0 (0.9–11)	
			Diffuse large B-cell lymphoma	Total PCBs	> 646 ng/g lipid	19	1.6 (0.6–4.0)	
				Immunotoxic	> 226 ng/g lipid	19	1.4 (0.6–3.3)	

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Nordström *et al.* (2000), Sweden	54 54	Population	Hairy cell leukaemia	Lipid-adjusted serum PCB concentrations, EBV EA antibody titre				Age, BMI Only men; OR for total PCB
				Total PCBs	> 831.6 ng/g lipid; EBV EA > 40	13	4.4 (1.2–18.5)	> 831.6 ng/g lipid = 0.8 (0.3–1.9)
				Immunotoxic PCBs	> 285.4 ng/g lipid; EBV EA > 40	15	11.3 (2.3–73.1)	
Spinelli *et al.* (2007), Canada	422 460	Population	NHL (200, 202)	Lipid-adjusted plasma PCB concentration	Quartiles of exposure (ng/g lipid)			Age, sex, region, ethnicity, education, family history of NHL, BMI and farming; sum of 14 congeners
				Sum of PCBs	101–155.6	103	1.41 (0.93–2.14)	
					155.7–220.0	77	1.11 (0.71–1.74)	
					> 220.0	142	2.14 (1.38–3.30)	*P* for trend < 0.001
				DL-PCBs (105, 118, 156)	10.13–15.35	96	1.41 (0.91–2.16)	
					15.36–23.72	82	1.57 (1.00–2.46)	
					> 23.72	143	2.40 (1.53–3.77)	*P* for trend < 0.001
				PCB-105	> 1.32	132	1.06 (0.93–1.42)	
				PCB-118	4.58–7.78	88	1.12 (0.74–1.69)	
					7.79–12.85	95	1.23 (0.81–1.88)	
					> 12.85	129	1.77 (1.15–2.72)	*P* for trend = 0.004
				PCB-156	3.66–5.51	85	1.10 (0.72–1.68)	
					5.52–8.32	105	1.43 (0.93–2.21)	
					> 8.32	128	1.77 (1.14–2.74)	*P* for trend = 0.004
				NDL-PCBs (28, 99 138, 153, 180, 183, 187)	88.58–136.2	96	1.30 (0.85–1.97)	
					136.21–196.4	93	1.19 (0.76–1.86)	
					> 196.4	148	2.18 (1.41–3.38)	*P* for trend < 0.001
				PCB-28	Undetected	348	1.0 (Ref)	
					> 1.38	74	0.95 (0.67–1.34)	

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Spinelli et al. (2007), Canada (cont.)				PCB-99	3.07–4.83	82	0.78 (0.52–1.15)	
					4.84–7.78	85	0.81 (0.54–1.21)	
					> 7.78	130	1.27 (0.86–1.87)	*P* for trend = 0.045
				PCB-138	11.62–19.28	90	0.93 (0.62–1.38)	
					19.29–29.72	94	0.99 (0.66–1.50)	
					> 29.72	138	1.46 (0.98–2.18)	*P* for trend = 0.02
				PCB-153	25.3–38.68	86	1.04 (0.68–1.57)	
					38.69–59.0	106	1.34 (0.87–2.04)	
					> 59.0	140	1.79 (1.17–2.72)	*P* for trend = 0.002
				PCB-170	7.17–11.17	93	1.17 (0.77–1.79)	
					11.18–17.23	107	1.41 (0.91–2.18)	
					> 17.24	134	1.80 (1.16–2.79)	*P* for trend = 0.005
				PCB-180	21.94–35.63	94	1.28 (0.82–2.00)	
					35.64–54.72	89	1.25 (0.78–2.00)	
					> 54.72	126	1.91 (1.19–3.07)	*P* for trend = 0.005
				PCB-183	1.87–3.95	107	0.83 (0.59–1.18)	
					> 3.95	153	1.22 (0.87–1.71)	*P* for trend = 0.113
				PCB-187	5.94–9.82	98	1.27 (0.83–1.95)	
					9.83–15.46	79	1.04 (0.66–1.63)	
					> 15.46	136	1.92 (1.23–2.98)	*P* for trend = 0.003
			Follicular lymphoma	Total PCBs	Largest vs smallest quartile		2.0 (1.1–3.7)	
				DL-PCBs			2.5 (1.3–4.7)	
				PCB-105			0.9 (0.6–1.4)	
				PCB-118			2.0 (1.1–3.7)	
				PCB-156			2.4 (1.2–4.5)	
				NDL-PCBs			2.1 (1.1–3.9)	
				PCB-28			0.7 (0.4–1.3)	
				PCB-99			1.3 (0.8–2.3)	
				PCB-138			1.5 (0.9–2.7)	
				PCB-153			2.0 (1.1–3.7)	
				PCB-170			1.5 (0.8–2.8)	
				PCB-180			1.6 (0.8–3.1)	

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Spinelli et al. (2007), Canada (cont.)			Diffuse large B-cell lymphoma	PCB-183			1.6 (1.0–2.7)	
				PCB-187			1.8 (1.0–3.3)	
				Total PCBs	Largest vs smallest quartile		1.8 (0.8–4.1)	
				DL-PCBs			2.1 (0.9–4.9)	
				PCB-105			0.8 (0.5–1.5)	
				PCB-118			2.0 (0.9–4.7)	
				PCB-156			1.3 (0.6–3.0)	
				NDL-PCBs			1.8 (0.8–4.1)	
				PCB-28			1.3 (0.7–2.4)	
				PCB-99			1.0 (0.5–2.0)	
				PCB-138			1.2 (0.6–2.6)	
				PCB-153			1.3 (0.6–2.7)	
				PCB-170			1.6 (0.7–3.6)	
				PCB-180			1.2 (0.5–2.9)	
				PCB-183			0.8 (0.4–1.6)	
				PCB-187			1.7 (0.7–4.0)	
Cocco *et al.* (2008), France, Spain, Germany	174 203	Hospital and population	NHL (200, 202)	Lipid-adjusted plasma PCB concentration (ng/g lipid) Total PCBs	200.43–387.79	50	1.2 (0.6–2.2)	Age, sex, education, centre Sum of 9 congeners LOD, 0.20–0.50 µg/L
					387.8–576.36	33	0.7 (0.3–1.4)	
					> 576.36	50	1.0 (0.5–2.0)	*P* for trend = 0.83
				PCB-28	10.51–31.70	25	0.9 (0.4–1.8)	
					31.71–67.94	21	0.7 (0.3–1.5)	
					> 67.94	45	1.6 (0.8–3.2)	*P* for trend = 0.23
				PCB-118	12.31–38.76	41	1.0 (0.5–2.0)	
					38.77–59.17	20	0.5 (0.2–2.0)	
					> 59.18	19	0.4 (0.2–0.8)	*P* for trend = 0.004
				PCB-138	45.74–72.41	37	1.1 (0.6–1.9)	
					72.42–116.12	42	1.1 (0.6–2.0)	
					> 116.12	44	1.1 (0.6–2.0)	*P* for trend = 0.88

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Cocco et al. (2008), France, Spain, Germany (cont.)				PCB-153	62.57–100.66	51	1.5 (0.8–2.8)	
					100.67–142.43	28	0.8 (0.4–1.6)	
					> 142.43	52	1.3 (0.7–2.5)	*P* for trend = 0.70
				PCB-170	0.21–21.53	40	1.1 (0.5–2.2)	
					21.54–34.28	36	0.8 (0.4–1.7)	
					> 34.28	45	1.0 (0.5–1.8)	*P* for trend = 0.83
				PCB-180	0.31–51.22	40	1.2 (0.6–2.6)	
					51.23–85.93	50	1.4 (0.6–3.0)	
					> 85.93	61	1.5 (0.7–3.2)	*P* for trend = 0.31
			Chronic lymphocytic leukaemia	Total PCBs	200.43–387.79	15	1.4 (0.5–4.4)	
					387.8–576.36	10	0.8 (0.2–2.8)	
					> 576.36	18	1.4 (0.4–4.5)	*P* for trend = 0.71
				Immunotoxic PCBs	> median	NR	3.2 (0.9–12)	Subgroup analysis of combined French and German subjects
			Diffuse large B-cell lymphoma	Total PCBs	200.43–387.79	12	0.8 (0.3–2.1)	
					387.8–576.36	7	0.5 (0.1–1.6)	
					> 576.36	13	0.9 (0.3–2.5)	
De Roos et al. (2005), USA	100 100	Population	NHL (200, 202)	Lipid-adjusted plasma PCB concentration (ng/g lipid)	Quartiles of PCB concentration			Sex, study site, birth date, and date of blood draw
				PCB-74	7.8–13.3	28	1.12 (0.51–2.45)	
					13.4–19.3	16	0.73 (0.30–1.75)	
					> 19.4	31	1.26 (0.52–3.03)	*P* for trend = 0.66
				PCB-99	5.6–9.3	22	0.63 (0.24–1.68)	
					9.4–16.1	30	1.04 (0.45–2.39)	
					> 16.1	24	0.77 (0.28–2.10)	*P* for trend = 1.0
				PCB-118	8.1–11.8	14	0.36 (0.13–0.98)	
					11.9–25.8	30	0.91 (0.42–1.98)	
					> 25.8	24	0.73 (0.29–1.84)	*P* for trend = 0.88

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
De Roos et al. (2005), USA (cont.)				PCB-138–158	25.2–38.3	20	0.82 (0.38–1.78)	
					38.4–55.5	25	1.04 (0.47–2.33)	
					> 55.5	29	1.42 (0.49–3.05)	*P* for trend = 0.53
				PCB-146	4.4–6.0	24	1.06 (0.36–3.08)	
					6.1–8.7	24	1.37 (0.50–3.79)	
					> 8.7	32	1.81 (0.70–4.64)	*P* for trend = 0.17
				PCB-153	37–56.2	27	1.36 (0.54–3.25)	
					56.3–71.3	16	0.80 (0.32–2.03)	
					> 71.3	34	1.59 (0.63–4.00)	*P* for trend = 0.40
				PCB 156	5.6–7.8	27	1.70 (0.48–6.03)	
					7.9–9.8	16	1.02 (0.32–3.26)	
					> 9.8	40	2.70 (0.97–7.50)	*P* for trend = 0.03
				PCB-170	12.2–17.0	16	0.84 (0.36–1.92)	
					17.1–22.5	27	1.59 (0.63–4.02)	
					> 22.5	31	1.73 (0.73–4.14)	*P* for trend = 0.13
				PCB-180	28.7–41.2	21	1.72 (0.65–4.54)	
					41.3–54.4	22	1.82 (0.70–4.76)	
					> 54.4	41	3.50 (1.53–9.15)	*P* for trend = 0.01
				PCB-183	2.8–4.4	21	0.93 (0.16–5.46)	
					4.5–6.3	22	0.73 (0.26–2.06)	
					> 6.3	27	1.02 (0.36–2.93)	*P* for trend = 0.96
				PCB-187	8.9–12.0	13	0.59 (0.22–1.57)	
					12.1–18.0	33	1.34 (0.59–3.04)	
					> 18.0	30	1.22 (0.49–3.08)	*P* for trend = 0.18
				PCB-194	8.0–11.2	24	1.59 (0.62–4.04)	
					11.3–15.6	20	1.35 (0.53–3.48)	
					> 15.6	37	2.68 (1.04–6.90)	*P* for trend = 0.04
				PCB-126 (pg/g lipid)	19.0–30.3	20	0.65 (0.29–1.49)	
					30.4–52.7	21	0.73 (0.31–1.72)	
					> 52.7	30	1.09 (0.49–2.41)	*P* for trend = 0.54

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
De Roos et al. (2005), USA (cont.)				PCB-169 (pg/g lipid)	18.6–28.4	23	1.14 (0.49–2.66)	
					28.5–37.7	20	1.08 (0.41–2.82)	
					> 37.7	35	2.62 (0.88–7.80)	*P* for trend = 0.11
				Lower chlorinated PCBs (2–4) (mmol/g lipid)	0.028–0.046	28	1.12 (0.51–2.45)	
					0.047–0.066	16	0.73 (0.30–1.75)	
					> 0.067	31	1.26 (0.52–3.03)	*P* for trend = 0.66
				Moderately chlorinated PCBs (5–7) (mmol/g lipid)	0.386–0.599	25	1.52 (0.58–4.01)	
					0.600–0.785	20	1.43 (0.49–4.11)	
					> 0.785	29	1.88 (0.67–5.26)	*P* for trend = 0.29
				Highly chlorinated PCBs (8–10) (mmol/g lipid)	0.019–0.026	24	1.59 (0.62–4.04)	
					0.027–0.036	20	1.35 (0.53–3.48)	
					> 0.036	37	2.68 (1.04–6.90)	*P* for trend = 0.04
				PCB TEQ (summed pg/g lipid, weighted by TEF)	6.41–8.69	16	0.59 (0.25–1.40)	
					8.70–13.17	20	0.86 (0.38–1.98)	
					> 13.17	33	1.51 (0.62–3.67)	*P* for trend = 0.06
Colt *et al.* (2009), USA	685 646	Population	NHL (ICDO-3)	PCB-180 in carpet dust			*Risk increase in % per 10% increase in concentration*	Age, sex, race, study centre, education
					IFNG (C–1615T) TT	243	1.2 (0.1–2.4)	
					IL4 (5′-UTR, Ex1-168C>T) CC	403	1.0 (0.1–1.9)	
					IL16 (3′-UTR, Ex22-871A>G) AA	330	1.1 (0.1–2.1)	
					IL8 (T–251A) TT	172	1.4 (0.05–2.8	
					IL10 (A–1082G) AG/GG	431	0.9 (0.05–1.8)	
	100 100	Population		PCB-180 in plasma	*IFNG* (C–1615T) TT	39	16.9 (3.7–31.6)	
					IL4 (5′-UTR, Ex1-168C>T) CC	62	9.3 (0.9–18.3)	
					IL16 (3′-UTR, Ex22-871A>G) AA	46	15 (3.2–28.0)	
					IL8 (T–251A) TT	27	28.9 (6.4–56.1)	
					IL10 (A–1082G) AG/GG	59	9.9 (1.2–19.4)	

Table 2.9 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Organ site (ICD code)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Colt et al. (2009), USA (cont.)				TEQ in plasma	*IFNG* (C–1615T) TT	39	19.2 (4.8–35.7)	
					IL4 (5′-UTR, Ex1-168C>T) CC	60	12.5 (3.0–22.9)	
					IL16 (3′-UTR, Ex22-871A>G) AA	44	11.2 (–0.3–24.0)	
					IL8 (T–251A) TT	61	9.1 (0.4–18.6)	
					IL10 (A–1082G) AG/GG	57	5.0 (–3.0–13.8)	
Wang *et al.* (2011), USA	685 646	Population	NHL (ICDO-3)	PCB-180 in carpet dust	> 20.7 ng/g HLA-DRB1*0101 absent	81	1.36 (0.93–1.99)	Age, sex, race, study centre
					> 20.7 ng/g HLA-DRB1*0101 present	17	1.25 (0.66–2.38)	No risk estimates presented for AH 8.1 present genotype. In analysis by major lymphoma subtypes, no increase in risk for DLBCL or follicular lymphoma
	100 100	Population		PCB-180 lipid-adjusted plasma concentration	> 28.7 ng/g lipid HLA-DRB1*0101 absent	65	3.93 (1.49–10.35)	
					> 28.7 ng/g lipid HLA-DRB1*0101 present	10	0.66 (0.18–2.37)	

BMI, body mass index; DLBCL, diffuse large B-cell lymphoma; DL-PCB, dioxin-like PCB; EA, early antigen; EBV, Epstein–Barr virus; Ex, exon; IFNG, inferon gamma; IL, interleukin; LOD, limit of detection; NA, not applicable; NHL, non-Hodgkin lymphoma; NR, not reported; OR, odds ratio; PCB, polychlorinated biphenyl; ref, reference; NDL-PCB, non-dioxin-like PCB; TEF, toxic equivalency factor; TEQ, toxic equivalent; vs, versus

Risk was highest for those who had resided 10–19 years in the most polluted area (OR, 3.8; 95% CI, 1.5–9.8) (Maifredi *et al.*, 2011). [The authors used the ICD-9 classification to define NHL, and therefore did not include chronic lymphocytic leukaemia among their cases, which precluded any feasible analysis of specific NHL subtypes.]

Several small case–control studies in Sweden used adipose tissue or serum levels of total PCBs and individual congeners as the exposure indicator. In a first study with 27 cases and 17 controls (Hardell *et al.*, 1996, 1997), risk of NHL was elevated for total PCB concentrations [17 congeners] above the median among controls (OR, 1.8; 95% CI, 0.4–7.4), after adjusting for age and sex. Thirty-six PCB congeners were measured in a second study with 82 cases of NHL and 83 controls. The odds ratio was significantly increased for concentration of immunotoxic PCBs (Moysich *et al.*, 1999a) above the median among the controls (OR, 3.2; 95% 1.4–7.4) (Hardell *et al.*, 2001). An interaction was observed between elevated concentrations of total and immunotoxic PCBs above the median and EBV-EA antibodies: EBV-EA seropositivity (EBV-EA antibody titre >80) and adipose total PCB concentrations were associated with an increase in risk of NHL of two- to fourfold, which was highest when the immunotoxic PCB subgroup was considered (OR, 6.4; 95% CI, 1.9–24). When the low-grade B-cell NHLs were analysed separately, risk associated with elevated median concentrations of immunotoxic PCBs among subjects with EBV-EA seropositivity was increased 17-fold (95% CI, 3.1–150; 16 cases) (Hardell *et al.*, 2001).

Another case–control study in Sweden included 99 cases of NHL and 99 population controls, matched to cases by age, sex, and health-service region (Hardell *et al.*, 2009). After adjusting by age, sex, and BMI, risk of NHL was elevated for values above the median among controls for the sum of PCBs (OR, 2.0; 95% CI, 0.99–3.9), and to a lesser extent for the subgroups of moderately chlorinated PCBs, highly chlorinated PCBs, or immunotoxic PCBs. Risk was highest for follicular lymphoma for the subgroup of highly chlorinated PCBs (OR, 9.6; 95% CI, 1.9–49; 18 cases); immunotoxic PCBs (OR, 3.0; 95% CI, 0.9–11); and less chlorinated PCBs (OR, 2.8; 95% CI, 0.9–9.0). Risks were only moderately and non-significantly elevated for diffuse large B-cell lymphoma. When stratified by EBV-EA antibody titre, risk of NHL associated with total PCB concentration above the median was 5.2 (95% CI, 1.9–14) among EBV-EA-positive subjects, and ranged from 3.0 to 5.0 for the above-mentioned PCB subgroups; risk for diffuse large B-cell lymphoma ranged from 3.8 to 7.0 by PCB subgroup (all statistically significant), and was 6.2 (95% CI, 1.6–25) for immunotoxic PCBs (Hardell *et al.*, 2009).

A case–control study focused on 54 cases of hairy cell leukaemia [a rare subtype of NHL] identified in the Swedish Cancer registry, and 54 controls drawn from the national population registry, matched to cases by age, sex, and county (Nordström *et al.*, 2000). Concentrations of 36 PCBs were measured in plasma. Overall, risk was not elevated for total PCB concentration greater than the median value (OR, 0.8; 95% CI, 0.3–1.9). When stratifying by EBV-EA antibody titre, the odds ratio for exposure above the median of values was 4.4 (95% CI, 1.2–18.5; 13 cases) for total PCBs and 11.3 (95% CI, 2.3–73.1; 15 cases) for immunotoxic PCBs among subjects with EBV-EA titres ≥ 40 (Nordström *et al.*, 2000). [The Working Group highlighted some methodological concerns about this group of studies, including poor precision, recruitment of cases and controls at different times, some with PCB measurements in adipose tissue and others with measurements in plasma.]

The largest case–control study of PCB body burden in relation to risk of NHL was conducted in Canada (Spinelli *et al.*, 2007). Lipid-adjusted concentrations of 14 PCB congeners were measured in pretreatment samples of plasma from

422 cases of NHL and 460 population controls, frequency-matched to cases by 5-year age-groups, sex, and residence. Odds ratios were adjusted for age, sex, education, BMI, ethnicity, farming, and family history of NHL. Risk of NHL was found to be highest in the highest quartile of the sum of dioxin-like PCBs (OR, 2.40; 95% CI, 1.53–3.77) and of non-dioxin-like congeners (OR, 2.18; 95% CI, 1.41–3.38). Individual congeners showing a significant excess risk in the top quartile of plasma concentration included PCB-118 and PCB-156, among the dioxin-like PCBs, and PCB-138, PCB-153, PCB-170, PCB-180, and PCB-187, among the non-dioxin-like PCBs. The observed associations were consistent across the four NHL subtypes examined, including DLBCL, follicular lymphoma, T-cell lymphoma, and other B-cell lymphomas (Spinelli *et al.*, 2007). [This was one of the largest studies of NHL and PCBs, and accounted for relevant confounders. The Working Group judged it to be a high-quality study, which was notable for providing results for individual congeners and lymphoma subtypes. While the participation rate for controls was less than 50%, the Working Group noted that this was typical of the available case–control studies and that potential confounding factors, including education, were comparable between cases and controls despite differences in participation. The most consistent associations were seen for follicular lymphoma and exposure to dioxin-like PCBs.]

A multicentre European study of NHL included 174 cases and 203 controls from France, Germany, and Spain (Cocco *et al.*, 2008). Patients admitted to the same hospital as the cases for non-cancer diseases not related to known risk factors for NHL were selected as controls in France and Spain; controls in Germany were a random sample of the general population. Concentrations of nine PCB congeners were measured in plasma, and risk estimates were adjusted by age (continuous), sex, education, and centre. Risk of NHL did not increase by quartile of plasma concentration of total PCBs, or specific congeners, or the functional PCB congener groups as defined by Hansen (Hansen, 1998). When exploring risk by lymphoma subtype, a nonsignificant increase was observed for chronic lymphocytic leukaemia in the top quartile of concentration of immunotoxic PCBs and BRCA1-inhibiting PCBs, with no indication of an increasing trend, or of an association with specific PCB congeners. No association was observed with risk of diffuse large B-cell lymphoma. However, risk of chronic lymphocytic leukaemia associated with plasma concentrations of immunotoxic PCBs above the median showed a threefold increase (OR, 3.2; 95% CI, 0.9–11.5), increasing to sixfold (OR, 6.1; 95% CI, 1.0–37.8) in the upper quartile, in subgroup analyses of the German and French subgroups combined, but not in the Spanish subgroup; a significant heterogeneity by country was observed for risk of chronic lymphocytic leukaemia associated with immunotoxic PCBs, but not for the sum of total PCBs. [The Working Group judged this international study to be high in quality; the classification of lymphoma was particularly meticulous. Although the overall results were null, the association of immunotoxic PCBs with chronic lymphocytic leukaemia in two of the three centres is noteworthy. The heterogeneity between countries may have been a result of differences in PCB exposure or distribution of confounding factors.]

Pretreatment plasma samples were available in a subset of 100 cases with a histologically confirmed diagnosis of NHL and 100 controls out of the 1321 cases and 1057 general population controls who participated in a case–control study on NHL conducted by the United States National Cancer Institute in 1998–2000 in four areas with population-based cancer registries (Iowa, Los Angeles, CA, Detroit, MI, and Seattle, WA) (De Roos *et al.*, 2005). Concentrations of 36 non-coplanar and 4 coplanar congeners were measured in plasma. Risk of NHL overall and of its major subtypes was analysed in relation

to 28 PCB congeners detected in at least 30% of samples. Values below the detection limit were estimated by multiple imputation. Odds ratios were adjusted for the matching factors, age, sex, study site, and date of blood draw. Other potential confounders were tested, including education, race, BMI, and family history of NHL, but no confounding was observed. The results showed significant upward trends in risk of NHL with increasing quartiles of plasma concentration of the subgroup of highly chlorinated PCB congeners (test for trend, $P = 0.04$), which included PCB-156, PCB-180, and PCB-194. An increase of 10 TEQ pg/g lipid was associated with a 35% excess risk of NHL (95% CI, 1.02–1.79). Some associations were stronger among the 14 cases of DLBCL than the 25 cases of follicular lymphoma, both in men and women, and trends by exposure quartiles became significant for follicular lymphoma for PCB-180 and PCB-187 (De Roos *et al.*, 2005). [Despite the extensive analysis, this was a relatively small study, with wide confidence intervals.]

Colt *et al.* (2009) used the same data set to explore the interaction between common variants in genes implicated in the immune and inflammatory response and PCB-180, (the non-dioxin like PCB that showed the strongest association between NHL and levels measured in plasma (100 cases and 100 controls) and carpet dust (682 cases and 513 controls) in the analysis by De Roos *et al.* (2005). Sixty-one single nucleotide polymorphisms in 36 proinflammatory and other immunoregulatory genes were analysed in samples of blood or buccal cells. Relative risk estimates were adjusted for sex, age, race, education, and study centre. The concentration of PCB-180 in plasma was associated with increased risk of NHL (OR, 8.3%; 95% CI, 1.9–14.6% per 10% increment), but the concentration in carpet dust was not (OR, 0.7%; 95% CI, 0.0–1.3% per 10% increment). Significant increases in risk of NHL were observed for PCB-180 in both plasma and carpet dust and for *IFNG* (C–1615T) TT, *IL4* (5′-UTR, Ex1-168C>T) CC, *IL16* (3′-UTR, Ex22-871A>G) AA, *IL8* (T–251A) TT, and *IL10* (A–1082G) AG/GG genotypes (Colt *et al.*, 2009).

Another analysis was conducted on the same data set to explore the interaction between status of HLA-DRB1*01:01 class II leukocyte surface antigen and of the extended ancestral haplotype (AH) 8.1 (HLA-A*01-B*08-DR*03-TNF-308A) and blood concentrations of PCB-180 above the median in the control group. Risk of NHL overall was elevated among study subjects lacking the HLA-DRB1*01:01 allele or the AH 8.1 allele (OR, 3.93; 95% CI, 1.49–10.35). No significant increase in risk was observed with PCB-180 in carpet dust or for DLBCL or follicular lymphoma (Wang *et al.*, 2011). [These related studies were well conducted, but the subgroup analyses were based on small numbers.]

2.3.2 Cancer of the breast

See Table 2.10

(a) Smaller studies

Case–control studies of cancer of the breast with 100 or fewer cases, most published before 2000, are reviewed here briefly and are not presented in the table. Most of these studies did not present risk estimates according to PCB concentrations.

One of the earliest studies looked at PCB concentrations in samples of breast adipose tissue from 14 living and 18 deceased patients with cancer of the breast, 21 similar samples from non-cancer patients, and samples of adipose tissue from 35 non-cancer autopsies, and found no significant differences (Unger *et al.*, 1984).

In another study, mean concentrations of PCBs in the breast tissue of 20 women with cancer of the breast were significantly higher ($P = 0.02$) than in 20 women with benign breast disease, and the association persisted after controlling for age, smoking, and BMI (Falck *et al.*, 1992).

Table 2.10 Case-control studies on cancer of the breast and exposure to PCBs

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Recio-Vega *et al.* (2011), Comarca Lagunera, Mexico	70 70	Hospital-based: 70 women with biopsies negative for malignancy, from the same hospital	Questionnaire; serum concentrations of 20 PCB congeners[a] measured by GC	Total PCBs	NR	1.09 (1.02–1.16)	Age, age at menarche, lactation, menopausal status, BMI, family history of breast cancer [The Working Group was not clear on how analysis was performed to obtain risk estimates.] PCb groups according to Wolff & Toniolo (1995)
				Premenopausal	NR	1.08 (0.99–1.17)	
				Postmenopausal	NR	1.13 (1.01–1.25)	
				Group 1a	NR	1.19 (0.81–1.7)	
				Group 1b	NR	1.40 (0.94–2.1)	
				Group 2a	NR	1.22 (0.99–1.49)	
				Group 2b	NR	1.90 (1.25–2.88)	
				Group 3	NR	1.81 (1.08–3.04)	
				Group 4	NR	1.57 (1.20–2.07)	
				Group 5	NR	1.30 (0.84–2.04)	
Moysich *et al.* (1998, 1999b), Erie and Niagara counties of western New York, USA, 1986–1991	154 postmenopausal women with incident primary breast cancer identified from hospitals 192 controls	Community controls frequency matched by age and county of residence	Structured interview; *CYP1A1* polymorphism was determined by PCR-RFLP; Lipid-adjusted serum PCBs (56 congener peaks based on the concentrations of 73 congeners) measured by GC (ng/g lipid)	*Total PCBs:* PCB low (0.75–3.72 ng/g):			Age, education, family history of breast cancer, parity, quetelet index (BMI), duration of lactation, age at first birth, serum lipids, years since last pregnancy, fruit and vegetable intake, serum lipids, and smoking status
				Ile:Ile	62	1.00	
				Ile:Val/Val:Val	8	0.88 (0.29–2.70)	
				PCBs > 3.72 ng/g:			
				Ile:Ile	65	1.08 (0.62–1.89)	
				Ile:Val/Val:Val	19	2.93 (1.18–7.45)	
				Total PCBs: All subjects:			
				2.93–4.43	45	0.70 (0.37–1.29)	
				4.44–19.04	56	1.14 (0.61–2.15)	$P = 0.51$
				Never lactated ($n = 107$):			
				2.93–4.43	15	1.71 (0.55–5.35)	
				4.44–19.04	20	2.87 (1.01–7.29)	$P = 0.07$
				Ever lactated ($n = 191$):			
				2.93–4.43	41	0.38 (0.17–1.03)	
				4.44–19.04	36	0.71 (0.31–1.61)	$P = 0.72$
				Less chlorinated			Data NR for women who never lactated
				0.01–0.31	59	2.04 (1.09–3.83)	
				0.32–1.65	63	1.40 (0.76–2.59)	

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Moysich et al. (1998, 1999b), (cont.)				*Moderately chlorinated*			
				All subjects:			
				2.20–3.12	41	0.57 (0.03–1.07)	
				3.13–15.07	60	1.37 (0.73–2.59)	*P* = 0.69
				Never lactated:			
				2.20–3.12	12	0.73 (0.22–2.63)	
				3.13–15.07	23	3.57 (1.10–8.60)	P = 0.08
				Highly chlorinated			
				All subjects:			
				0.26–0.44	43	0.79 (0.42–1.52)	
				0.45–1.30	54	1.19 (0.60–2.36)	
				Never lactated:			
				0.26–0.44	11	0.51 (0.15–1.69)	
				0.45–1.30	21	1.53 (0.47–4.95)	
Wolff *et al.* (2000b), New York, New York, USA	175 cases with incident breast cancer 355 controls	Hospital controls matched by age, race/ethnicity	Structured interview in person or by telephone Lipid-adjusted serum PCB concentration (µg/g lipid)	Tertiles of PCB concentration (µg/g lipid)			Age, age^2, menopausal status, race, BMI, family history of breast cancer, lactation, parity Tumor stage and markers (ER, PR, p53, erbB-2) identified histologically and immunohistochemically by pathologist ORs not reported by tumour marker status
				Highly chlorinated			
				0.460–0.798	46	0.88 (0.52–1.5)	
				0.799–3.3	46	0.78 (0.45–1.3)	
				Less chlorinated			
				0.085–0.162	54	1.47 (0.84–2.6)	
				0.163–2.39	38	0.96 (0.53–1.7)	

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Millikan *et al.* (2000), North Carolina, USA 1993–1996	748 cases, aged 20–74 years 659 controls	Population-based, frequency-matched to cases on race and age	Structured interview. Lipid-adjusted plasma concentrations of PCBs measured by GC (μg/g lipid)	Tertiles of PCB concentration			Age, age^2, race (all participants), menopausal status, BMI, parity, lactation, use of HRT, and income Response rates: cases, 76%; controls, 55%. PCB and lipid measurements were available for 748 cases (84%) and 659 controls (78%)
				Total PCBs			
				All women:			
				0.283–0.468	266	1.29 (0.97–1.72)	
				≥ 0.469	243	1.09 (0.79–1.52)	
				African-American:			
				0.312–0.53	97	1.35 (0.84–2.16)	
				≥ 0.54	116	1.74 (1.00–3.01)	
				White:			
				0.265–0.416	172	1.32 (0.92–1.90)	
				≥ 0.417	135	1.03 (0.68–1.56)	
				Low to moderately chlorinated			
				Tertile 2	NR	0.96 (0.73–1.27)	
				Tertile 3	NR	0.99 (0.73–1.35)	
				Highly chlorinated			
				Tertile 2	NR	1.41 (1.05–1.87)	
				Tertile 3	NR	1.35 (0.97–1.88)	
Li *et al.* (2005), North Carolina, USA, 1993–1996 (same population as Millikan *et al.*, 2000)	612 cases 599 controls	Population	Lipid-adjusted plasma PCB concentration by GC (ng/g lipid)	*Total PCBs*		*CYP1A1 M1* genotype	Age, race, parity, use of HRT, oral-contraceptive use, breast feeding, smoking, alcohol consumption, income, education, height, waist/hip ratio, BMI See Millikan *et al.* (2000) for details
				African-American:			
				< 0.430	66	Non-M1: 1.0 (Ref)	
				≥ 0.430	75	Non-M1: 1.5 (0.9–2.5)	
				< 0.430	42	Any M1: 1.0 (0.6–1.7)	
				≥ 0.430	59	Any M1: 1.4 (0.8–2.5)	Interaction contrast ratio: 0.0 (–0.9–0.9)
				White:			
				< 0.349	174	Non-M1: 1.0 (Ref)	
				≥ 0.349	122	Non-M1: 0.7 (0.5–1.0)	
				< 0.349	45	Any M1: 0.8 (0.5–1.2)	
				≥ 0.349	29	Any M1: 0.8 (0.4–1.4)	Interaction contrast ratio: 0.4 (–0.2–0.9)

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Li et al. (2005), (cont.)							Likelihood ratio test for both groups not statistically significant
				White:		*CYP1A1 M2* genotype	
				< 0.349	210	Non-M2: 1.0 (Ref)	
				≥ 0.349	138	Non-M2: 0.7 (0.5–1.0)	
				< 0.349	11	Any-M2: 0.4 (0.2–0.8)	
				≥ 0.349	15	Any-M2: 0.9 (0.4–1.9)	Interaction contrast ratio: 0.8 (0.1–1.6)
							Likelihood ratio test: $P = 0.02$
				African-American:		*CYP1A1 M3* genotype	
				< 0.430	95	Non-M3: 1.0 (Ref)	
				≥ 0.430	105	Non-M3: 1.3 (0.8–2.0)	
				< 0.430	13	Any M3: 0.6 (0.3–1.2)	
				≥ 0.430	29	Any M3: 1.6 (0.8–3.2)	Interaction contrast ratio: 0.8 (–0.3–1.9)
							Likelihood ratio test: $P = 0.10$
Demers *et al.* (2000, 2002), Quebec City, Quebec, Canada, 1994–1997	315 women with histologically confirmed infiltrating primary breast cancer 523 controls	Hospital and population, 523 cases frequency-matched by age and rural/urban residence	Telephone interview. Lipid-adjusted serum concentrations for 14 PCB congeners[b] measured by GC/ECD (μg/g lipid)	Quartiles of PCB concentration			Age, region of residence, BMI, history of benign breast disease, breastfeeding duration Participation rate: cases, 91%; hospital controls, 89%; and population controls, 47%. PCBs 28, 52, 101, 105 and 128 were detected in < 70% of women and were excluded from analysis. Results for other PCBs were not statistically significant
				PCB-118			
				9.4– < 14.3	64	0.90 (0.58–1.39)	
				14.3– < 22.1	78	1.12 (0.73–1.74)	
				≥ 22.1	104	1.60 (1.01–2.53)	
				PCB-156:			
				5.8–< 7.6	83	1.44 (0.91–2.26)	
				7.6–< 9.8	80	1.44 (0.90–2.31)	
				≥ 9.8	101	1.80 (1.11–2.94)	
				DL-PCBs (PCB-105, PCB-118, and PCB-156 in TEQ ng/kg)			
				4.2 to < 5.7	85	1.63 (1.04–2.55)	
				5.7 to < 7.4	78	1.45 (0.90–2.32)	
				≥ 7.4	102	2.02 (1.24–3.28)	

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Gammon *et al.* (2002), Long Island, New York, USA 1996–1997	646 cases 429 controls	Population based, matched by age	In-person interview and non-fasting blood sample Lipid-adjusted serum concentrations for 24 PCB congeners[c]	Quintiles of PCB concentration *(Sum of PCBs 118, 138, 153, and 180)*			Age, race, reproductive history, benign breast disease Interview response rates: cases, 83.2%; controls, 68.0%. No statistically significant results for other PCBs measured Results reported for four most common congeners. Numerous potential confounders investigated
				262.58–325.56	112	0.76 (0.51–1.15)	
				325.57–427.78	132	0.90 (0.60–1.35)	
				427.79–586.74	123	0.82 (0.54–1.24)	
				583.74–3287.34	126	0.83 (0.54–1.29)	
				PCB-118			
				32.66–46.45	133	0.96 (0.64–1.42)	
				46.46–63.39	109	0.77 (0.52–1.16)	
				63.40–94.94	114	0.82 (0.54–1.24)	
				94.95–1015.88	136	0.93 (0.60–1.43)	
				PCB-138			
				49.38–81.09	153	1.26 (0.85–1.88)	
				81.10–111.15	129	1.04 (0.69–1.55)	
				111.16–156.22	106	0.80 (0.52–1.21)	
				156.23–936.75	120	0.96 (0.63–1.48)	
				PCB-153			
				103.75–130.02	115	0.75 (0.50–1.13)	
				130.03–170.81	132	0.85 (0.57–1.27)	
				170.82–227.54	107	0.68 (0.45–1.03)	
				227.55–1130.08	132	0.86 (0.56–1.32)	
				PCB-180			
				51.49–69.70	121	0.87 (0.58–1.31)	
				69.71–87.41	117	0.81 (0.54–1.23)	
				87.42–120.37	128	0.89 (0.58–1.34)	
				120.38–721.29	134	0.95 (0.62–1.46)	

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Gatto *et al.* (2007), Los Angeles County, USA, 1994–1998	355 African-American women with histologically confirmed invasive breast cancer 327 controls	Population based, African-American women matched by age	Interview with structured questionnaire. Lipid-adjusted serum PCB concentration (congeners NR) measured by GC	Quintiles of total PCBs (μg/g) ≥ 0–0.38 > 0.38–0.47 > 0.47–0.60 > 0.60	 61 46 42 61	 1.06 (0.67–1.67) 0.82 (0.50–1.33) 0.76 (0.47–1.24) 1.01 (0.63–1.63)	Age, BMI, breastfeeding No statistically significant results by ER+/–, *p53*, or HER-2 status *P* for trend = 0.56
Itoh *et al.* (2009), Nagano Prefecture, Japan, 2001–2005	403 women aged 20–74 years with newly diagnosed invasive breast cancer 403 controls	Hospital-based	Self-administered questionnaire; hormone receptor status obtained from medical records; lipid-adjusted serum concentrations of 41 PCB congeners (ng/g lipid)	Total PCB quartiles (median) 110 160 200 290 *Highest vs lowest quartiles of exposure* PCB-153 PCB-138 PCB-180	 126 96 102 79 NR NR NR	 1.00 (ref) 0.79 (0.36–1.72) 0.57 (0.28–1.15) 0.33 (0.14–0.78) 0.40 (0.18–0.91) 0.61 (0.28–1.35) 0.29 (0.13–0.66)	Total lipid concentration in serum, BMI, reproductive risk factors, smoking, diet, medical history *P* for trend = 0.008 *P* for trend = 0.04 *P* for trend = 0.29 *P* for trend = 0.004
Zheng *et al.* (2000a), Connecticut, USA, 1994–1997	304 cases 186 controls	Hospital-based, with benign breast disease or normal tissue	Structured interview; lipid-adjusted breast adipose tissue concentrations of 9 PCB congeners[d] measured by GC (ng/g lipid)	Total PCBs 396.0–562.9 ≥ 563.0	 79 114	 0.6 (0.4–1.0) 0.7 (0.4–1.1)	Age, BMI, fat consumption, income, race, family history of breast cancer, and reproductive risk factors Participation rate: cases, 79%; controls, 74%. Stratification by type of breast disease, menopausal status, parity, lactation and body size showed no association with PCBs

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Zheng *et al.* (2000b), Connecticut, USA, 1995–1997	475 cases 502 controls	Hospital, with benign disease or population matched by age	Structured interview; lipid-adjusted serum concentrations of 9 PCB congeners[d] measured by GC (ng/g lipid)	Total PCBs 604.1–800.0 > 800.0	 160 160	 1.04 (0.76–1.45) 0.95 (0.68–1.32) *P* for trend = 0.41	Age, BMI, reproductive risk factors, HRT, dietary fat intake, family history of breast cancer, income, race, and study site When stratifying by parity, lactation and menopausal and ER status, no association was identified between PCBs and risk of breast cancer
Holford *et al.* (2000), Connecticut, USA, 1994–1997 (same population as Zheng *et al.*, 2000a)	304 cases 186 controls	Hospital-based	Breast adipose tissue analysed for 9 PCB congeners measured by GC (ng/g lipid)	*Linear logistic model* PCB-74 PCB-118 PCB-138 PCB-153 PCB-156 PCB-170 PCB-180 PCB-183 PCB-187 *Logistic ridge regression model* PCB-153 PCB-156 PCB-180 PCB-183	 	10-ppb change in exposure 0.93 (0.84–1.04) 1.04 (0.96–1.12) 1.04 (0.94–1.16) 0.87 (0.78–0.98) 0.79 (0.64–0.99) 0.85 (0.65–1.11) 1.14 (1.0–1.29) 1.82 (1.12–2.98) 1.11 (0.90–1.37) 0.98 (0.96–1.01) 0.87 (0.78–0.99) 1.02 (0.99–1.05) 1.23 (0.98–1.54)	Age, BMI, reproductive risk factors, dietary fat intake, income, fat concentrations of DDE See Zheng *et al.* (2000a) for details

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Zhang *et al.* (2004), Connecticut, USA, 1999–2002	374 Caucasian women 406 controls	Hospital- and population-based, matched by age	Structured in-person interview; lipid-adjusted serum concentrations of 9 PCB congeners[d] measured by GC (ng/g lipid) Genotyping of *CYP1A1 m1*, *m2*, and *m4* by PCR-RFLP	Total PCBs: 310–610 611–2600 *CYP 1A1 m2* genotype All women: Wildtype, low Wildtype, high Variants, low Variants, high Postmenopausal women: Wildtype, low Wildtype, high Variants, low Variants, high	 173 201 157 177 16 24 130 125 13 21	 1.00 (ref.) 1.2 (0.9–1.6) 1.00 (ref.) 1.2 (0.9–1.6) 1.6 (0.7–3.5) 3.6 (1.5–8.2) 1.0 1.1 (0.8–1.6) 1.8 (0.7–4.5) 4.3 (1.6–1.2)	See Zheng *et al.* (2000a, b) for details No significant association for *CYP1A1* m1 or m4 genotype or in premenopausal women
Rusiecki *et al.* (2004), Connecticut USA, 1994–97 (subgroup from same population as Zheng *et al.*, 2000a)	266 cases 347 controls	Hospital-based, benign breast disease	Interview; serum and breast adipose tissue analysed for 9 PCB congeners	*Total PCBs* ER+PR+ 394.31–558.69 > 558.69 ER–PR– 394.31–558.69 > 558.69 ER+PR- 394.31–558.69 > 558.69 ER–PR+ 394.31–558.69 > 558.69	 21 33 20 24 17 16 4 12	 0.6 (0.3–1.2) 0.6 (0.3–1.3) 0.5 (0.3–1.0) 0.5 (0.3–1.1) 1.0 (0.4–2.5) 0.6 (0.2–1.6) 0.2 (0.1–0.7) 0.5 (0.2–12.0)	Age, reproductive risk factors, BMI, family history of breast cancer in a first-degree relative Tumours were apparent with concentrations of PCB-183 (third tertile vs first: OR, 2.4; 95% CI, 1.0–6.0, *P* for trend = 0.03, but data not otherwise shown) Analyses for individual congeners did not show any association

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Stellman *et al.* (2000), Long Island, New York, USA, 1994–1996	232 cases 323 controls	Hospital	Structured interviews; 14 PCB congeners[c] in breast adipose tissue using GC	Total PCBs (ng/g) 181.82–332.24 > 332.24 PCB-156 5.87–13.59 > 13.60 PCB-183 3.16–5.66 > 5.67	 74 103 NR NR NR NR	 1.06 (0.67–1.69) 1.01 (0.60–1.69) 1.9 (1.1–3.0) 1.5 (0.9–2.5) 1.3 (0.8–2.1) 2.0 (1.2–3.4)	Age, BMI, race > 95% of eligible patients agreed to participate. Adipose tissue was obtained from 86% of all subjects. ORs for other PCB congeners, NR
Aronson *et al.* (2000), Ontario, Canada, 1995–1997	217 cases 213 controls	Hospital-based, cancer-free women, matched by age and study site	Telephone interview or mailed questionnaire; breast tissue analysed for 14 PCB congeners[b] expressed in µg/kg	*PCB-105* 4.2–6.1 6.2–12 ≥ 13 Premenopausal 4.2–6.1 > 6.1 Postmenopausal 4.2–6.1 > 6.1 *PCB-118* 17–27 28–49 ≥ 50 Premenopausal 17–27 > 27 Postmenopausal 17–27 > 27	 NR NR NR 12 30 25 86 NR NR NR 19 28 30 91	 1.16 (0.62–2.14) 2.03 (1.12–3.68) 3.17 (1.51–6.68) *P* for trend ≤ 0.01 1.29 (0.52–3.20) 3.91 (1.73–8.86) 0.98 (0.38–1.49) 1.49 (0.70–3.16) 1.25 (0.68–2.29) 1.88 (1.00–3.55) 2.31 (1.11–4.78) 1.04 (0.46–2.35) 2.85 (1.24–6.52) 1.39 (0.57–3.41) 1.58 (0.70–3.58)	Age, study site, HRT, ethnicity, family history of breast cancer, BMI, fat intake, alcohol intake, smoking, reproductive history Most controls were diagnosed with benign breast disease PCBs 28, 52, 101 and 128 were < LOD for > 30% of subjects and were not investigated

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
Aronson et al. (2000), (cont.)				*PCB-170*			
				24–34	NR	1.60 (0.92–2.78)	
				35–53	NR	1.09 (0.61–1.96)	
				≥ 54	NR	1.15 (0.60–2.22)	
				Premenopausal			
				24–34	24	0.83 (0.39–1.78)	
				> 34	25	0.89 (0.49–1.91)	
				Postmenopausal			
				24–34	51	3.27 (1.44–7.44)	
				> 34	76	1.63 (0.77–3.45)	
				PCB-180			
				52–71	NR	1.56 (0.90–2.70)	
				72–105	NR	1.21 (0.68–2.14)	
				≥ 106	NR	1.27 (0.66–2.46)	
				Premenopausal			
				52–714	26	1.07 (0.55–2.27)	
				> 71	23	0.89 (0.42–1.91)	
				Postmenopausal			
				52–714	46	2.43 (1.09–5.43)	
				> 71	80	1.77 (0.85–3.69)	
Aschengrau *et al.* (1998), Cape Cod, Massachusetts, USA, 1983–1986	261 incident cases 753 controls	Population, similar age and race	Structured interview, JEM and expert assessment	Possible or probable	5	3.2 (0.8–12.2).	Age, vital status, family history of breast cancer, age at first birth, personal history of prior breast cancer, benign breast disease, educational level and race PCB congeners to which cases were potentially exposed are not specified. Response rate: cases, 79%; controls, 74–81%

Table 2.10 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	OR (95% CI)	Covariates Comments
McElroy *et al.* (2004), Wisconsin, USA, 1998–2000	1481 cases 1301 controls	Population-based, of similar age	Telephone interview; consumption of sport-caught fish	*Recent consumption of sport-caught fish*			Age, family history of breast cancer, alcohol consumption, weight gain, weight at age 18 years, education, reproductive history
				Any	701	1.00 (0.86–1.17)	
				Premenopausal	286	1.24 (0.96–1.59)	
				Postmenopausal	388	0.91 (0.74–1.11)	
				Recent consumption of Great Lakes fish			
				Any	210	1.06 (0.84–1.33)	
				Premenopausal	95	1.70 (1.16–2.50)	
				Postmenopausal	104	0.78 (0.57–1.07)	

[a] The 20 PCB congeners were PCBs 8, 18, 28, 44, 52, 66, 77, 101, 105, 118, 126, 138, 148, 153, 170, 180, 187, 195, 206 and 209
[b] The 14 PCB congeners were PCBs 28, 52, 99, 101, 105, 118, 128, 138, 153, 156, 170, 180, 183, and 187
[c] The 24 PCB congeners were PCBs 15, 28, 74, 66, 56, 101, 99, 82, 118, 146, 153, 105, 138, 178, 187, 183, 167, 174, 177, 156, 180, 170, 199, and 203
[d] The 9 congeners were PCBs 74, 118, 138, 153, 156, 170, 180, 183, and 187
[e] The 14 PCB congeners were PCBs 74, 99, 118, 138, 146, 153, 156, 167, 170, 172, 178, 180, 183, and 187

BMI, body mass index; CI, confidence interval; ER, estrogen receptor; GC, gas chromatography; HRT, hormone replacement therapy; Ile, isoleucine; JEM, job-exposure matrix; LOD, limit of detection; NR, not reported; OR, odds ratio; PCB, polychlorinated biphenyl; PCR-RFLP, polymerase chain reaction–restriction fragment length polymorphism; PR, progesterone receptor; ref., reference; TEQ, toxic equivalent; Val, valine; vs, versus

In a study in Quebec City, Canada, in 17 women with cancer of the breast and 17 controls (Dewailly *et al.*, 1994), the concentration of PCB-99 was higher in the breast adipose tissue of women with ER-positive (ER+) infiltrating adenocarcinoma than in controls, while there were no significant differences for ER– women with cancer of the breast compared with controls, or for other PCB congeners or total PCBs.

In a study in Sweden, PCB concentrations were measured in non-tumour breast adipose tissue of 43 women with breast cancer and 35 controls (Liljegren *et al.*, 1998). Odds ratios adjusted for age and parity showed no association with concentrations of total PCB congeners in all subjects. However, among the subgroup of women with ER+ tumours, increased risk was observed for PCB-77 (OR, 33; 95% CI, 1.8–588) and PCB-126 [odds ratio not calculated as there were no unexposed cases].

In Hesse, Germany, concentrations of 12 PCB congeners in breast tissue from 45 women with cancer of the breast were compared with those in breast tissue from 20 women with benign breast disease: the average concentration of PCB-118 was significantly higher in the cases, with no statistical difference for other congeners (Güttes *et al.*, 1998).

A case–control study in eastern Slovakia included 24 cases of cancer of the breast diagnosed between 1997 and 1999 and 88 population controls, and measurements were made of 15 PCBs in serum (Pavuk *et al.*, 2003). Median concentrations of total PCBs were slightly higher among controls, and although odds ratios were less than unity, no finding was statistically significant.

In two reports of studies of 100 cases of cancer of the breast and 100 surgical controls in Belgium (Charlier *et al.*, 2003), concentrations of PCB-101 and PCB-153 were significantly higher for cases than controls. A second study of 60 cases and controls by the same authors reported an association only for PCB-153 (OR, 1.8; 95% CI, 1.4–2.5) after adjusting for age and reproductive risk factors (Charlier *et al.*, 2004). [It was not clear whether the same population was studied in both articles.]

In a case–control study in Mexico, 70 cases of cancer of the breast were compared with 70 hospital controls, and blood samples were taken for measurement of 20 PCB congeners (Recio-Vega *et al.*, 2011). An increased risk of cancer of the breast was apparent for total PCBs (OR, 1.09; 95% CI, 1.02–1.16) and for the exposure groups 2b (OR, 1.90; 95% CI, 1.25–2.88), 3 (OR, 1.81; 95% CI, 1.08–3.04), and 4 (OR, 1.57; 95% CI, 1.20–2.07) defined according to Wolff & Toniolo (1995). Elevated odds ratios were reported for several PCB congeners (PCB-118, PCB-128, PCB-138, PCB-170, PCB-180, PCB-187, PCB-195, PCB-206 and PCB-209) and risks were generally higher in postmenopausal women. [Although this was a small study, several increased risks were reported. However, the analytical approach was unclear to the Working Group and the age distribution was notably different in cases and controls, suggesting potential for residual confounding by age.]

Using a registry of banked serum collected between 1981 and 1987 from 63 Alaskan native women who subsequently developed cancer of the breast and 63 age-matched cancer-free women, analyses adjusting for ethnicity, family history of cancer of the breast, and parity showed no association with PCB exposure (Rubin *et al.*, 2006). In a study in Greenland of 31 cases of cancer of the breast and 115 controls, all of Inuit descent, some evidence of higher serum concentrations of PCBs was found for patients with cancer of the breast compared with controls; however, the odds ratios for total PCBs did not demonstrate any association (Bonefeld-Jørgensen *et al.*, 2011). [The populations included in these studies were of special interest due to their documented high exposures to PCBs.]

(b) *Larger studies of PCB concentrations in blood*

In a case–control study in western New York State, USA, 154 postmenopausal women with incident cancer of the breast and 192 postmenopausal community controls were compared in terms of serum concentrations of 73 detected congeners (Moysich *et al.*, 1998). No association with total PCBs, moderately chlorinated PCBs or highly chlorinated PCBs was found, but increased risk was apparent for less chlorinated PCBs above the detection limit (OR, 1.66; 95% CI, 1.07–2.88 for the combined second and third tertiles); among parous women who had never lactated the magnitude of risk was higher in association with total PCBs (OR, 2.87; 95% CI, 1.01–7.29) and moderately chlorinated PCBs (OR, 3.57; 95% CI, 1.10–8.60). In a subsequent study on PCBs and *CYP1A1* polymorphism (found to be induced by PCBs in experimental studies, see Section 4), no association with *CYP1A1* genotype was found among women with a low PCB body burden; among women with a PCB burden above the median for the control group, an increased risk of cancer of the breast was observed when at least one valine allele was present (OR, 2.93; 95% CI, 1.18–7.45) when compared with women who were homozygous for the isoleucine allele (Moysich *et al.*, 1999b). Adjustment for serum lipids and BMI did not affect the magnitude of this association. [Although not large, this study was rigorous in terms of design and implementation.]

Among patients of several ethnic groups in a hospital-based case–control study in New York City, USA, 175 patients with cancer of the breast and 355 control patients were frequency-matched by age and race/ethnicity (Wolff *et al.*, 2000b). Highly chlorinated and less chlorinated biphenyls and other chlorinated compounds were measured in serum, and the tumour markers ER, progesterone receptor (PR), *p53*, and erbB-2 were assessed. Concentration of PCBs was not associated with risk of cancer of the breast. Risk of cancer of the breast was not examined with respect to tumour stage or markers, but PCB concentrations did not differ according to these factors. [This was a high quality study notable for the number of tumour markers investigated, but the analysis focused largely on exposure markers, rather than exposure–disease associations.]

In a population-based case–control study of cancer of the breast in African-American and white women in North Carolina, USA, 748 cases and 659 controls were enrolled (Millikan *et al.*, 2000). Lipid-adjusted concentrations of 35 PCB congeners were measured in plasma, but detailed analyses were presented only for total PCBs. Odds ratios were adjusted for age and age squared, and additionally for race, menopausal status, BMI, parity/lactation, hormone replacement therapy, and income, depending on the stratification factors. Results were presented in strata of race, parity plus lactation, BMI and history of farming. Risk of cancer of the breast was increased with total PCB exposure among African-American women (third tertile OR, 1.74; 95% CI, 1.00–3.01), but not among white women (third tertile OR, 1.03; 95% CI, 0.68–1.56). This risk was particularly high for African-Americans with BMI > 34.2 (third tertile total PCBs, OR, 4.92; 95% CI, 1.63–14.83). [This was a large, high-quality study, and included African-Americans.]

In the same study population as Millikan *et al.* (2000), Li *et al.* (2005) investigated *CYP1A1* polymorphisms and their interaction with PCB exposure in relation to risk of cancer of the breast among the 612 cases and 599 controls who had provided blood. Results showed no evidence of joint effects between *CYP1A1* M1-containing genotypes and total PCBs for either race. Among white women, statistically significant multiplicative interactions were observed between *CYP1A1* M2-containing genotypes and total PCBs (P = 0.02), but the association between PCBs and cancer of the breast was inverse. A multiplicative interaction was suggested among African-American women between *CYP1A1*

M3-containing genotypes and total PCBs, with an odds ratio of 1.6 (95% CI, 0.8–3.2) for women with total plasma PCB concentrations ≥ 0.430 ng/mL and any *CYP1A1* M3 genotype compared with lower PCB concentration and no M3 genotype (*P* for interaction = 0.10). [This large study was able to assess interactions with *CYP1A1*.]

In a case–control study conducted in 1994–7 in Quebec City, Canada, plasma concentrations of 14 PCB congeners were measured in 314 women with cancer of the breast and 523 controls (219 hospital controls, 304 population controls) (Demers *et al.*, 2002). Analyses in relation to cancer of the breast excluded five congeners that were detected in < 70% of the women. The remaining PCB congeners were correlated (Pearson correlation coefficients, 0.29 to 0.96). Risk of cancer of the breast was associated with the highest quartile of concentration of PCB-118 (OR, 1.60; 95% CI, 1.01–2.53) and PCB-156 (OR, 1.80; 95% CI, 1.11–2.94). Among the subgroup of premenopausal women, the odds ratio for the highest quartile of concentration of PCB-118 was 2.87 (95% CI, 1.13–7.31), and for PCB-156 it was 2.90 (95% CI, 1.18–7.15). No significant increase in risk was seen in postmenopausal women. When PCB-105, PCB-118 and PCB-156 were grouped, higher concentration was associated with increased risk of cancer of the breast (OR, 2.02; 95% CI, 1.24–3.28), but the PCBs that were the most abundant (PCB-138, PCB-153 and PCB-180) were not associated with risk of cancer of the breast. An earlier publication from this study investigated associations between organochlorine compounds and cancer of the breast, specifically in relation to axillary-lymph-node involvement and tumour size (Demers *et al.*, 2000). PCB-153 was selected as a surrogate for all PCB congeners because it was the most abundant in plasma samples and was strongly correlated with other prevalent congeners ($r \geq 0.72$; $P < 0.0001$). The relative risk of having a tumour size ≥ 2 cm was increased, but not significantly, with increasing plasma concentration of PCB-153. However, a higher concentration of PCB-153 was significantly associated with increased risk among those with axillary lymph-node involvement (OR, 2.12; 95% CI, 1.05–4.30, adjusted for confounders) and when tumour size > 2 cm and node involvement were considered together, (OR, 3.51; 95% CI, 1.41–8.73), with an exposure–response trend. [This was a well-designed and well-implemented study with two control groups and stratification for menopausal status.]

In a large population-based case–control study of environmental exposures and cancer of the breast conducted in 1996–7 on Long Island, NY, USA, serum concentrations of 24 PCB congeners were measured for 646 cases and 429 controls, with results presented for the four most commonly occurring congeners (PCB-118, PCB-138, PCB-153 and PCB-180) (Gammon *et al.*, 2002). There was no association between cancer of the breast and the sum concentration of the four PCBs, or any specific congener, and there was no effect of lactation, menopausal status, stage of disease, or hormone receptor status. [This was a large, well-designed and well-implemented study.]

In a population-based case–control study of African-American women, serum concentrations of PCBs [congeners not specified] were measured in 355 cases and 327 controls (Gatto *et al.*, 2007). Risk of cancer of the breast was not associated with total PCBs (OR comparing highest with lowest quintile, 1.01; 95% CI, 0.64–1.63), and BMI, parity, breastfeeding, and menopausal status did not modify the measures of effect. PCBs were not associated with an increase in the risk of any subtype of cancer of the breast as defined by PR, ER, *p53*, or HER-2/*neu* status. [Statistical power was limited for subgroup analyses.]

In a hospital-based case–control study of cancer of the breast in Nagano, Japan, including 403 matched pairs collected from 2001 to 2005, serum concentrations of total PCBs were associated with decreased risk of cancer of the

breast for the highest versus lowest quartile of concentration of total PCBs (OR, 0.33; 95% CI, 0.14–0.78) (Itoh *et al.*, 2009). For the specific congeners PCB-153 and PCB-180, the odds ratios were 0.40 (95% CI, 0.18–0.91) and 0.29 (95% CI, 0.13–0.66), respectively. The trend in the inverse relationship persisted when results were stratified by hormone-receptor and menopausal status. [The Working Group was not able to explain the inverse associations reported in this study.]

(c) *Larger studies of PCB concentrations in blood and breast adipose tissue*

Five publications from a research group in Connecticut, USA, were informative, although their potential overlap was not clear. In 1994–1997, 304 cases of cancer of the breast and 186 controls aged 40–79 years were recruited and breast adipose tissue was analysed for nine PCB congeners (PCB-74, PCB-118, PCB-138, PCB-153, PCB-156, PCB-170, PCB-180, PCB-183 and PCB-187) (Zheng *et al.*, 2000a). Age- and lipid-adjusted risk estimates were null in relation to total PCBs, PCB groups, and any of the congeners. Stratification by type of breast disease, menopausal status, parity, lactation, and body size showed null associations with concentrations of PCBs. From the same study population, Holford *et al.* (2000) calculated risk in relation to both linear logistic and logistic ridge regression analyses for nine PCB congeners by incremental (10 ng/g) changes in exposure: PCB-153 and PCB-156 were associated with decreased risk and PCB-180 and PCB-183 were associated with increased risk of cancer of the breast. In analyses using ridge regression and adjusting for covariates, no congeners remained associated with cancer of the breast. In another case–control study from this research group, subjects were recruited in 1995–1997 (overlap in years of study with Zheng *et al.*, 2000a): 475 incident cases of cancer of the breast were included, and 502 controls were randomly selected from the population or from patients with newly diagnosed benign breast disease at the same hospital (Zheng *et al.*, 2000b). Serum concentrations of nine PCB congeners were determined. After adjustment for confounding factors, all odds ratios were null. A related study focused on the potential interaction between CYP1A1 and lipid-adjusted serum concentrations of PCBs on risk of cancer of the breast among Caucasian women recruited in 1999–2002, with 374 cases and 406 controls (Zhang *et al.*, 2004). The odds ratio for high exposure (> 610 ng/g) to PCBs was 1.2 (95% CI, 0.9–1.6). With respect to *CYP1A1* genotype, the risks associated with higher serum concentration of total PCBs was highest for carriers of the *m2* variant genotype both among all women combined (OR, 3.6; 95% CI, 1.5–8.2), and in postmenopausal women (OR, 4.3; 95% CI, 1.6–12.0). No significant association was reported for *CYP1A1 m1* or *m4* genotypes or among premenopausal women. Finally, in another publication on a subset of 266 cases of cancer of the breast and 347 controls with benign breast disease, there was no association for total subjects, adjusted for standard risk factors, between cancer of the breast by joint ER/PR status and serum concentrations of total PCBs and adipose-tissue concentrations of nine PCB congeners (Rusiecki *et al.*, 2004). However, among postmenopausal women, increased risk of cancer of the breast was seen in relation to increased concentrations of PCB-183 among women with ER+PR+ tumours (third versus first tertile, OR, 2.4; 95% CI, 1.0–6.0; *P* for trend = 0.03). [While there appeared to be overlap between this group of studies from Connecticut, the extent of the overlap was difficult to determine, therefore the independence of the findings was not known. Controls were drawn from a mix of hospital and population sources, and the impact of this selection method was difficult to gauge. The large number of subgroup analyses, particularly in the study by Rusiecki *et al.* (2004), which presented 80 odds ratios, increased the probability of chance findings.]

(d) Larger studies of PCB concentrations in adipose tissue

On Long Island, New York, USA, concentrations of 14 PCB congeners in adipose tissue did not differ for 232 women with cancer of the breast and 323 hospital controls with benign breast disease or non-breast-related conditions, after adjustment for age, race, and BMI (Stellman *et al.*, 2000). No increase in risk was observed for total PCBs, but congeners PCB-156 and PCB-183 were associated with significantly increased risk (OR, 1.9; 95% CI, 1.1–3.0 for the second tertile of exposure distribution for PCB-156; and OR, 2.0; 95% 1.2–3.4 for the highest tertile of PCB-183). No other congener was associated with risk of cancer of the breast, and no clear difference in risk was seen for ER+ and ER– tumours. [This was a large, well-designed study, but results were only presented for total PCBs and two congeners.]

In a case–control study in Kingston and Toronto, Ontario, Canada, noncancerous breast adipose tissue collected before treatment from 217 incident cases of cancer of the breast and 213 controls undergoing biopsy was analysed for 14 PCB congeners (Aronson *et al.*, 2000). PCB-105 and PCB-118 were associated consistently with risk of cancer of the breast after adjusting for other factors (OR, 3.17; 95% CI, 1.51–6.68; and OR, 2.31; 95% CI, 1.11–4.78, respectively, for the fourth versus first quartile of the exposure distribution) and these effects increased monotonically. PCB-138 was also associated consistently with increased risk, but the odds ratios were imprecise. Stronger associations were apparent among premenopausal women (PCB-105: OR, 3.91; 95% CI, 1.73–8.86; and PCB-118: OR, 2.85; 95% CI, 1.24–6.52, for the highest exposure category). Among postmenopausal women, risks associated with PCB-170 and PCB-180 were also elevated in the second of three exposure groups (OR, 3.27; 95% CI, 1.44–7.44; and OR, 2.43; 95% CI, 1.09–5.43, respectively), but declined below significance in the highest group (ORs 1.63 and 1.77, respectively). No other PCB congener was significantly associated with risk. Although the odds ratios did not differ significantly by subtype of cancer, the odds ratios for total PCBs were higher for ER– than for ER+ cancer of the breast (Woolcott *et al.*, 2001). Investigation of specific genotype–PCB interactions among 68 cases and 52 controls with blood samples in this study showed increased risk of cancer of the breast for *CYP1A1* M1 wildtype homozygotes with high exposure to PCB-105 (OR, 3.20; 95% CI, 1.14–8.98) (McCready *et al.*, 2004). [This was a large, well-designed study.]

(e) Exposure estimates from occupational or dietary histories

A few case–control studies have estimated PCB exposures from occupational or dietary histories.

A population-based case–control study in Massachusetts, USA, included 261 incident cases of cancer of the breast diagnosed between 1983 and 1986 and 753 controls. The subjects were interviewed to ascertain all full-time jobs held since age 18 years. Probable exposure to PCBs was associated with non-significant increases in the risk of cancer of the breast (adjusted OR, 3.2; 95% CI, 0.8–12.2; five exposed cases and six exposed controls) (Aschengrau *et al.*, 1998). [The Working Group noted imprecise findings.]

Consumption of fish from the Great Lakes as a source of exposure to PCBs was investigated as a potential risk factor for cancer of the breast in a population-based case–control study in Wisconsin, USA (McElroy *et al.*, 2004). There were 1481 cases aged 20–69 years, diagnosed in 1998–2000 in the Wisconsin Cancer Reporting System, and 1301 controls of similar age were randomly selected from licensed drivers and Medicare lists; telephone interviews were used to obtain information on consumption of all sport-caught (Great Lakes and other lakes) fish and risk factors for cancer of the breast. After adjustment for risk factors, including age, education, weight,

alcohol consumption, reproductive history, and family history of cancer of the breast, no association was found between risk of cancer of the breast and recent consumption of sport-caught fish (OR, 1.00; 95% CI, 0.86–1.17), recent consumption of fish from the Great Lakes (OR, 1.06; 95% CI, 0.84–1.33), or the number of fish meals per year. Menopausal status appeared to be an effect modifier, with recent consumption of fish from the Great Lakes not associated with postmenopausal cancer of the breast (OR, 0.78; 95% CI, 0.57–1.07), but with premenopausal breast cancer (OR, 1.70; 95% CI, 1.16–2.50). [This was a large study with exposure assessment that used consumption of sport fish as a proxy for PCB exposure, but did not use biomarkers.]

(f) Combined analysis of five studies in the USA

The results of five case–control studies in the north-east USA conducted before 2000 (of which three are nested in cohort studies) (Moysich *et al.*, 1998; Helzlsouer *et al.*, 1999; Laden *et al.*, 2001a, b and Hunter *et al.*, 1997; Zheng *et al.*, 2000a, b; Wolff *et al.*, 2000b) and in which plasma or serum concentrations of PCBs were measured have been combined into an analysis of 1400 cases and 1642 controls using a standardized approach to confounder and effect-modification assessment, and a random-effects model to estimate associations (Laden *et al.*, 2001b). For women in the fifth quintile of lipid-adjusted values compared with those in the first quintile, the multivariate pooled odds ratio for cancer of the breast associated with the sum of PCBs (PCB-118, PCB-138, PCB-153 and PCB-180) was 0.94 (95% CI, 0.74–1.21). No consistent increase in risk was observed in subgroups defined by parity or lactation. [This combined analysis focused on the most prevalent PCBs that were analysed in all five studies; while this enhanced precision for the overall relationship, it did not show associations for specific PCB congeners and PCB subgroups. The Working Group noted that several informative studies were published after this combined analysis.]

2.3.3 Cancer of the prostate

Several epidemiological studies have investigated possible associations between cancer of the prostate and exposure to PCBs. These studies differed in study design (i.e. case–control studies, nested case–control studies) and in the assessment of PCBs (i.e. job-exposure matrices, measurement of PCB concentrations in blood or adipose tissue).

Seidler *et al.* (1998) described the results of a case-referent study including 192 patients with cancer of the prostate and 210 controls from medical practices or clinic in Germany. Occupational exposure to PCBs was estimated using a British job-exposure matrix (Pannett *et al.*, 1985). Most subjects had no or low exposure to PCBs and no association between exposure and risk of cancer of the prostate was reported. [Due to the relative low participation rate among controls (55%), selection bias could not be excluded. Furthermore, the validity of the job-exposure matrix was unknown and significant exposure misclassification could not be ruled out.]

Ritchie *et al.* (2003, 2005) conducted a hospital-based case–control study in Iowa, USA, in which 30 PCB congeners were measured in serum samples from 58 patients with cancer of the prostate and 99 age-matched controls. Odds ratios were elevated for total PCBs, and for PCB-153, and PCB-180. A monotonic, not statistically significant, exposure–response trend was observed for total PCBs. For PCB-180, the odds ratio was significantly increased (OR, 3.13; 95% CI, 1.33–7.34) only in the middle (but not the highest) category of exposure. [This study was small with multiple comparisons.]

In a population-based case–control study in Sweden, Hardell *et al.* (2006a) compared

concentrations of 37 PCB congeners in samples of fat tissue from 58 cases of cancer of the prostate and 20 controls with benign prostate hyperplasia. The odds ratio for the sum of PCBs and cancer of the prostate was 1.21 (95% CI, 0.42–3.50) in all men. PCB-153 was associated with an increased risk of cancer of the prostate (OR, 3.15; 95% CI, 1.04–9.54). Stronger associations were observed in men with prostate-specific antigen (PSA) > 16.5 ng/mL; the odds ratio was 1.91 (95% CI, 0.55–6.55) for total PCBs, and risks for enzyme and phenobarbital-inducing PCBs (Wolff *et al.*, 1997) and for less chlorinated PCBs (Moysich *et al.*, 1999a) were significantly increased in this subgroup of men. [This study was small and involved multiple comparisons.]

Aronson *et al.* (2010) conducted a case–control study among urology patients in Ontario, Canada. Concentrations of 14 PCB congeners were measured in serum of 79 men with incident cancer of the prostate and 329 age-matched controls. No association was observed between concentrations of individual PCB congeners or the sum of PCBs, and the risk of prostate cancer. [As both cases and controls underwent the same diagnostic procedures and were screened by PSA and digital rectal examination, selection bias was unlikely in this study].

2.3.4 Melanoma

(a) Cutaneous malignant melanoma

See Table 2.11

Gallagher *et al.* (2011) conducted a case–control study of 80 patients with malignant melanoma of the skin and 310 controls. The cases were part of a larger case–control study and were originally recruited to evaluate the effect of exposure to ultraviolet (UV) light and gene variants on risk of melanoma, and the controls were recruited using population-based registries. Lipid-adjusted plasma concentrations of 14 PCB congeners were determined and data were reported for 8, as well as for total PCBs, and dioxin-like and non-dioxin-like PCBs. Statistically significant associations with malignant melanoma were observed for the highest compared with the lowest quartile for: total PCBs (OR, 6.02; 95% CI, 2.0–18.17); summed non-dioxin-like PCBs (OR, 7.02; 95% CI, 2.30 –21.43); summed dioxin-like PCBs (OR, 2.84; 1.01–7.97), and all of the individual PCB congeners examined (PCB-118, PCB-138, PCB-153, PCB-156, PCB-170, PCB-180, PCB-183 and PCB-187). [The Working Group considered that, in light of its appropriate design and control of relevant potential confounders, this was a high-quality study, despite the relatively small sample size and being described as "preliminary" by the authors. The positive associations for all the individual PCB congeners may have been a result of correlations among congeners. Multiple comparisons were not formally addressed, but it is likely that adjustment for multiple comparisons would not change the interpretation of the results.]

(b) Uveal melanoma

See Table 2.11

In a multicentric case–control study in nine European countries, Behrens *et al.* (2010) investigated the association between risk of uveal melanoma and exposure to PCBs. The 293 men and women with uveal melanoma were frequency-matched to 3198 population and hospital controls by country, age, and sex. Exposure to transformer oils was assessed by questionnaire, with exposures to PCBs classified as "potential" or "confirmed," depending on whether subjects reported exposure to a named brand of oil with known PCB content. Analyses were adjusted for age, country, eye colour, and history of ocular damage from ultraviolet light. Only men reported exposure to transformer/capacitor oils. The odds ratio for any exposure was 2.74 (99.3% CI, 1.07–7.02), and was similar in magnitude for men with more than 10 years of exposure and for "confirmed" exposure. For exposure to Pyralene (the most frequently reported PCB-containing

Table 2.11 Case–control studies on melanoma and exposure to PCBs

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Cutaneous malignant melanoma							
Gallagher *et al.* (2011) British Columbia, Canada, 2000–2004	80 310	Population	Lipid-adjusted concentrations of 14 PCBs[a] (units NR)				Age, sex, education, skin reaction to repeated sun exposure, and total recreational sun exposure
				Total PCBs			
				98.01–148.71	11	1.36 (0.45–4.09)	
				148.72–213.44	12	1.27 (0.39–4.12)	
				> 213.44	29	6.02 (2.00–18.17)	*P* for trend < 0.001
				DL-PCBs			
				9.37–15.10	8	0.31 (0.10–0.98)	
				15.11–22.57	16	1.16 (0.41–3.26)	
				> 22.57	25	2.84 (1.01–7.97)	*P* for trend = 0.003
				NDL-PCBs			
				86.68–133.66	12	2.05 (0.66–6.39)	
				133.67–192.39	11	1.19 (0.36–3.90)	
				> 192.39	30	7.02 (2.30–21.43)	*P* for trend < 0.001
				PCB-118			
				> 4.90–8.16	13	0.89 (0.34–2.34)	
				> 8.16–13.32	14	1.13 (0.40–3.23)	
				> 13.32–46.19	23	3.04 (1.05–8.74)	*P* for trend = 0.012
				PCB-138			
				> 12.79–20.76	19	1.89 (0.68–5.28)	
				> 20.76–30.65	8	1.30 (0.37–4.56)	
				> 30.65–104.49	28	4.91 (1.69–14.32)	
				PCB-153			
				> 27.75–42.07	14	2.01 (0.70–5.77)	
				> 42.07–60.43	12	1.35 (0.43–4.25)	
				> 60.43–735.90	27	4.86 (1.68–14.08)	*P* for trend = 0.002
				PCB-156			
				> 4.09–6.07	13	1.04 (0.36–2.97)	
				> 6.07–8.65	13	1.48 (0.49–4.45)	
				> 8.65–113.32	29	4.22 (1.51–11.78)	*P* for trend = 0.001

Table 2.11 (continued)

Reference, study location and period	Total No. cases Total No. controls	Control source (hospital, population)	Exposure assessment	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates Comments
Gallagher et al. (2011) British Columbia, Canada, 2000–2004 (cont.)				*PCB-170*			
				> 7.97–12.16	13	1.50 (0.53–4.29)	
				> 12.16–18.51	13	1.10 (0.32–3.77)	
				> 18.51–901.52	29	4.60 (1.60–13.22)	*P* for trend = 0.001
				PCB-180			
				> 25.20–38.16	12	1.46 (0.49–4.37)	
				> 38.16–59.40	14	1.55 (0.44–5.43)	
				> 59.40–3786.60	30	5.89 (1.87–18.50)	*P* for trend = 0.001
				PCB-183			
				> 1.87–84.86	54	4.27 (1.71–10.68)	
				PCB-187			
				> 6.64–10.45	11	2.54 (0.75–8.58)	
				> 10.45–16.10	15	2.56 (0.76–8.62)	
				> 16.10–833.15	30	11.47 (3.32–39.68)	*P* for trend < 0.001
Uveal melanoma							
Behrens *et al.* (2010), Denmark, France, Germany, Italy, Latvia, Portugal, Sweden, Spain, UK Jan 1994–Dec 1997	293 3198	Hospital and population	Questionnaire on occupational exposure	Exposure to oils potentially containing PCBs			Country, age, ocular damage due to UV, eye colour, exposure to high-voltage installations ORs were Bonferroni-corrected for seven independent tests to control for multiple comparisons, thus all CI are 99.3%. Response rates: cases, 84%; hospital, 84%; population controls, 61% Only men were exposed to oils
				Never exposed	150	1.00	
				Ever exposed	6	2.74 (0.72–10.37)	
				Duration > 10 years	2	2.62 (0.29–24.06)	
				"Confirmed" exposure to PCB oil	4	2.61 (0.54–12.63)	
				Exposure to Pyralene	4	6.43 (1.17–35.30)	

[a] The 14 PCB congeners were PCBs 28, 52, 99, 101, 105, 118, 128, 138, 153, 156, 170, 180, 183, and 187.

CI, confidence intervals; DL-PCB, dioxin-like PCB; NDL-PCB, non-dioxin-like PCB; OR, odds ratio; PCB, polychlorinated biphenyl; UK, United Kingdom; UV, ultraviolet

oil), the odds ratio was 6.43 (99.3% CI, 1.17–35.30; four cases). [This study was notable in being the only large study of a rare cancer. Multiple comparisons were addressed via adjusted 99.3% confidence intervals, but exposure was rare and estimates were imprecise.]

2.3.5 Other cancers

(a) Urothelial cancer

Steineck *et al.* (1990) carried out a population-based case-referent study of urothelial cancer in men in Stockholm, Sweden. Occupational exposures to PCBs and several other agents were assigned by an industrial hygienist. The adjusted odds ratio for estimated exposure to PCBs was 3.3 (95% CI, 0.6–18.4). [The precision of this study was quite limited and the definition of the cancer sites was broad.]

(b) Cancer of the testis

Hardell *et al.* (2003) analysed 38 PCB congeners in blood samples collected from 61 incident cases of cancer of the testis and 58 age-matched controls from the Swedish population registry. No association between cancer of the testis and the sum of PCB concentrations in blood was found. Mothers of 44 cases and 45 controls also provided blood samples; significantly higher PCB concentrations were found for mothers of cases compared with mothers of controls (OR, 3.8; 95% CI, 1.4–10). A difference in the sum of PCBs between mothers of cases and mothers of controls was also reported in two subsequent publications by the same authors (Hardell *et al.*, 2004, 2006b). [Due to the timing of blood collection of the mothers, which was decades after the cases' births, the interpretation of these results was difficult. PCB concentrations in women may be affected by weight changes, child bearing, lactation, and subsequent exposure. Thus it could not be assumed that the concentrations measured in women at the time of the study were representative of their sons' exposures in utero.]

(c) Cancer of the lung

Recio-Vega *et al.* (2012) investigated the association between PCB concentrations, *CYP1A1* polymorphisms and the risk of cancer of the lung in a case–control study in northern Mexico including 43 cases of cancer of the lung and 86 controls without cancer who were recruited from two hospitals. Information including history of exposure to PCBs was collected through in-person interview and 20 PCB congeners were measured in serum. Odds ratios were adjusted for age, agricultural occupation, and tobacco smoking. There was a significant association between PCB-18 and cancer of the lung (OR, 1.13; 95% CI, 1.04–1.38). Odds ratios for PCB-52, PCB-118, and PCB-170 were similar in magnitude, but did not reach statistical significance, while odds ratios for other congeners were close to unity. *CYP1A1* polymorphism was not associated with serum concentrations of total PCBs. [The Working Group noted that this study provided information about less chlorinated PCBs, which are rarely measured; however, the etiological relevance of measurements of PCBs of short half-life was questionable. In addition, the methods used for subject recruitment and for statistical analysis were not clearly described, and the possibility of residual confounding by age was noted.]

(d) Cancer of the colorectum

Howsam *et al.* (2004) assessed associations between cancer of the colorectum and exposure to PCBs and gene–environment interactions in 132 cases and 76 controls sampled from a larger hospital-based case–control study in Barcelona, Spain. Serum concentrations of PCB-28, PCB-52, PCB-101, PCB-118, PCB-138, PCB-153, and PCB-180 were measured. Point mutations in *K-ras* and *p53* genes and expression of p53 protein were assessed in tumour tissue. PCB-28 and PCB-118 were significantly associated with an increased risk of cancer of the colorectum (ORs, 2.75;

95% CI, 1.29–5.83; and 2.02; 95% CI, 1.00–4.08, respectively), for the more exposed category. A statistically significant exposure–response trend was observed for the mono-*ortho* PCB group that combined PCB-28 and PCB-118 (*P* for trend = 0.004). Odds ratios for the other PCBs were not consistently or significantly increased. No significant interaction of mono-*ortho* PCBs with *p53* or *K-ras* mutations was found. [The use of controls representing several diagnostic groups and control for potential confounding factors were strengths of this study. However, the case definition combining cancers of the colon and rectum may mix diseases with potentially different etiologies.]

(e) Cancer of the pancreas

In a population-based case–control study of cancer of the pancreas in the San Francisco area, USA, Hoppin *et al.* (2000) analysed 11 PCB congeners in serum samples from 108 cases of cancer of the pancreas and 82 controls matched by sex and age-group. Total lipid-adjusted PCB concentrations were estimated using the sum of all congeners. A statistically significant dose–response relationship ($P < 0.001$) was observed for total PCBs, with an odds ratio of 4.2 (95% CI, 1.8–9.4) for ≥ 360 versus < 185 ng/g. Significantly elevated odds ratios were also observed for the highest tertiles of PCB-153 (OR, 3.0; 95% CI, 1.4–6.6) and PCB-180 (OR, 8.4; 95% CI, 3.4–21). Odds ratios remained elevated after adjusting for dichlorodiphenyldichloroethylene (DDE) content, and in a sensitivity analysis of the effects of bioconcentration. [A strength of the study was that the issue of confounding by bioconcentration in fat due to adipose-tissue loss was addressed. Nevertheless, the small number of subjects limited a clear interpretation of the results.]

(f) Cancer of the biliary tract

Ahrens *et al.* (2007) investigated the association between cancer of the extrahepatic biliary tract and occupational exposure to endocrine-disrupting compounds in a European multicentre case–control study of 183 men with histologically confirmed carcinoma of the extrahepatic biliary tract and 1938 matched controls. Self-reported job descriptions were converted to semiquantitative indicators of occupational exposure to 14 types of suspected endocrine-disrupting compounds, including PCBs, hormones, phthalates, and pesticides. Odds ratios were adjusted for age, country, and history of gallstones. The adjusted odds ratio for cancer of the extrahepatic biliary tract and ever-exposure to PCBs was 2.8 (95% CI, 1.3–5.9). When exposure intensity was analysed, the highest odds ratio was observed in the low-intensity category. [These results were based on a small number of exposed cases and trends were inconsistent.]

(g) Childhood cancer

Ward *et al.* (2009) conducted a population-based case–control in California, USA of 184 children aged 0–7 years with acute lymphocytic leukaemia and 212 controls from birth certificates matched by birth date, sex, race, and ethnicity. Concentrations of six PCB congeners in residential carpet dust were used as an exposure indicator. The odds ratio for detection of any PCB in dust was 1.97 (95% CI, 1.22–3.17) and the odds ratio for the highest quartile of total PCBs compared with the lowest was 2.78 (95% CI, 1.41–5.48). Significant exposure–response trends were reported for PCB-118, PCB-138 and PCB-153. [The study was well-designed and the method of exposure assessment used was a strength. The authors were able to rule out confounding by several organochlorine pesticides. The Working Group was unable to replicate the *P* values for trend tests.]

(h) Cancer of the endometrium

Sturgeon *et al.* (1998) conducted a multicentric hospital-based case–control study of cancer of the endometrium in five areas of the USA. Serum concentrations of 27 PCB congeners were measured for 90 individually matched case–control pairs. No associations were observed between elevated serum concentrations of several PCB groups, including total PCBs and potentially estrogenic PCBs, and risk of cancer of the endometrium. [The results did not appear to be affected by selection bias, but precision was limited.]

Weiderpass *et al.* (2000) measured serum concentrations of 10 PCB congeners in a population-based case–control study of 154 cases of cancer of the endometrium and 205 controls in Sweden. After adjustment there was no increase in risk associated with high concentrations of any of the congeners evaluated, and there were no significant trends in risk. [The power of this study was limited due to the small number of subjects. However, selection bias was unlikely, as the main reason for non-participation was the failure of the hospital staff to collect blood samples before surgery.]

Hardell *et al.* (2004) conducted a hospital-based case–control study with 76 cases and 39 controls to evaluate the risk of cancer of the endometrium associated with environmental endocrine disruptors. Concentrations of 37 PCB congeners were measured in adipose tissue. No association was found for the sum of PCBs or for any grouping of PCBs by structure or activity. [The power of this study was limited due to the small number of subjects.]

(i) Cancer of the male breast

Occupational risk factors for cancer of the male breast were investigated in a multicentric study of 104 cases and 1901 controls in eight European countries (Villeneuve *et al.*, 2010). Lifetime work history was obtained by in-person interviews, and potential occupational exposures including to PCBs were assessed using expert judgment. Results were reported for PCBs and dioxins combined, for which the fully-adjusted odds ratio was 1.6 (95% CI, 0.7–3.7). [This study had limited power to detect excess risk.]

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3. CANCER IN EXPERIMENTAL ANIMALS

In previous evaluations in 1978, 1979, 1987, and 2012 (IARC, 1978, 1979, 1987, 2012), the Working Group concluded that there was *sufficient evidence* in experimental animals for the carcinogenicity of polychlorinated biphenyls (PCBs). New data have since become available, and these have been taken into account in the present evaluation.

3.1 Oral administration

See Table 3.1 and Table 3.2

3.1.1 Individual PCBs and binary mixtures

The United States National Toxicology Program (NTP) has conducted a series of studies to evaluate the carcinogenicity of some PCB congeners administered alone or as binary mixtures in female Harlan Sprague-Dawley rats treated by gavage.

(a) PCB-126

Rat

Groups of 81 female Harlan Sprague-Dawley rats (age, 8 weeks) were given the dioxin-like congener PCB-126 at a dose of 0 (vehicle control), 30, 100, 175, 300, 550, or 1000 ng/kg body weight (bw) by gavage in corn oil : acetone (99 : 1), 5 days per week, for up to 104 weeks (core study) (Brix *et al.*, 2004; Nyska *et al.*, 2004; Walker *et al.*, 2005; Yoshizawa *et al.*, 2005, 2007, 2009; NTP, 2006a). Ten rats per group were evaluated at 14, 31, or 53 weeks. A stop-exposure group of 50 female rats was given PCB-126 at a dose of 1000 ng/kg bw in corn oil : acetone (99 : 1) by gavage for 30 weeks, then the vehicle only for the remainder of the study. There were treatment-related increases in the incidences of cholangiocarcinoma and hepatocellular adenoma in rats treated with PCB-126 at doses of 300 ng/kg bw or higher, and 550 ng/kg bw or higher, respectively, for up to 104 weeks. There were three hepatocholangiomas in the group at 1000 ng/kg bw, and single incidences of cholangioma in the groups at 550 and 1000 ng/kg bw. [These tumours are rare, and it was uncertain whether they were related to treatment.] There were also statistically significant, dose-related increases in the incidences of a spectrum of non-neoplastic lesions that collectively were diagnosed as toxic hepatopathy. Significant increases in the incidence of cystic keratinizing epithelioma of the lung occurred in rats at 550 ng/kg bw or higher, and non-statistically significant low incidences of squamous cell carcinoma of the lung were also observed at the highest doses in the core-study groups. Gingival squamous cell carcinomas were observed in all exposure groups, and incidence was significantly increased in the group at 1000 ng/kg bw (core study). Adenomas and/or carcinomas were present in the adrenal cortex of rats in most groups, including the stop-exposure group, with a positive trend in the incidence of adenoma or carcinoma (combined) with increasing dose.

Table 3.1 Studies of carcinogenicity in rats exposed to PCBs and related compounds

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Individual PCBs and binary mixtures				
Harlan Sprague-Dawley (F) 104 wk NTP (2006a)	Core study: PCB-126 in corn oil : acetone (99 : 1) by gavage at doses of 0, 30, 100, 175, 300, 550, or 1000 ng/kg bw, 5 days/wk, for 104 wk 81 rats/group Stop-exposure study: PCB-126 at 1000 ng/kg for 30 wk followed by vehicle for the remainder of the study 50/group Interim evaluations: 10 rats per core study group were evaluated at wks 14, 31, and 53	*Liver* Cholangiocarcinoma (includes multiple): 0/53, 0/55, 1/53, 0/53, 5/53, 6/51, 22/53; 0/53, 2/50 (stop-exposure) Multiple: 0/53, 0/55, 0/53, 0/53, 0/53, 4/51, 15/53; 0/53, 0/50 (stop-exposure) Hepatocellular adenoma[a] (includes multiple): 1/53, 2/55, 1/53, 0/53, 2/53, 4/51, 7/53; 1/53, 0/50 (stop-exposure) Multiple: 0/53, 0/55, 0/53, 0/53, 0/53, 0/51, 1/53; 0/53, 0/50 (stop-exposure) Hepatocholangioma[b] (includes multiple): 0/53, 0/55, 0/53, 0/53, 0/53, 0/51, 3/53; 0/53, 0/50 (stop-exposure) Multiple: 0/53, 0/55, 0/53, 0/53, 0/53, 0/51, 1/53; 0/53, 0/50 (stop-exposure) Cholangioma[b]: 0/53, 0/55, 0/53, 0/53, 0/53, 1/51, 1/53; 0/53, 0/50 (stop-exposure) *Lung* Cystic keratinizing epithelioma (includes multiple): 0/53, 0/55, 0/53, 0/53, 1/53, 11/51**, 35/51*; 0/53, 0/50 (stop-exposure) Multiple: 0/53, 0/55, 0/53, 0/53, 0/53, 8/51*, 30/51*; 0/53, 0/50 (stop-exposure) Squamous cell carcinoma: 0/53, 0/55, 0/53, 0/53, 0/53, 1/51, 2/51; 0/53, 0/50 (stop-exposure)	$P < 0.001$ (1000 ng/kg bw) $P < 0.001$ (trend) $P \leq 0.001$ (1000 ng/kg bw) $P = 0.033$ (1000 ng/kg bw) $P < 0.001$ (trend) $P < 0.001$ (trend) NS NS *$P < 0.001$ **$P = 0.002$ $P < 0.001$ (trend) *$P \leq 0.001$ NS	Purity, 99% The overall incidence values are presented, but statistical analyses are based on the poly 3 test used by NTP that takes survival differences into account *Non-neoplastic lesions* Liver: toxic hepatopathy that included hepatocyte hypertrophy and hyperplasia, bile duct and oval cell hyperplasia, nodular hyperplasia, cholangiofibrosis, multinucleated hepatocytes, diffuse fatty change, bile duct cyst, necrosis, pigmentation, inflammation, portal fibrosis Lung: squamous metaplasia, and bronchiolar metaplasia of the alveolar epithelium No tumours were observed at interim evaluations at wk 14 and 31

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Harlan Sprague-Dawley (F) 104 wk NTP (2006a) (cont.)		*Oral mucosa* Gingival squamous cell carcinoma[c]: 0/53, 1/55, 1/53, 1/53, 2/53, 2/53, 7/53*; 0/53, 2/50 (stop-exposure) *Adrenal cortex* Adenoma: 0/52, 1/55, 1/53, 0/53, 0/53, 1/52, 2/53; 0/52, 2/50 (stop-exposure) Carcinoma: 0/52, 1/55, 0/53, 0/53, 1/53, 0/52, 2/53; 0/52, 1/50 (stop-exposure) Adenoma or carcinoma (combined): 0/52, 2/55, 1/53, 0/53, 1/53, 1/52, 4/53; 0/52, 3/50 (stop-exposure)	*P = 0.010 P < 0.001 (trend) NS NS P = 0.022 (trend)	
Harlan Sprague-Dawley (F) 105 wk NTP (2006b)	Core study: PCB-153 in corn oil : acetone (99 : 1) by gavage at doses of 0, 10, 100, 300, 1000 or 3000 µg/kg bw by gavage, 5 days/wk for 105 wk 80–82 rats/group Stop-exposure study: 3000 µg/kg bw for 30 wk followed by vehicle for the remainder of the study 50/group Interim evaluations: 10 rats per core study group were evaluated at 14, 31, and 53 wk	*Liver* Cholangioma: 0/53, 0/54, 0/53, 0/53, 2/53, 0/51; 0/53, 2/50 (stop-exposure) Hepatocellular adenoma: 0/53, 0/54, 0/53, 0/53, 0/53, 1/51; 0/53, 0/50 (stop-exposure) *Thyroid gland* Follicular cell adenoma: 0/51, 0/52, 0/53, 0/53, 0/53, 0/51; 0/51, 2/49 (stop-exposure) *Interim evaluation (wk 53)* *Thyroid gland* Follicular cell adenoma: 0/10, 0/10, 1/10, 0/10, 0/10, 0/10	NS NS NS NS	Purity, 99% *Non-neoplastic lesions* Liver: hepatocyte hypertrophy, bile duct hyperplasia, oval cell hyperplasia, fatty change and pigmentation Thyroid gland: folicular cell hypertrophy Ovary and oviduct: chronic active inflammation Uterus: suppurative inflammation No tumours were observed at 14 and 31 wk

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Harlan Sprague-Dawley (F) 105 wk NTP (2010)	Core study: PCB-118 by gavage in corn oil : acetone (99 : 1) at doses of 0, 100, 220, 460, 1000 or 4600 µg/kg bw, by gavage 5 days/wk for 105 wk. 80/group Stop-exposure study: 4600 µg/kg bw for 30 wk followed by vehicle only for the remainder of the study 50/group Interim evaluations: 10 rats per core-study group were evaluated at 14, 31, and 53 wk	*Liver* Cholangiocarcinoma (includes multiple): 0/52, 0/51, 0/52, 0/52, 3/52, 36/49; 0/52, 29/49 (stop-exposure) Multiple: 0/52, 0/51, 0/52, 0/52, 0/52, 30/49; 0/52, 17/49 (stop exposure) Hepatocellular adenoma (includes multiple): 0/52, 1/51, 1/52, 4/52, 12/52, 24/49; 0/52, 1/49 (stop-exposure) Multiple: 0/52, 0/51, 0/52, 0/52, 4/52, 14/49; 0/52, 1/49 (stop-exposure) Hepatocellular carcinoma: 0/52, 0/51, 0/52, 0/52, 0/52, 1/49; 0/52, 0/49 (stop-exposure) Hepatocholangioma: 0/52, 0/51, 0/52, 0/52, 0/52, 4/49; 0/52, 0/49 (stop-exposure) *Lung* Cystic keratinizing epithelioma (includes multiple): 0/51, 0/52, 0/52, 0/52, 0/52, 20/50; 0/51, 0/50 (stop-exposure) Multiple: 0/51, 0/52, 0/52, 0/52, 0/52, 8/50; 0/51, 0/50 (stop-exposure) *Uterus* Carcinoma[d]: 2/52, 2/52, 1/52, 3/52, 4/52, 3/52; 2/52, 11/50 (stop-exposure)	$P < 0.001$ (4600 µg/kg and stop-exposure) $P < 0.001$ (trend) $P \leq 0.001$ (4600 µg/kg) $P < 0.001$ (1000 and 4600 µg/kg) $P < 0.001$ (trend) $P \leq 0.01$ (4600 µg/kg) NS $P < 0.001$ (trend) $P < 0.001$ (4600 µg/kg) $P < 0.001$ (trend) $P \leq 0.05$ (4600 µg/kg) $P = 0.014$ (stop-exposure)	Purity, > 99% PCB-118 was analysed for the presence of PCDDs, PCDFs, and PCBs; trace amounts of TCDD (0.000005%), TCDF (0.000005%), PCB-126 (0.0000170%), PCB-169 (0.0000003%) and various other PCB congeners were found. The calculated total non-PCB-118 TEQ contribution was 0.39 ng TEQ/1000 µg of PCB-118 bulk test article *Non-neoplastic lesions* Liver: toxic hepatopathy that included hepatocyte hypertrophy and hyperplasia, bile duct and oval cell hyperplasia, nodular hyperplasia, cholangiofibrosis, multinucleated hepatocytes, diffuse fatty change bile duct cyst, necrosis, pigmentation, inflammation, portal fibrosis Lung: alveolar epithelium, metaplasia; bronchiolar epithelium, squamous metaplasia Adrenal cortex: atrophy and hyperplasia Thyroid gland: follicular cell, hypertrophy Nose: respiratory epithelium, hyperplasia

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Harlan Sprague-Dawley (F) 105 wk NTP (2010) (cont.)		Squamous cell carcinoma: 0/52, 0/52, 3/52, 1/52, 1/52, 0/52; 0/52, 1/50 (stop exposure)	NS	
		Pancreas		Pancreas: acinus, cytoplasmic vacuolization
		Acinar adenoma: 0/52, 0/52, 0/52, 2/52, 3/52, 1/47; 0/52, 0/49 (stop-exposure)	NS	Nose: inflammation
		Acinar adenoma or carcinoma (combined): 0/52, 0/52, 0/52, 2/52, 3/52, 2/47; 0/52, 0/49 (stop exposure)	NS	Kidney: pigmentation No tumours were observed at interim evaluations at wk 14 and 31.
		Interim evaluation (wk 53)		
		Liver		
		Cholangiocarcinoma (includes multiple): 0/8, 0/8, 0/10, 0/8, 0/8, 3/8		
		Hepatocellular adenoma: 0/8, 0/8 0/10, 0/8, 0/8, 1/8		
Harlan Sprague-Dawley (F) 105 wk NTP (2006c)	*Constant-ratio study:* PCB-126 and PCB-153 as binary mixture with PCB-126 at doses of 0, 10, 100, 300, 1000 ng/kg bw per day, and PCB-153 at 0, 10, 100, 300, 1000 µg/kg bw per day in corn oil : acetone (99 : 1) by gavage *Varying-ratio study:* PCB-126 and PCB-153 as binary mixture at doses of PCB-126 at 300, 300, 300 ng/kg bw per day, and PCB-153 at 100, 300, 1000 µg/kg bw per day by gavage in corn oil : acetone 80–81/group *Interim evaluations:* 10 rats per core-study group were evaluated at wk 14, 31, and 53	*Constant-ratio study:* *Liver* Hepatocellular adenoma: 0/53, 0/53, 3/52, 5/52, 27/51*	*$P < 0.001$ $P < 0.001$ (trend)	Purity, > 99% (PCB-126 and PCB-153) *Non-neoplastic lesions* Liver: toxic hepatopathy that included hepatocyte hypertrophy and hyperplasia, bile duct and oval cell hyperplasia, nodular hyperplasia, cholangiofibrosis, multinucleated hepatocytes, diffuse fatty change bile duct cyst, necrosis, pigmentation, inflammation, portal fibrosis Lung: alveolar epithelium, metaplasia; bronchiolar epithelium, squamous metaplasia Adrenal cortex: atrophy and hyperplasia Thyroid gland: follicular cell hypertrophy Oral mucosa: gingival squamous hyperplasia
		Multiple: 0/53, 0/53, 0/52, 0/52, 16/51*	*$P \leq 0.01$	
		Hepatocellular carcinoma: 0/53, 0/53, 0/52, 0/52, 2/51	NS	
		Cholangiocarcinoma: 0/53, 0/53, 1/52, 9/52*, 30/51**	*$P \leq 0.05$ **$P \leq 0.01$	
		Multiple: 0/53, 0/53, 1/52, 5/53*, 21/52**	*$P \leq 0.05$ **$P \leq 0.01$ $P \leq 0.001$ (trend)	
		Hepatocholangioma: 0/53, 0/53, 0/52, 2/52, 6/51*	*$P = 0.012$ $P \leq 0.001$ (trend)	
		Multiple: 0/53, 0/53, 0/52, 0/52, 16/51*	*$P \leq 0.01$	

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Harlan Sprague-Dawley (F) 105 wk NTP (2006c) (cont.)		*Lung* Cystic keratinizing epithelioma: 0/53, 0/53, 0/52, 1/53, 11/52* Multiple: 0/53, 0/53, 0/52, 0/53, 8/52* Squamous cell carcinoma: 0/53, 0/53, 0/52, 1/53, 1/52 *Oral mucosa* Squamous cell carcinoma: 0/53, 0/53, 2/53*, 5/53, 9/53** *Pancreas, acinus* Adenoma: 0/53, 1/53, 1/52, 3/52, 1/50 Adenoma or carcinoma (combined): 0/53, 1/53, 1/52, 4/52, 2/50 *Uterus* Squamous cell carcinoma[c]: 1/53, 1/53, 1/53, 4/53, 0/53 *Adrenal cortex* Adenoma: 0/53, 0/53, 0/52, 1/52, 1/51 *Varying-ratio study:* *Liver* Hepatocellular adenoma: 2/50, 5/52, 21/51 Multiple: 2/50, 0/52, 7/51 Cholangiocarcinoma: 7/50, 9/52, 25/51 Multiple: 1/50, 5/52, 13/51 Hepatocholangioma: 0/53, 0/50, 2/52, 2/51	*$P < 0.001$ $P < 0.001$ (trend) *$P \leq 0.01$ NS *$P = 0.031$ **$P = 0.002$ $P < 0.001$ (trend) NS NS NS NS $P \leq 0.001$ NR $P \leq 0.001$ NR NR	Pancreas acinus atrophy and cytoplasmic vacuolization For the varying-ratio study, note that *P* values represent a trend test across the three groups of PCB-126/PCB-153 mixtures and indicated the significance of the effect of increasing the proportion of PCB-153 in the mixture

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Harlan Sprague-Dawley (F) 104 wk NTP (2006d)	*Core study:* PCB-126 and PCB-118 by gavage as binary mixture at doses of PCB-126 at 0, 62, 187, 622, 1866 or 3110 ng/kg bw per day, and PCB-118 at 0, 10, 30, 100, 300 or 500 µg/kg bw per day, in corn oil : acetone (99 : 1). [0, 7, 22, 72, 216 or 360 ng TEQ/kg bw] *Stop-exposure study:* PCB-126/PCB-118 at 3110 ng//500 µg/kg bw for 30 wk and then vehicle only for the remainder of the study. 81–86/group *Interim evaluations:* 10 rats per core study group were evaluated at 14, 31 and 53 wk	*Liver* Cholangiocarcinoma: 0/53, 0/51, 5/53, 19/53, 28/53, 12/65; 0/53, 19/50 (stop exposure) Multiple: 0/53, 0/51, 1/53, 12/53, 21/53, 72/65; 0/53, 12/50 (stop-exposure) Hepatocellular adenoma: 2/53, 1/51, 0/53, 4/53, 17/53, 5/65; 2/53, 1/50 (stop exposure) Multiple: 0/53, 0/51, 0/53, 2/53, 10/53, 2/65; 0/53, 0/50 (stop exposure) Hepatocellular carcinoma: 0/53, 0/51, 0/53, 0/53, 1/53, 0/65; 0/53, 0/50 (stop exposure) Hepatocholangioma: 0/53, 0/51, 0/53, 1/53, 1/53, 1/65; 0/53, 1/50 (stop exposure) Cholangioma: 0/53, 0/51, 0/53, 1/53, 0/53, 0/65; 0/53, 0/50 (stop exposure) *Lung* Cystic keratinizing epithelioma: 0/53, 0/51, 0/53, 20/53, 49/53, 41/66; 0/53, 12/50 (stop-exposure) Multiple: 0/53, 0/51, 0/53, 14/53, 48/53, 35/66; 0/53, 5/50 (stop exposure)	$P < 0.001$ (≥ 72 ng TEQ), $P < 0.001$ (trend) $P < 0.001$ (stop-exposure) $P \leq 0.05$ (≥ 72 ng TEQ) $P < 0.001$ (216 ng), $P = 0.021$ (360 ng) $P < 0.001$ (trend) $P \leq 0.001$ (216 ng) NS NS NS $P < 0.001$ (≥72 ng TEQ), $P < 0.001$ (trend) $P < 0.001$ (stop-exposure) $P \leq 0.01$ (≥ 72 ng TEQ) $P \leq 0.05$ (stop-exposure)	PCB-118, purity, > 98.5% (0.6% PCB-126; 0.2% PCB-77; 0.55% PCB-167) No animals in the core-study groups receiving the two higher doses survived to the end of the study, and survival in the stop-exposure group was significantly lower than in the vehicle-control group. Mean body weights in groups receiving PCB-126/PCB-118 at 622 ng/100 µg/kg bw or more were lower than in the vehicle-control groups throughout most of the study *Non-neoplastic lesions* Liver: the spectrum and severity of effects at the interim and 2-year time-points increased with dose and duration of exposure. At the end of the study in all groups receiving PCBs, there were significantly increased incidences and severity of toxic hepatopathy characterized by hepatocyte hypertrophy, multinucleated hepatocytes, pigmentation, diffuse fatty change, nodular hyperplasia, centrilobular fibrosis, cholangiofibrosis, oval cell hyperplasia, bile duct cyst, bile duct hyperplasia, and portal fibrosis

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Harlan Sprague-Dawley (F) 104 wk NTP (2006d) (cont.)		*Oral mucosa* Gingival squamous cell carcinoma: 1/53, 1/51, 2/53, 4/53, 0/53, 1/66; 1/53, 1/50 (stop-exposure) *Interim evaluation (at 53 wk)* *Lung* Cystic keratinizing epithelioma: 0/8, 0/7, 0/8, 1/8, 5/8, 0/0	NS $P \leq 0.05$ (216 ng TEQ)	Lung: bronchiolar metaplasia of the alveolar epithelium, squamous metaplasia, serosal fibrosis, and (in the stop-exposure group) keratin cysts Oral mucosa: gingival squamous hyperplasia
Known mixtures of PCBs				
Wistar (M) 120 d Rao & Banerji (1988), Silberhorn *et al.* (1990)	Aroclor 1260 at 0, 50, 100 ppm in the diet 32/group	Liver neoplastic nodules [tumours]: 0/32, 24/32*, 16/32*	*$P < 0.02$	Purity, NR *Non-neoplastic lesions:* adenofibrosis, centrilobular hypertrophy, individual hepatocyte necrosis, and vacuolated hepatocytes
F344 (M, F) 105 wk NTP (1978), Ward (1985), Morgan *et al.* (1981)	Aroclor 1254 at 0, 25, 50, 100 ppm in diet 24 M + 24 F per group	Hepatocellular adenoma: 0/24, 1/24, 2/24, 5/24* (M) 0/24, 0/24, 3/24, 2/24 (F) Hepatocellular carcinoma: 0/24, 0/24, 0/24, 2/24 (M) 0/24, 0/24, 0/24, 0/24 (F) Hepatocellular adenoma or carcinoma (combined): 0/24, 1/24, 2/24, 7/24* (M) 0/24, 0/24, 3/24, 2/24 (F) Adenocarcinoma of the glandular stomach: 0/47, 1/48, 3/48, 2/48 (M + F) Lymphoma or leukaemia: 3/24, 2/24, 5/24, 9/24 (M)	*$P < 0.05$ NS *$P < 0.05$ NS $P = 0.009$ (trend)	Chlorination, 54.67%; impurities not identified or quantitated Survival in males: controls, 92%; lowest dose, 83%; intermediate dose; 58%; highest dose, 46%. All females survived to the end of the bioassay *Non-neoplastic lesions* Liver: pigmented macrophages with cytoplasmic crystalline structures and hepatocellular degeneration Stomach: gastric intestinal metaplasia in males and females; gastric intestinal metaplasia and adenocarcinoma commonly coexist and may share initiating mechanisms

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
CR Sprague-Dawley (M, F) 24 mo Mayes *et al.* (1998), Faroon *et al.* (2001), Brown *et al.* (2007)	In the diet: Aroclor 1016: 0, 50, 100, 200 ppm Aroclor 1242: 0, 50, 100 ppm Aroclor 1254: 0, 25, 50, 100 ppm Aroclor 1260: 0, 25, 50, 100 ppm Treated: 50 M + 50 F/group Controls: 100 M + 100 F/group	*Liver (M)* *Aroclor 1016:* Hepatocellular adenoma: 4/100, 1/50, 1/50, 2/50 Hepatocellular carcinoma: 3/100, 1/50, 1/50, 2/50 Total liver tumours: 7/100, 2/50, 2/50, 4/50 *Aroclor 1242:* Hepatocellular adenoma: 4/100, 1/50, 3/50 Hepatocellular carcinoma: 3/100, 1/50, 1/50 Total liver tumours: 7/100, 1/50, 4/50 *Aroclor 1254:* Hepatocellular adenoma: 4/100, 2/50, 2/50, 6/50 Hepatocellular carcinoma: 3/100, 2/50, 2/50, 0/50 Total liver tumours: 7/100, 4/50, 4/50, 6/50 *Aroclor 1260:* Hepatocellular adenoma: 4/100, 2/50, 5/50, 7/50* Multiple: 0/100, 0/50, 2/50, 3/50 Hepatocellular carcinoma: 3/100, 1/50, 1/50, 3/50 Multiple: 0/100, 0/50, 0/50, 1/50 Hepatocholangioma: 0/100, 0/50, 0/50, 2/50 Total liver tumours: 7/100, 3/50, 6/50, 10/50*	 NS NS NS NS NS NS NS NS NS *$P \leq 0.05$ NS NS NS NS *$P \leq 0.05$	Purity, NR Total liver tumours include hepatocellular adenoma and carcinoma, hepatocholangioma and hepatocholangiocarcinoma Liver toxicity was distinctly more severe in females than in males. Non-neoplastic lesions observed in the liver: centrilobular hypertrophy, bile duct hyperplasia, hepatocyte vacuolization, and basophilic, clear cell, eosinophilic, and mixed cell foci In males given Aroclor 1242, 1254, or 1260, there were non-statistically significant increases in the incidence of follicular cell hyperplasia

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
CR Sprague-Dawley (M, F) 24 mo Mayes et al. (1998), Faroon et al. (2001), Brown et al. (2007) (cont.)		*Liver (F)* *Aroclor 1016:* Hepatocellular adenoma: 1/100, 1/50, 5/50*, 5/50* Multiple: 0/100, 0/50, 1/50, 3/50 Hepatocellular carcinoma: 0/100, 0/50, 1/50, 0/50 Total liver tumours: 1/100, 1/50, 6/50*, 5/50** *Aroclor 1242:* Hepatocellular adenoma: 1/100, 10/50*, 12/50* Multiple: 0/100, 3/50*, 7/50** Hepatocellular carcinoma: 0/100, 0/50, 2/50 Hepatocholangioma: 0/100, 1/50, 2/50 Hepatocholangiocarcinoma: 0/100, 1/50, 0/50 Total liver tumours: 1/100, 11/50*, 15/50* *Aroclor 1254:* Hepatocellular adenoma: 1/100, 18/50*, 26/50*, 27/50* Multiple: 0/100, 9/50*, 15/50*, 21/50* Hepatocellular carcinoma: 0/100, 0/50, 4/50*, 6/50** Multiple: 0/100, 0/50, 1/50, 4/50*	 *$P \leq 0.05$ $P \leq 0.05$ (trend) NS *$P \leq 0.01$ **$P \leq 0.05$ *$P \leq 0.01$ *$P \leq 0.05$ **$P \leq 0.01$ NS NS NS *$P \leq 0.01$ *$P \leq 0.01$ *$P \leq 0.01$ *$P \leq 0.05$ **$P < 0.01$ *$P \leq 0.05$	

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
CR Sprague-Dawley (M, F) 24 mo Mayes et al. (1998), Faroon et al. (2001), Brown et al. (2007) (cont.)		Hepatocholangioma: 0/100, 2/50, 6/50*, 1/50	*$P \leq 0.01$	
		Total liver tumours: 1/100, 19/50*, 28/50*, 28/50*	*$P \leq 0.01$	
		Aroclor 1260:		
		Hepatocellular adenoma: 1/100, 9/50*, 10/50*, 21/50*	*$P \leq 0.01$	
		Multiple: 0/100, 6/50*, 8/50*, 16/50*	*$P \leq 0.01$	
		Hepatocellular carcinoma: 0/100, 1/50, 1/50, 5/50*	*$P \leq 0.01$	
		Multiple: 0/100, 0/50, 0/50, 1/50	NS	
		Hepatocholangioma: 0/100, 0/50, 0/50, 3/50*	*$P \leq 0.05$	
		Total liver tumours: 1/100, 10/50*, 11/50*, 24/50*	*$P \leq 0.01$	
		Thyroid gland (M)		
		Aroclor 1016:		
		Follicular cell adenoma: 1/100, 3/50, 2/50, 0/50	NS	
		Follicular cell carcinoma: 1/100, 1/50, 1/50, 1/50	NS	
		Total thyroid tumours: 2/100, 4/50, 3/50, 1/50	NS	
		Aroclor 1242:		
		Follicular cell adenoma: 1/100, 5/50*, 5/50*	*$P \leq 0.05$	
		Follicular cell carcinoma: 1/100, 2/50, 1/50	NS	
		Total thyroid tumours: 2/100, 7/50*, 6/50**	*$P \leq 0.01$ **$P \leq 0.05$	

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
CR Sprague-Dawley (M, F) 24 mo Mayes et al. (1998), Faroon et al. (2001), Brown et al. (2007) (cont.)		*Aroclor 1254:* Follicular cell adenoma: 1/100, 6/50*, 4/50**, 5/50** Follicular cell carcinoma: 1/100, 1/50, 3/50, 1/50 Total thyroid tumours: 2/100, 7/50*, 7/50*, 6/50** *Aroclor 1260:* Follicular cell adenoma: 1/100, 6/50*, 4/50*, 3/50 Follicular cell carcinoma: 1/100, 1/50, 1/50, 1/50 Total thyroid tumours: 2/100, 7/50*, 5/50**, 4/50 *Mammary gland (F)* *Aroclor 1254:* Fibroadenoma: 34/100, 22/50, 29/50*, 10/50	*$P \leq 0.01$ **$P \leq 0.05$ NS *$P \leq 0.01$ **$P \leq 0.05$ *$P \leq 0.01$ NS *$P \leq 0.01$ **$P \leq 0.05$ *$P \leq 0.01$	
Sprague-Dawley (M, F) 29 mo Norback & Weltman (1985)	Feed containing Aroclor 1260 (mixed with corn oil) at 100 ppm for 16 mo, then at 50 ppm for an additional 8 mo, and then the control diet for an additional 5 mo. Controls received basal diet with added corn oil for 18 mo, then the basal diet only for 10 mo The medial and left lobes of the liver of 10 rats (2 M and 2 F controls, and 3 M and 3 F PCB-treated rats, for each period) were removed (partial hepatectomy) at 1, 3, 6, 9, 12, 15, and 18 mo Control: 63/group (M, F) Aroclor 1260: 70/group (M, F)	*Liver* Neoplastic nodule: 0/32, 5/46 (M); 1/49, 2/47 (F) Trabecular carcinoma: 0/32, 2/46 (M); 0/49, 19/47 (F) Adenocarcinoma: 0/32, 0/46 (M); 0/32, 24/47 (F) Cholangioma (simple): 2/32, 14/46 (M); 2/49, 21/47 (F) Cholangioma (cystic): 0/32, 2/46 (M); 1/49, 5/47 (F)	NS $P < 0.0001$ (F) $P < 0.0001$ (F) $P = 0.01$ (M) $P < 0.0001$ (F) NS	Purity, NR Some adenocarcinoma-bearing rats also had trabecular carcinoma (not included in the incidence of trabecular carcinoma) PCB-exposed rats developed hepatocellular lesions in the following sequence: centrilobular cell hypertrophy at 1 mo, foci of cell alteration at 3 mo, areas of cell alteration at 6 mo, neoplastic nodules at 12 mo, trabecular carcinoma at 15 mo, and adenocarcinoma at 24 mo

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Wistar (M) Up to 832 days Schaeffer *et al.* (1984), Faroon *et al.* (2001)	Basic diet for 8 wk, then: Group 1: basic diet; 139 rats (controls) Group 2: basic diet supplemented with Clophen A 30 at 100 ppm; 152 rats Group 3: basic diet supplemented with Clophen A 60 at 100 ppm; 141 rats After 801 days, randomly selected rats from all three groups were killed daily up to day 832	Hepatocellular neoplastic nodules: 5/131, 38/130*, 63/126* Hepatocellular carcinoma: 1/131, 4/130, 61/126* Thymoma: 16/131, 4/130, 2/129 Other neoplasms: 88/131, 66/138, 33/129	*$P < 0.05$ *$P < 0.05$ NS NS	Purity of Clophen A 30, 99.1%; purity of Clophen A 60, 99.9% Over the first 800 days on study, total mortality in groups 2 and 3 was significantly lower than in group 1 (controls) Hepatic foci of cellular alteration were observed in all groups, but were more frequent in the treated groups. There was a trend from foci to neoplastic nodules to hepatocellular carcinoma. Other non-neoplastic hepatic lesions observed in control and treated groups included bile duct hyperplasia The results of a re-evaluation of the hepatocellular tumours using contemporary diagnostic criteria and nomenclature were in general consistent with the original evaluation (Moore *et al.*, 1994) Tumour data were reported in six 100-day periods; the data reflected incidences from day 1 until day 832
Sherman (F) 22 mo Kimbrough *et al.* (1975), Moore *et al.* (1994)	Diets containing Aroclor 1260 at 0 or 100 ppm for up to 21 mo 200/group	*Liver* Hepatic neoplastic nodules: 0/173, 144/184 Hepatocellular carcinoma: 1/173, 26/184	$P < 0.0001$ $P < 0.0001$	Purity, NR The incidences of the hepatocellular lesions were re-evaluated by a panel of pathologists using contemporary diagnostic criteria and nomenclature (Moore *et al.*, 1994). Lesions that had been previously diagnosed as neoplastic nodules were now classified as either hepatocellular hyperplasia or hepatocellular adenoma. In general, the results were consistent between the original evaluation and the re-evaluation

Table 3.1 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence and/or multiplicity of tumours	Significance	Comments
Donryu (M, F) ≤ 560 days Kimura & Baba (1973), Silberhorn *et al.* (1990)	Diets containing Kanechlor 400 (in olive oil) at 38.5 ppm for 4 wk, then, based on bw-gain, the initial concentration was sequentially increased: 2× for 8 wk 4× for 3 wk 8× for 3 wk 16× for 8 wk, decreased to 12× for 32 wk because bw decreased markedly; two 4-wk periods with no treatment during this time Controls were fed powdered diet mixed with pure olive oil Controls: 5 M + 5 F/group Treated: 10 M + 10 F/group	Liver adenomatous nodules: 0/5, 0/10 (M); 0/5, 6/10 (F) Adrenal gland adenoma: 0/5, 0/10 (M); 0/5, 1/10 (F)	 $P = 0.044$ (F) NS	Purity, NR Multiple small nodules observed in the livers of females, but not males Fatty degeneration observed in the liver of all dosed groups, but only in two females in the control groups Study may have been limited by short duration, small number of rats/group, and may have exceeded the maximum tolerated dose The Working Group noted that current terminology for adenomatous nodules is hepatocellular adenoma

[a] Historical controls: 4/371 (1.1% ± 1.5%); range, 0–4%
[b] Historical controls: 0/371
[c] Historical controls: 4/371 (1.1% ± 1.0%); range, 0–2%
[d] Historical controls: 6/473 (1.3% ± 1.4%); range, 0–4%
[e] Historical controls: 1/371 (0.3% ± 0.7%); range, 0–2%

bw, body weight; F, female; M, male; mo, month; NR, not reported; NS, not significant; PCB, polychlorinated biphenyl; wk, week; yr, year

Table 3.2 Studies of carcinogenicity in mice exposed to PCBs and related compounds

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence of tumours	Significance	Comments
C57BL/6, B6D2F1 or DBA/2 (M) 44 wk Beebe *et al.* (1995)	Initiation with a single intraperitoneal dose of NDEA, 90 mg/kg bw, in tricaprylin, or tricaprylin only, and promoted 3 wk later ± Aroclor 1254 (100 ppm) in the diet for 20 wk 18–39/group	*Tricaprylin only, or tricaprylin + Aroclor 1254:* C57BL/6 mice: Liver tumours (all types): 0/27, 0/27 Lung tumours: 1/27, 1/27 B6D2F1 mice: Liver tumours (all types): 0/31, 2/34 Lung tumours: 0/31, 2/34 DBA/2 mice: Liver tumours (all types): 0/23, 0/24 Lung tumours: 3/31, 1/24	 NS NS NS	Purity, NR
dd (M, F) 24 or 32 wk Nagasaki *et al.* (1975)	Diet containing Kanechlor 300, 400 or 500 for 24 or 32 wk 24-wk study: Kanechlor 400 (0, 100, 250 ppm) or Kanechlor 500 (0, 100, 250 ppm) 32-wk study: Kanechlor 300 (0, 100, 250, 500 ppm) Kanechlor 400 (0, 100, 250, 500 ppm) Kanechlor 500 (0, 100, 250, 500 ppm) 20/group	*Hepatocellular carcinoma, 24 wk study:* Kanechlor 400: 0/20, 0/20, 0/20 (M) Kanechlor 500: 0/20, 0/20, 0/20 (M) *Hepatocellular carcinoma, 32-wk study:* Kanechlor 300: 0/20, 0/19, 0/19, 0/20 (M); 0/12, 0/19, 0/20, 0/20 (F) Kanechlor 400: 0/20, 0/17, 0/19, 0/20 (M); 0/12, 0/20, 0/20, 0/17 (F) Kanechlor 500: 0/20, 0/18, 0/20, 9/17*(M); 0/12, 0/19, 0/20, 4/17*(F)	 NS NS NS NS *$P < 0.05$	Purity, NR Other proliferative lesions observed in the liver of mice treated with Kanechlor 400 or 500 included oval cell hyperplasia, bile duct proliferation, cellular hypertrophy and nodular hyperplasia
BALB/cJ (M) 11 mo Kimbrough & Linder (1974), Faroon *et al.* (2001)	Diets containing Aroclor 1254 (mixed with corn starch) at 0 or 300 ppm for 6 mo, followed by basal diet for 5 mo, or Aroclor 1254 at 0 or 300 ppm for 11 mo Group 1: control diet for 6 mo Group 2: Aroclor 1254 for 6 mo Group 3: control diet for 11 mo Group 4: Aroclor 1254 for 11 mo 50/group	*Hepatoma* 6 mo: 0/24, 1/24, 11 mo: 0/34, 10/22	 NS $P < 0.001$	Purity, NR The Working Group noted that "hepatoma" is not a nomenclature currently used in toxicological pathology. In studies before 1978, the term "hepatoma" may have been used to denote benign or malignant liver tumours. In this study it was not clear whether hepatoma referred to a benign or malignant hepatic tumour

Table 3.2 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence of tumours	Significance	Comments
dd (M) 32 wk Ito *et al.* (1973), Silberhorn *et al.* (1990), Faroon *et al.* (2001)	Basal diet supplemented with Kanechlor for 32 wk: Kanechlor 300 at 0, 100, 250 or 500 ppm Kanechlor 400 at 0, 100, 250 or 500 ppm Kanechlor 500 at 0, 100, 250 or 500 ppm 12 mice/treated group; 6 controls	*Liver* *Kanechlor 300* Nodular hyperplasia: 0/6, 0/12, 0/12, 0/12 Hepatocellular carcinoma: 0/6, 0/12, 0/12, 0/12 *Kanechlor 400* Nodular hyperplasia: 0/6, 0/12, 0/12, 0/12 Hepatocellular carcinoma: 0/6, 0/12, 0/12, 0/12 *Kanechlor 500* Nodular hyperplasia: 0/6, 0/12, 0/12, 7/12* Hepatocellular carcinoma: 0/6, 0/12, 0/12, 5/12*	 NS NS NS NS *[P<0.05] *[NS]	Purity: Kanechlor 300: 59.8% trichlorobiphenyl, 23.0% tetrachlorobiphenyl, 16.6% dichlorobiphenyl, 0.6% pentachlorobiphenyl Kanechlor 400: 43.8% tetrachlorobiphenyl, 32.8% trichlorobiphenyl, 5.8% pentachlorobiphenyl, 4.6% hexachlorobiphenyl, 3.0% dichlorobiphenyl Kanechlor 500: 55.0% pentachlorobiphenyl, 26.5% tetrachlorobiphenyl, 12.8% hexachlorobiphenyl, 5.0% trichlorobiphenyl The description of nodular hyperplasias provided was not sufficiently detailed to determine whether these hyperplastic nodules were benign hepatocellur adenomas according to current nomenclature Other histopathological changes in mice treated with PCBs included oval cell proliferation, bile duct proliferation and hepatocyte hypertrophy. Amyloid deposition was also observed in the livers of mice fed diets containing various commercial PCB mixtures at 100 or 250 ppm

d, day; mo, month; NDEA, *N*-nitrosodiethylamine; NR, not reported; NS, not significant; PCB, polychlorinated biphenyl; wk, week; yr, year

(b) PCB-153

Rat

Groups of 80–82 female Harlan Sprague-Dawley rats (age, 8 weeks) were given the di-*ortho*-substituted non-dioxin-like congener PCB-153 (purity, 99%) at a dose of 0 (81 rats; vehicle control), 10, 100, 300, 1000, or 3000 µg/kg bw, in corn oil:acetone (99 : 1) by gavage, 5 days per week, for up to 105 weeks (core study) (Yoshizawa *et al.*, 2005, 2007, 2009; NTP, 2006b). Ten rats per group were evaluated at 14, 31, or 53 weeks. A stop-exposure group of 50 female rats was given PCB-153 at 3000 µg/kg bw corn oil : acetone (99 : 1) by gavage for 30 weeks, and then the vehicle only for the remainder of the study. At 2 years, cholangiomas occurred in two rats at 1000 µg/kg bw and in two rats in the stop-exposure group. A single hepatocellular adenoma was observed in the group at 3000 µg/kg bw. Cholangioma did not occur in the historical vehicle controls (0 out of 371) of the NTP studies. [One factor limiting interpretation of effects in this bioassay was that the highest dose of PCB-153 used (3000 µg/kg bw) was chosen to match the highest dose used in an NTP bioassay with a mixture of PCB-126 and PCB-153 (NTP, 2006c), rather than on the basis of the results of a previous short-term study of toxicity. There was no effect of PCB-153 at 3000 µg/kg bw on survival or body weight in this 2-year study, suggesting that higher doses would probably have been tolerated. In a tumour-promotion study in F344 female rats, Dean *et al.* (2002) gave PCB-153 at a dose of 10 000 µg/kg bw by gavage, three times per week, for 8 weeks, and observed only a significant increase in liver weight.]

(c) PCB-118

Rat

Groups of 80 female Harlan Sprague-Dawley rats (age, 8 weeks) were given PCB-118 (purity, > 99%) at a dose of 0 (vehicle control), 100, 220, 460, 1000, or 4600 µg/kg bw in corn oil : acetone (99 : 1) by gavage, 5 days per week, for up to 105 weeks (core study) (Yoshizawa *et al.*, 2009; NTP, 2010). Ten rats per group were evaluated at 14, 31, or 53 weeks. A stop-exposure group of 50 female rats was given PCB-118 at a dose of 4600 µg/kg bw in corn oil : acetone (99 : 1) by gavage for 30 weeks, then the vehicle for the remainder of the study. At the 53-week interim evaluation, three cholangiocarcinomas and one hepatocellular adenoma were observed in the group at 4600 µg/kg bw. At 2 years, the incidences of multiple cholangiocarcinoma, and single or multiple cholangiocarcinoma (combined) in the group at 4600 µg/kg bw and the stop-exposure group were significantly greater than those in the vehicle-control group. The incidences of multiple hepatocellular adenoma in the group at 4600 µg/kg bw, and single or multiple hepatocellular adenoma (combined) in the groups at 1000 µg/kg bw or 4600 µg/kg bw were significantly greater than those in the vehicle-control group. Four rats developed hepatocholangioma and one rat developed hepatocellular carcinoma in the group at 4600 µg/kg bw. Significantly increased incidences of multiple cystic keratinizing epithelioma of the lung and of single or multiple cystic keratinizing epithelioma (combined) occurred in the group at 4600 µg/kg bw compared with the vehicle-control group. The incidence of uterine carcinoma in the stop-exposure group was significantly greater than that in the vehicle-control group; a slight increase in the incidence of squamous cell carcinoma of the uterus occurred in the group at 220 µg/kg bw, and single incidences occurred at 460 µg/kg bw, 1000 µg/kg bw, and in the stop-exposure group. There were slightly increased incidences of exocrine pancreatic adenoma in core-study groups receiving PCB-118 at doses of 460 µg/kg bw or higher.

Table 3.3 Description of binary mixtures of PCB-126 and PCB-153 given to rats in a study of carcinogenicity by the NTP (2006c)

Group	Total TEQ (ng TEQ/kg bw)	Mass	
		PCB-126 (ng/kg bw)	PCB-153 (μg/kg bw)
Constant ratio mixture			
1	Vehicle control	0	0
2	1	10	10
3	10	100	100
5	30	300	300
7	100	1000	1000
Varying ratio mixture			
1	Vehicle control	0	0
4	30	300	100
5	30	300	300
6	30	300	1000

PCB, polychlorinated biphenyl; TEQ, toxic equivalent

(d) PCB-126 and PCB-153

Rat

The NTP conducted a 2-year study that was designed to assess the carcinogenicity of a mixture of PCB-126 and PCB-153 in a constant ratio, and a mixture of PCB-126 and PCB-153 in varying ratios to assess the effect of increasing PCB-153 (NTP, 2006c; Yoshizawa *et al.*, 2009). Groups of 81 or 80 female Harlan Sprague-Dawley rats (age, 8 weeks) received a mixture of PCB-126 and PCB-153 in corn oil : acetone (99 : 1) by gavage, 5 days per week, for up to 105 weeks. Dose groups were referred to by the total concentrations of toxic equivalents (TEQ) provided by the PCBs in the mixture per kg bw in each group (see Table 3.3); a control group of 81 female rats received the corn oil : acetone vehicle only (group 1). Ten rats per group were evaluated at 14, 31, and 53 weeks. At 2 years, the incidences of hepatocellular adenoma (single or multiple) in group 7 (constant ratio; TEQ, 100 ng/kg bw), and of cholangiocarcinoma (single or multiple) in group 5 (constant ratio; TEQ, 30 ng/kg bw) or group 7 were significantly increased. The incidence of hepatocholangioma was also significantly increased in group 7. Two rats in group 7 had hepatocellular carcinoma; no hepatocellular carcinoma was reported in the historical vehicle controls. In the varying-ratio study, increasing the proportion of PCB-153 significantly increased the incidences of hepatocellular adenoma and cholangiocarcinoma. In the constant-ratio study, the incidence of cystic keratinizing epithelioma of the lung was significantly increased in group 7. In addition, one squamous cell carcinoma was reported in group 5 and one in group 7. Significantly increased incidences of gingival squamous cell carcinoma of the oral mucosa occurred in groups 5 and 7. There was also a slight increase in the incidence of uterine squamous cell carcinoma in group 5.

(e) PCB-118 and PCB-126

Rat

Groups of 81 female Harlan Sprague-Dawley rats (age, 9 weeks) were given a binary mixture of PCB-118 and PCB-126 (see Table 3.4) at a dose of 0 (vehicle control), 7, 22, 72, 216 ng TEQ/kg bw, by gavage in corn oil : acetone (99 : 1), 5 days per week, for up to 104 weeks; a group of 86 female rats received the mixture at a dose of 360 ng TEQ/kg bw (Yoshizawa *et al.*, 2005, 2007,

Table 3.4 Composition of a mixture of PCB-118 and PCB-126 given to rats in a study of carcinogenicity by the NTP (2006d)

	PCB-118	PCB-126	PCB-77[a]	PCB-167[a]
Percentage of bulk mass[b]	98.5	0.6	0.2	0.5
Percentage of total TEQ[c]	13.7	86.3	0.03	0.007

[a] Present as contaminants that were not considered to contribute to the dioxin-like activity of the bulk synthesized test article
[b] Based on the level of each compound present in the bulk synthesized test article
[c] Assuming WHO toxic equivalency factor (TEF) values of 0.1 (PCB-126), 0.0001 (PCB-118), 0.0001 (PCB-77) and 0.00001 (PCB-167)
PCB, polychlorinated biphenyl; TEQ, toxic equivalent

2009; NTP, 2006d). Ten rats per group were evaluated at 14, 31, or 53 weeks. In the stop-exposure group, 50 female rats received the mixture at a dose of 360 ng TEQ/kg bw for 30 weeks, and then the vehicle only for the remainder of the study. The dose groups are described in Table 3.5. The mixture contained predominantly PCB-118 (by mass) and PCB-126 (by TEQ), but also contained PCB-77 and PCB-167 as contaminants that were not considered to contribute to the dioxin-like activity of the bulk synthesized material (see Table 3.4).

No rats at 216 or 360 ng TEQ/kg bw survived to the end of the study; survival in the stop-exposure group was also significantly lower than in the vehicle-control group, with only 10 rats surviving to the end of the study. Mean body weights of rats receiving 72 ng TEQ/kg bw or more were lower than those of rats in the vehicle-control group throughout most of the study. At 2 years, the incidences of cholangiocarcinoma (single or multiple, combined) and cholangiocarcinoma (multiple) were significantly increased in groups receiving 72 ng TEQ/kg bw or more. The incidence of hepatocellular adenoma was also significantly increased in the groups at 216 and 360 ng TEQ/kg bw. In addition, single occurrences of hepatocholangioma, cholangioma, or hepatocellular carcinoma were observed in some groups receiving 72 ng TEQ/kg bw or more. At 53 weeks, the incidence of cystic keratinizing epithelioma of the lung was significantly increased in the group at 216 ng TEQ/kg bw. At 2 years, significantly increased incidences of cystic keratinizing epithelioma (single or multiple, combined) and of cystic keratinizing epithelioma (multiple) were reported in groups receiving 72 ng TEQ/kg bw or more. Non-statistically significant increased incidences of gingival squamous cell carcinoma of the oral mucosa were observed at the end of the 2-year study.

3.1.2 Commercial mixtures of PCBs

(a) Aroclor 1254

(i) Mouse

In a study on the activity of Aroclor 1254 in mice with different aryl hydrocarbon receptor (AhR) phenotypes, groups of 23–34 male C57BL/6, DBA/2, or B6D2F1 mice (age, 5 weeks) were initiated with a single intraperitoneal dose of *N*-nitrosodiethylamine (NDEA) at 0 or 90 mg/kg bw, in tricaprylin. Three weeks later, the mice were placed on a diet containing Aroclor 1254 at a concentration of 100 ppm or the control diet for 20 weeks. After the promotion phase, the mice were left untreated until the terminal kill at age 52 weeks. Aroclor 1254 alone did not increase the incidence of tumours of the lung or liver in any of the three strains compared with their respective controls (Beebe *et al.*, 1995).

Four groups of 50 male BALB/cJ inbred mice (age, 5–6 weeks) were fed diets containing Aroclor 1254 (mixed with corn starch) at a concentration of 0 (control) or 300 ppm for up to 11 months (Kimbrough & Linder, 1974; Faroon *et al.*, 2001). After 6 months of exposure, one group of treated mice was fed the standard diet,

Table 3.5 Doses of PCB-118 and PCB-126 given to rats in a study of carcinogenicity by the NTP (2006d)

Dose (ng TEQ/kg bw)	Contribution to dose by mass[b]				Contribution to dose by TEQ[c] (ng TEQ/kg bw)				Total nominal dose by TEQ[c] (ng TEQ/kg bw)
	PCB-118 (µg/kg bw)	PCB-126 (ng/kg bw)	PCB-77[a] (ng/kg bw)	PCB-167[a] (ng/kg bw)	PCB-118	PCB-126	PCB-77[a]	PCB-167[a]	
7	10[d]	62	20	50	1.0	6.2	0.002	0.0005	7.2
22	30[d]	187	60	150	3.0	18.7	0.006	0.0015	21.6
72	100[d]	622	200	500	9.9	62.2	0.02	0.005	72.1
216	300[d]	1866	600	1500	29.6	186.6	0.06	0.015	216.2
360	500[d]	3110	1000	2500	49.3	311.0	0.1	0.025	360.4

[a] Present as contaminants that were not considered to contribute to the dioxin-like activity of the bulk synthesized test article
[b] Based on the level of each compound present in the bulk synthesized test article
[c] Assuming WHO TEF (toxic equivalency factor) values of 0.1 (PCB-126), 0.0001 (PCB-118), 0.0001 (PCB-77) and 0.00001 (PCB-167). TEQ value for PCB-118 was calculated assuming 98.5% of bulk material is PCB-118
[d] Nominal dose (µg/kg bw) of bulk synthesized material
PCB, polychlorinated biphenyl; TEQ, toxic equivalent

while the other treated group was fed the experimental diet for an additional 5 months; the two control groups were fed plain chow for an additional 5 months. Only one of 24 surviving mice given Aroclor 1254 for 6 months had a hepatoma [histopathology not further specified], while the incidence of hepatoma in the 22 surviving mice fed Aroclor 1254 for 11 months was significantly increased (10 out of 22; $P < 0.001$). Hepatomas were not found in any of the mice in the control groups.

(ii) Rat

Groups of 24 male and 24 female F344 rats (age, 7 weeks) were fed diets containing Aroclor 1254 at a concentration of 0, 25, 50, or 100 ppm in corn oil for up to 105 weeks (NTP, 1978; Ward, 1985; Safe, 1989; Silberhorn *et al.*, 1990; Faroon *et al.*, 2001). In males, hepatocellular adenoma was observed in one, two, and five of the rats at the lowest, intermediate, and highest dose, respectively, and hepatocellular carcinoma was observed in two rats at the highest dose; the incidences of hepatocellular adenoma and of hepatocellular adenoma or carcinoma (combined) in males at the highest dose were statistically significantly increased. Hepatocellular tumours were not observed in controls. Non-statistically significant low incidences of rare adenocarcinomas of the glandular stomach were observed in both sexes. Adenocarcinoma of the glandular stomach was not observed in controls. The historical incidence of adenocarcinoma of the glandular stomach at the study laboratory (6 out of 600 males [1%], 2 out of 600 females [0.3%]) suggested that the occurrence of these tumours, although not statistically significant, may have been related to the administration of Aroclor 1254. There was a statistically significant dose-related trend in the combined incidences of lymphoma and leukaemia in male rats, but incidence in each dose group was not statistically significantly different from that in matched controls. Morgan *et al.* (1981) and Ward (1985) re-examined the gastrointestinal lesions observed in the study by the NTP (1978) and found a dose-related increase in the incidence of metaplasia of the glandular stomach, and also found adenocarcinoma of the glandular stomach in six treated rats. When compared with the incidence of adenocarcinoma of the glandular stomach in historical controls (1 out of 3548), the total incidence (6 out of 144 male and female rats treated with Aroclor 1254) was statistically significant.

(b) Aroclor 1260

Rat

Groups of 200 female Sherman rats (age, 21–26 days) were fed diets containing Aroclor 1260 at a concentration of 0 (control) or 100 ppm for approximately 21 months (Kimbrough *et al.*, 1975). The rats were killed at age 23 months. There were statistically significant increases in the incidences of "hepatic neoplastic nodules" and of hepatocellular carcinoma in rats receiving Aroclor 1260 compared with controls. The hepatocellular tumours were re-evaluated histologically by a panel of pathologists using contemporary diagnostic criteria and nomenclature (Moore *et al.*, 1994). Lesions that had been previously diagnosed as "neoplastic nodules" were reclassified as either hepatocellular hyperplasia or hepatocellular adenoma. In general, the results of the re-evaluation were consistent with those of the original evaluation.

Groups of 32 male Wistar rats (age, 5 weeks) were fed a 10% protein diet containing Aroclor 1260 (dissolved in coconut oil) at a concentration of 0 (control), 50, or 100 ppm for 120 days (Rao & Banerji, 1988; Silberhorn *et al.*, 1990). Controls were fed diet mixed with coconut oil. The incidences of "liver neoplastic nodules" [liver tumours] were significantly increased in both groups of treated rats; however, the incidence of tumours in rats fed the higher dose was lower than that in rats fed the lower dose.

Groups of 70 male and 70 female Sprague-Dawley rats were fed a diet containing Aroclor 1260 at a concentration of 100 ppm for 16 months, followed by diet containing Aroclor 1260 at 50 ppm for an additional 8 months, and then basal diet for 5 months (Norback & Weltman, 1985; Safe, 1989; Silberhorn *et al.*, 1990; Moore *et al.*, 1994; Faroon *et al.*, 2001). Groups of 63 males and 63 females served as controls and received the basal diet supplemented with corn oil for 18 months, and then the basal diet only for the remainder of the study. The medial and left lobes of the liver of 10 rats (two male controls, two female controls, three PCB-treated males and three PCB-treated females, for each time-point) were removed at 1, 3, 6, 9, 12, 15, and 18 months. In treated rats that survived 18 months or longer, malignant hepatic tumours (adenocarcinoma and/or trabecular carcinoma) were found in 43 out of 47 females, but only in 2 out of 46 males. The individual incidences of adenocarcinoma and of trabecular carcinoma in PCB-treated females were significantly greater than in controls. Hepatic neoplastic nodules [benign hepatocellular tumours] occurred in 5 out of 46 males, and 2 out of 47 females. A single hepatic neoplastic nodule occurred in a female control rat. PCB-exposed rats developed cystic cholangioma in 2 out of 46 males, and 5 out of 47 females [non-significant], versus 0 out of 32 males and 1 out of 49 females among the controls. Preneoplastic lesions of the biliary tract, referred to as simple and cystic cholangioma, also occurred at a higher incidence in treated males and females (30% and 45%, respectively).

(c) Aroclor 1016, 1242, 1254, and 1260

Rat

A comprehensive comparative long-term study of toxicity and carcinogenicity was conducted with four of the most widely used commercial Aroclor mixtures: Aroclor 1016, 1242, 1254, and 1260 (Mayes *et al.*, 1998; Faroon *et al.*, 2001; Brown *et al.*, 2007). Groups of 50 male and 50 female Sprague–Dawley rats (age, 6–8 weeks) were fed diets containing Aroclor 1016, 1242, 1254, or 1260 at doses ranging from 25 to 200 ppm (three dose levels for Aroclor 1016, 1254 and 1260, and two dose levels for Aroclor 1242) for 24 months. Groups of 100 males and 100 females served as controls. Aroclor 1016, 1242, 1254, and 1260 contained polychlorinated dibenzodioxins (PCDDs) at concentrations of 0.6, 0, 20, and 0 ppb, respectively, and polychlorinated dibenzofurans (PCDFs) at concentrations

of 0.035, 2.9, 23, and 4.9 ppm, respectively. The basal diet contained PCBs at less than 0.15 ppm (estimated dose, < 0.01 mg/kg bw per day). Aroclor 1254 was treated to remove > 99% of the PCDFs. Feeding with diets containing Aroclor led to increased incidences of hepatic neoplasms (primarily hepatocellular adenoma) that were highly sex-dependent (females > males) and that differed between Aroclor mixtures. For females, the incidences of hepatocellular adenoma and of hepatocellular carcinoma increased with dose, with the following pattern: Aroclor 1254 > Aroclor 1260 > Aroclor 1242 > Aroclor 1016. The number of females bearing multiple hepatocellular tumours also increased in a dose-related manner for all Aroclor mixtures, and the highest numbers were in the groups receiving the intermediate and highest dose of Aroclor 1254, and the highest dose of Aroclor 1260. In addition, in females receiving Aroclor 1260, there was an increase in the incidence of cholangioma. In males, an increased incidence of hepatocellular adenoma was observed only in the group receiving Aroclor 1260 at the highest dose. The incidence of follicular cell adenoma of the thyroid gland was significantly increased in males in a non-dose-dependent manner; these increases were induced by Aroclor 1242 (both doses), Aroclor 1254 (all doses), and Aroclor 1260 (lowest and intermediate doses).

(d) Kanechlor 300, 400, and 500

(i) Mouse

Groups of 20 male and 20 female dd strain albino mice [age not reported] were given diets containing one of three PCB mixtures (Kanechlor 300, 400, or 500) at a concentration of 0, 100, 250, or 500 ppm for 24 or 32 weeks (Nagasaki *et al.*, 1975). The incidence of hepatocellular carcinoma was significantly increased in male and female mice given Kanechlor 500 at 500 ppm for 32 weeks. No tumours of the liver were found in mice fed Kanechlor 500 at dietary concentrations of 100 or 250 ppm, or the lesser chlorinated commercial mixtures Kanechlor 400 or Kanechlor 300 at any of the three dietary concentrations at 24 or 32 weeks.

Groups of 12 male dd strain albino mice (age, 8 weeks) were fed basal diets supplemented with one of three PCB mixtures (Kanechlor 300, 400, or 500) at a concentration of 100, 250, or 500 ppm for 32 weeks; a control group of six mice was fed basal diet alone (Ito *et al.*, 1973; Silberhorn *et al.*, 1990; Faroon *et al.*, 2001). The incidences of hepatocellular carcinoma (5 out of 12 [not significant]) and liver hyperplastic nodules [some of which may have been hepatocellular adenomas] (7 out of 12 [$P < 0.05$]) were increased in mice fed diets containing Kanechlor 500 at 500 ppm compared with controls (0 out of 6). Hepatic lesions were not found in mice fed Kanechlor 500 at lower doses, or in mice exposed to the less chlorinated mixtures Kanechlor 400 or Kanechlor 300 for 32 weeks. Other histopathological changes in mice treated with PCBs included oval-cell proliferation, bile duct proliferation, hepatocyte hypertrophy, and amyloidosis. [The Working Group noted that the study may have been limited by the small number of mice, the relatively short treatment period, and the absence of an observation period after treatment.]

(ii) Rat

A group of 10 male and 10 female Donryu rats (age, 10 weeks) were fed diet containing Kaneclor 400 at a concentration of 38.5 ppm for 4 weeks, then the initial concentration was increased (based on body weights) twice for 8 weeks, 4 times for 3 weeks, 8 times for 3 weeks, and 16 times for 8 weeks (Kimura & Baba, 1973; Silberhorn *et al.*, 1990). The latter concentration was decreased to 12 times for 32 weeks because body weights were decreasing markedly. Rats were then fed basal diet until moribund, up to 560 days. A group of five males and five females fed basal diets served as controls. Treatment with Kanechlor 400 (duration, 400 days) caused

a significant increase in the incidence of multiple adenomatous nodules [hepatocellular adenoma] in females. None of the treated males developed adenomatous nodules. [The Working Group noted that the study may have been limited by the small numbers of animals, and may have exceeded the maximum tolerated dose.]

(e) Clophen A 30 and Clophen A 60

Rat

Male weanling Wistar rats were fed a diet supplemented with Clophen A 30 (42% chlorine by weight) or Clophen A 60 (60% chlorine by weight) at a concentration of 100 ppm, or an estimated dose of 5 mg/kg bw per day, for up to 832 days; controls were fed the basal diet (Schaeffer *et al.*, 1984; Young, 1985; Safe, 1989). Tumour incidence was investigated at intervals of 100 days. After 800 days, the overall incidence of hepatocellular neoplastic nodules, irrespective of time period, was significantly increased in rats fed Clophen A 30 (38 out of 130) or Clophen A 60 (63 out of 126) compared with controls (5 out of 131). The incidence of hepatocellular carcinoma was significantly increased in rats fed Clophen A 60 (61 out of 126 compared with 1 out of 131 controls). The incidences of hepatocellular lesions were re-evaluated by a panel of pathologists using contemporary diagnostic criteria and nomenclature (Moore *et al.*, 1994). Lesions that had been previously diagnosed as neoplastic nodules were now classified as either hepatocellular hyperplasia or hepatocellular adenoma. The results of the re-evaluation were generally consistent with those of the original evaluation.

3.2 Transplacental and perinatal exposure

This section covers those studies for which exposure to PCBs occurred either transplacentally and/or perinatally. This period generally covers exposure from day 1 of gestation until weaning on postnatal day 21, although it should be noted that weaning can occur at up to age 28 days.

3.2.1 Individual PCBs and binary mixtures

(a) PCB-126

See Table 3.6

Rat

Five groups of pregnant Sprague-Dawley rats were given PCB-126 at a dose of 0 (corn oil), 0.025, 2.5, 250, or 7500 ng/kg bw by gavage on days 13 to 19 of gestation. Female pups from the exposed dams were weaned on postnatal day 21, and subsequently exposed at age 50 days to 7,12-dimethylbenz[*a*]anthracene (DMBA) at a dose of 20 mg/kg bw in corn oil by gavage, and followed until age 170 days (Muto *et al.*, 2001). There was no specific perinatal oral exposure to PCB-126. There was a significant reduction in body weight in the groups of pups at 250 ng/kg bw and 7500 ng/kg bw at postnatal day 21, and at 7500 ng/kg bw at age 30 days. There was a significant reduction in the incidence of DMBA-induced tumours of the mammary gland in the group at 7500 ng/kg bw. In the group at 7500 ng/kg bw, 41% of tumours were adenomas, while tumours in all other groups were mainly adenocarcinomas. [The study design was not a full carcinogenesis bioassay of PCBs.]

In a similar study by Wakui *et al.* (2005), four groups of pregnant Sprague-Dawley rats were given PCB-126 at a dose of 0 (corn oil vehicle), 2.5, 250, or 7500 ng/kg bw by gavage on days 13 to 19 of gestation. Female pups from the exposed dams were weaned at postnatal day 21, and subsequently exposed at age 50 days to DMBA at a dose of 100 mg/kg bw in corn oil by gavage, and followed until age 150 days. As in the study by Muto *et al.* (2001), there was a significant reduction in the incidence of adenocarcinoma of the mammary gland in the group at 7500 ng/kg bw. [The study design was not a full carcinogenesis bioassay of PCBs.]

Table 3.6 Studies of carcinogenicity in rats exposed perinatally or transplacentally to PCB-126

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), multiplicity of tumours	Significance	Comments
Sprague-Dawley (Japan SLC) (F) 170 day Muto *et al.* (2001)	Dams were treated with PCB-126 at 0 (corn oil vehicle), 0.025, 2.5, 250, or 7500 ng/kg bw (0.5 mL/rat) by gavage on days 13–19 of gestation. Pups were weaned at PND 21 Female pups (age 50 days) received DMBA at 20 mg/kg bw in corn oil by gavage and observed until age 170 days, or until tumours reached 20 mm in size Group 1: corn oil vehicle Group 2: 0.025 ng/kg bw Group 3: 2.5 ng/kg bw Group 4: 250 ng/kg bw Group 5: 7500 ng/kg bw 45/group	Tumours of the mammary gland: Group 1: 42/45, 3.12 ± 0.74 Group 2: 44/45, 2.77 ± 1.89 Group 3: 42/45, 3.98 ± 2.82 Group 4: 43/45, 5.09 ± 2.42 Group 5: 35/45*, 2.25 ± 1.55	*$P < 0.05$, χ^2 test (decrease)	Not a full carcinogenesis bioassay In the group at 7500 ng/kg bw, 41% of tumours were adenomas, whereas in all other groups the tumours were mainly adenocarcinomas
Sprague-Dawley (Japan SLC) (F) 150 day Wakui *et al.* (2005)	Dams were treated with PCB-126 at 0 (corn oil vehicle), 2.5, 250, 7500 ng/kg bw (0.5 mL/rat) by gavage on days 13–19 of gestation. Pups were weaned at PND 21 Females (age 50 days) received DMBA at 100 mg/kg bw in corn oil by gavage, and were observed until age 150 days Group 1: corn oil vehicle Group 2: 2.5 ng/kg bw Group 3: 250 ng/kg bw Group 4: 7500 ng/kg bw 25/group	Mammary gland, adenocarcinoma: Group 1: 22/25 (88%) Group 2: 21/25 (84%) Group 3: 23/25 (92%) Group 4: 16/25 (64%)*	*$P < 0.05$, χ^2 test (decrease)	Not a full carcinogenicity bioassay

DMBA, 7,12-dimethylbenz[*a*]anthracene; F, female; M, male; NDMA, *N*-nitrosodimethylamine; PND, postnatal day; wk, week

(b) PCB-153 and PCB-138

See Table 3.7

Mouse

Eight groups of male Swiss Cr:NIH(s) mice were given an intraperitoneal injection of *N*-nitrosodimethylamine (NDMA) at 0 (saline vehicle) or 5 mg/kg bw on postnatal day 4. On postnatal day 8, the mice were treated by gavage with PCB-153 or PCB-138, or a mixture of the two PCBs, each at a single dose of 20 mg/kg bw, or with the vehicle, olive oil (Anderson *et al.*, 1991). The concentration selected, 20 mg/kg bw, is approximately equivalent to the concentration of each PCB congener in a dose of 500 mg/kg bw of Aroclor 1254. The mice were killed at age 16 weeks. There was no effect of either PCB congener alone or in combination on the incidence of bronchioloalveolar adenoma in the absence of treatment with NDMA. In NDMA-initiated mice, there was a significant increase in the multiplicity of bronchioloalveolar adenoma in mice also exposed to PCB-138. There was no effect of PCB-153, or of PCB-153 plus PCB-138, when compared with controls treated with NDMA only. [This study was not a full carcinogenesis bioassay. It was limited regarding the effect of the PCBs alone without initiation, due to the short duration of observation.]

3.2.2 Commercial mixtures of PCBs

(a) Aroclor 1254

See Table 3.8

Mouse

Pregnant CD-1 mice were given a single intraperitoneal injection of Aroclor 1254 at a dose of 0 (corn oil) or 500 mg/kg bw on day 19 of gestation (Anderson *et al.*, 1983). Suckling mice were given NDMA at 0 (saline vehicle) or 5 mg/kg bw by intraperitoneal injection on postnatal day 4 or 14, or every 3 days on postnatal days 1–22. Mice were weaned at age 4 weeks and examined at approximately 28 weeks and 18 months. No tumours of the liver were found at 28 weeks in male or female mice exposed in utero to the vehicle or Aroclor 1254 alone without exposure to NDMA. At 18 months, there was no increase in the incidence of tumours of the liver in mice treated with Aroclor 1254 without NDMA exposure. In the groups that were exposed to NDMA on postnatal day 4 or 14, there was no effect of maternal exposure to Aroclor 1254 on the incidence or multiplicity of tumours of the liver in male or female mice. Nevertheless, at 18 months, there was a significant increase in the incidence of "coalescing" tumours of the liver in females exposed on postnatal day 4 and in males exposed on postnatal day 14. There was no effect of maternal exposure to Aroclor 1254 on the incidence or multiplicity of tumours of the liver in male or female pups treated with NDMA between postnatal days 1 and 22. [This study design was not a full carcinogenesis bioassay of PCBs. Although mice were exposed to PCBs before being exposed to NDMA, NDMA acts as an initiator. Thus results from the groups exposed to NDMA plus PCBs are more likely to reflect an effect of the exposure to PCBs in utero on NDMA carcinogenesis.]

Groups of male neonatal Swiss Cr:NIH(s) mice were injected intraperitoneally with NDMA at a dose of 5 mg/kg bw in saline on postnatal day 4 (Anderson *et al.*, 1986). On postnatal day 8, mice were exposed to Aroclor 1254 at a dose of 0 (control), 50, 250, or 500 mg/kg bw in olive oil by gavage, for 16 or 28 weeks. The study also included two non-initiated groups exposed to Aroclor 1254 at a dose of 0 or 500 mg/kg bw. A significant increase in the average number of bronchioloalveolar adenomas was observed in mice exposed to both NDMA and Aroclor 1254 compared with mice exposed to NDMA only, but not in mice exposed to Aroclor 1254 without NDMA initiation compared with mice exposed to vehicle only.

Table 3.7 Study of carcinogenicity in mice exposed perinatally to PCB-153 and PCB-138

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), multiplicity of tumours	Significance	Comments
Swiss Cr:NIH(s) (M) 16 wk Anderson *et al.* (1991)	Intraperitoneal injection on PND 4 with NDMA at 5 mg/kg bw or saline vehicle Exposure on PND 8 to PCBs (in olive oil) at 20 mg/kg bw by gavage until age 16 wk Group 1: NDMA Group 5: NDMA + PCB-153 Group 6: NDMA + PCB-138 Group 7: NDMA + PCB-153 + PCB-138 Group 8: saline/olive oil Group 2: PCB-153 Group 3: PCB-138 Group 4: PCB-153 + PCB-138 Number/group, NR	Bronchioloalveolar adenoma: Group 1: 15/55 (27%), 0.42 ± 0.11 Group 5: 13/53 (24%), 0.3 ± 0.08 Group 6: 21/50 (42%), 1.0 ± 0.3* Group 7: 14/46 (30%), 0.52 ± 0.13 Group 8: 0/26 Group 2: 0/32 Group 3: 0/31 Group 4: 0/34	*P = 0.014 vs group 1	Purity, NR Not a full carcinogenicity bioassay Concentration of PCBs (20 mg/kg bw) is approximately equivalent to that of each PCB congener in Aroclor 1254 at 500 mg/kg bw

M, male; NDMA, *N*-nitrosodimethylamine; PCB, polychlorinated biphenyl; PND, postnatal day; vs, versus

Table 3.8 Studies of carcinogenicity in mice exposed perinatally or transplacentally to Aroclor 1254

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), multiplicity of tumours	Significance	Comments
CD-1 (M, F) 28 wk and 18 mo Anderson *et al.* (1983)	Pregnant dams given a single intraperitoneal injection of Aroclor 1254 at 0 (olive oil vehicle) or 500 mg/kg bw on day 19 of gestation. Progeny then injected intraperitoneally with saline (experiment 1) or NDMA at 5 mg/kg bw on PND 4 (experiment 2), PND 14 (experiment 3), or every 3 days from PND 1 to 22 (experiment 4) Group 1: olive oil (F, 28 wk) Group 2: Aroclor 1254 (F, 28 wk) Group 3: olive oil (F, 18 mo) Group 4: Aroclor 1254 (F, 18 mo) Group 5: olive oil (M, 28 wk) Group 6: Aroclor 1254 (M, 28 wk) Group 7: olive oil (M, 18 mo) Group 8: Aroclor 1254 (M, 18 mo) Number of mice/group, NR	*Experiment 1 (no NDMA):* Liver tumours: 0/23, 0/21, 1/31, 1/23, 0/21, 0/23, 12/23, 8/25 *Experiment 2 (NDMA on PND 4):* Liver tumours: 3/17, 3/21, 21/29, 17/20, 17/23, 14/24, 27/28, 17/17 Liver (coalescing) tumours: 0/17, 0/21, 7/29, 13/20*, 17/23, 14/24, 27/28, 17/17 *Experiment 3 (NDMA on PND 14):* Liver tumours: 2/18, 0/19, 16/24, 9/19, 9/26, 1/19**, 18/19, 18/19 Liver (coalescing) tumours: 0/18, 0/19, 3/24, 1/19, 0/26, 0/19, 8/19, 14/19***	NS NS *$P < 0.01$ (Fisher exact test) **$P < 0.04$ (Fisher exact test), decrease ***$P < 0.035$ (Fisher exact test)	Purity, NR Tumour incidence and multiplicity in progeny (from dams treated with Aroclor 1254) exposed to NDMA every 3 days from PND 1 to PND 22 (experiment 4) were not increased and are not shown
Swiss Cr:NIH(s) (M) 16 or 28 wk Anderson *et al.* (1986)	Intraperitoneal injection of NDMA (0 or 5 mg/kg bw) in saline on PND 4 followed on PND 8 by exposure to Aroclor 1254 in olive oil by gavage Groups were exposed for 16 or 28 wk to: NDMA + Aroclor 1254 (50 mg/kg bw); NDMA + Aroclor 1254 (250 mg/kg bw); NDMA + Aroclor 1254 (500 mg/kg bw); NDMA + olive oil; saline + Aroclor 1254 (500 mg/kg bw); saline + olive oil Number/group, NR	Bronchioloalveolar adenoma (average no. of tumours/no. of examined animals): 16 wk: 5.7/16, 5.1/12, 11.8/14*, 6.1/17, 0/13, 0.2/6 28 wk: 7.9/15, 8.6/14, 11.9/16**, 6.6/16, 0.2/19, 0.1/7	*$P < 0.05$ **$P < 0.01$	Purity, NR Not a full carcinogenicity bioassay. Short duration

Table 3.8 (continued)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), multiplicity of tumours	Significance	Comments
Swiss Cr:NIH(s) (M) up to 72 wk Anderson *et al.* (1994)	Intraperitoneal injection on PND 4 with NDMA at 5 mg/kg bw or saline vehicle. At age 8 days, mice received Aroclor 1254 at 250 mg/kg bw by gavage in olive oil or vehicle only. Mice were killed when moribund or at age 16, 28, 52, or 72 wk Groups were exposed to: NDMA; NDMA + Aroclor 1254; Aroclor 1254; saline/oil Number/group, NR	*Bronchioloalveolar adenoma:* Age 28 wk: 7/23[a] (30%), 0.5 ± 1.1[b]; 19/27[a] (70%), 1.9 ± 2.9[b]; 0/13; 0/16 Age 52 wk: 12/25 (48%), 0.6 ± 0.8[c]; 15/23 (65%), 2.7 ± 3.8[c]; 4/24 (17%), 0.17 ± 0.38; 6/27 (22%), 0.26 ± 0.4 Age 72 wk: 21/23 (91%), 5.1 ± 4.5; 17/23 (74%), 3.9 ± 4.3; 17/25 (68%), 0.9 ± 0.8; 17/39 (44%), 0.6 ± 0.7 *Liver adenoma:* Age 52 wk: 1/25[d] (4%), 0.04 ± 0.2; 9/23[d] (39%), 0.6 ± 0.8; 0/24; 0/27 Age 72 wk: 16/23 (70%), 1.8 ± 2.2; 14/25 (56%), 1.5 ± 2.0; 0/25; 0/39	Matched letters are significantly different from each other [a]$P = 0.01$ [b]$P = 0.0033$ [c]$P = 0.0496$ [d]$P = 0.004$	Purity, NR Not a full carcinogenicity bioassay

mo, month; NDMA, *N*-nitrosodimethylamine; NR, not reported; NS, not significant; PND, postnatal day; wk, week

In a subsequent experiment, groups of neonatal male Swiss Cr:NIH(s) mice were given an intraperitoneal injection of NDMA at a dose of 0 (saline vehicle) or 5 mg/kg bw on postnatal day 4, then given Aroclor 1254 at a dose of 0 or 250 mg/kg bw in olive oil on day 8 by gavage, and killed at age 16, 28, 52, or 72 weeks (Anderson *et al.*, 1994). At age 28 weeks, the incidence of bronchioloalveolar adenoma in mice initiated with NDMA was increased 2.5-fold by treatment with Aroclor 1254. The multiplicity of bronchioloalveolar adenoma was enhanced fourfold by treatment with Aroclor 1254 for 28 or 52 weeks. By 72 weeks, tumour numbers, although high, were similar in the groups receiving NDMA only, and NDMA plus Aroclor 1254. There was an increased incidence of liver adenoma at 52 weeks in mice receiving NDMA plus Aroclor 1254 compared with mice receiving NDMA only. By 72 weeks, the incidences in the groups receiving NDMA or NDMA plus Arochlor 1254 were similar. [This study was not a full carcinogenesis bioassay of PCBs.]

(b) Kanechlor 500

See Table 3.9

Rat

Pregnant Wistar rats were exposed to Kanechlor 500 at a dose of 0 (olive oil vehicle), 40, or 200 mg/kg bw by gavage on days 5, 10, and 15 of gestation (Nishizumi, 1980). Male and female pups were subsequently weaned and given drinking-water containing NDEA at 50 ppm for 5 weeks to induce liver tumours [mainly hepatocellular carcinomas] that were evaluated after 20 and 24 weeks. The average concentration of total PCBs in the liver at 4 weeks was 1 ppm, 18 ppm and 360 ppm in the groups at 0 (vehicle), 40 mg/kg bw and 200 mg/kg bw, respectively, indicating clear transfer from the dam to the offspring. In both males and females, there was a decrease in the multiplicity of NDEA-initiated tumours of the liver. [This study was not a full carcinogenesis bioassay.]

3.2.3 Mixtures of PCBs and other chlorinated agents found in human milk fat

*(a) Mixture of non-*ortho *PCBs, PCDFs, and PCDDs*

See Table 3.10

Rat

Female Sprague-Dawley rats were exposed by gavage at age 1, 5, 10, 15, and 20 days to a mixture of three non-*ortho* PCBs [PCB-77, PCB-126, and PCB-169], six PCDDs, and seven PCDFs, or were exposed to the vehicle (corn oil) only (Desaulniers *et al.*, 2004). The concentrations of these agents in the mixture were based on the concentrations of dioxin-like congeners found in human milk fat, and the doses administered were equal to 10 times, 100 times, or 1000 times the quantities found in milk fat. At age 50 days, groups of rats were injected intraperitoneally with *N*-methyl-*N*-nitrosourea (MNU) at a dose of 0 or 30 mg/kg bw to induce the development of tumours of the mammary gland. At age 32 weeks, in those groups not treated with MNU, there was a significant increase in the incidence of benign lesions of the mammary gland (adenoma, fibroadenoma, and hyperplasia) after exposure to the 1000-times mixture. In the MNU-treated groups, there was no effect of exposure to the mixture on the incidences of benign lesions or malignant tumours of the mammary gland. [This study was not a full carcinogenesis bioassay. Given the presence of PCDDs and PCDFs in the mixture, conclusions regarding the effect of PCBs alone could not be drawn from this study.]

(b) Mixture of PCBs, DDT, and DDE

See Table 3.11

Rat

Neonatal female Sprague Dawley rats were exposed to a mixture of 19 PCB-congeners, *p,p´*-dichlorodiphenyltrichloroethane (DDT), and *p,p´*-dichlorodiphenyldichloroethene (DDE)

Table 3.9 Study of carcinogenicity in rats exposed transplacentally and perinatally to Kanechlor 500

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), multiplicity of tumours	Significance	Comments
Wistar (M, F) up to 29 wk Nishizumi (1980)	Dams were given Kanechlor 500 at 0 (olive oil vehicle), 40, or 200 mg/kg bw by gavage on days 5, 10 and 15 of gestation. Male and female offspring were given drinking-water containing NDEA at 50 ppm for 5 wk, and were evaluated 20 and 24 wk after NDEA exposure Group 1: vehicle (olive oil) Group 2: Kanechlor 500 at 40 mg/kg bw Group 3: Kanechlor 500 at 200 mg/kg bw 6–8 M and 6–8 F/group	Liver tumours (≥ 5 mm) *M (20 wk):* Group 1: 6/7 (86%), 3.0 ± 0.7 Group 2: 6/8 (75%), 1.3 ± 0.4* Group 3: 4/6 (50%), 1.0 ± 0.4* *F (20 wk):* Group 1: 5/8 (62.5%), 1.1 ± 0.4 Group 2: 4/8 (50%), 0.6 ± 0.3 Group 3: 0/8, 0* *M (24 wk):* Group 1: 8/8 (100%), 4.6 ± 0.7 Group 2: 6/6 (100%), 2.8 ± 0.7 Group 3: 5/7 (71%), 2.0 ± 0.7* *F (24 wk):* Group 1: 4/7 (57%), 1.4 ± 0.5 Group 2: 3/7 (43%), 0.7 ± 0.4 Group 3: 2/8 (25%), 0.4 ± 0.3	*$P < 0.05$ (decrease)	Not a full carcinogenesis bioassay Liver tumours were mainly hepatocellular carcinomas, with some neoplastic nodules

F, female; M, male; NDEA, *N*-nitrosodiethylamine; wk, week

Table 3.10 Studies of carcinogenicity in rats exposed perinatally to a mixture of non-*ortho* PCBs, PCDDs, and PCDFs

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: Incidence of tumours	Significance	Comments
Sprague-Dawley Charles River, St-Constant, QC (F) 32 wk Desaulniers *et al.* (2004)	Mixture (5 mL/kg bw) in corn oil given to neonates at age 1, 5, 10, 15, and 20 days, by gavage. Mixture contained 0 (vehicle), 1, 10, 100, or 1000 times the amount a human baby would consume. MNU was injected intraperitoneally (30 mg/kg bw in saline) at age 50 days. The rats were killed at age 32 wk	*Mammary gland:*		Purity, NR Short duration; not a full carcinogenicity bioassay The concentrations of each chemical included in the mixture (three non-*ortho* PCBs [PCB-77, PCB-126, and PCB-169], six PCDDs and seven PCDFs) were based on the concentrations found in human milk fat Description of benign lesions of the mammary gland did not differentiate between non-neoplastic (hyperplasia) and neoplastic (adenoma, fibroadenoma) lesions Mixture included PCDDs and PCDFs, so conclusions could not be made regarding the effect of PCBs alone
	Without MNU: vehicle (controls), 1000× mixture	Benign lesions (adenoma, fibroadenoma, hyperplasia): 4/37, 11/37* Malignant (carcinoma in situ and adenocarcinoma): 1/37, 4/37	* $P < 0.05$	
	With MNU: vehicle (controls), 1 × mixture, 10 × mixture, 100 × mixture, 1000 × mixture 31–40/group	Benign lesions: 11/35, 8/32, 14/32, 12/31, 10/40 Malignant tumours: 24/35, 18/32, 19/32, 21/31, 25/40	NS	

F, female; M, male; MNU, *N*-methyl-*N*-nitrosourea; PCBs, polychlorinated biphenyls; PCDDs, polychlorinated dibenzodioxins; PCDFs, polychlorinated dibenzofurans; wk, week

Table 3.11 Study of carcinogenicity in rats exposed perinatally to a mixture of PCBs, DDT, and DDE found in breast milk

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%) of tumours	Significance	Comments
Sprague-Dawley (F) 308 days or when tumour size reached 1 cm Desaulniers *et al.* (2001)	Neonates treated by gavage at age 1, 5, 10, 15, and 20 days with a mixture[a] containing 0 (vehicle), 10, 100 or 1000 times the amount of PCBs, DDT, DDE that a human baby would consume. A separate group received TCDD at 2.5 µg/kg bw by gavage on day 18. On day 21, groups 3–7 received a single intraperitoneal injection of MNU at 30 mg/kg bw in saline Group 1: corn oil vehicle controls Group 2: 1000 × mixture Group 3: MNU + corn oil vehicle Group 4: MNU + 10 × mixture Group 5: MNU + 100 × mixture Group 6: MNU + 1000 × mixture Group 7: MNU + TCDD 33–41/group	*Mammary gland* Groups 1 and 2: Fibroadenoma: 1/30, 0/33 Adenoma: 0/30, 0/33 Papilloma: 0/30, 0/33 Carcinoma in situ: 0/30, 1/33 Adenocarcinoma: 0/30, 0/33 Benign or malignant lesions (combined): 1/30, 2/33 Groups 3–7: Fibroadenoma: 12/41, 13/28, 6/31, 9/34, 10/32 Adenoma: 5/41, 4/28, 4/31, 8/34, 6/32 Papilloma: 3/41, 1/28, 3/31, 1/34, 5/32 Carcinoma in situ: 5/41, 5/28, 8/31, 7/34, 4/32 Adenocarcinoma: 11/41, 12/28, 10/31, 12/34, 13/32 Benign or malignant lesions (combined): 28/41, 24/28, 22/31, 25/34, 25/34 Benign or malignant lesions (median number of lesions): 2, 2, 1, 4.5*, 5.5	Group 2 vs group 1: NS Groups 4–7 vs group 3: NS for incidence *P = 0.05	Purity, NR Mixture included DDT and DDE, so conclusions could not be made regarding the effect of PCBs alone Not a full carcinogenesis bioassay

[a] Mixture consists of *p,p´*-dichlorodiphenyltrichloroethane (DDT), *p,p´*-dichlorodiphenyldichloroethene (DDE) and PCBs mixture comprised of non-*ortho* (PCB-77, -126, -169), mono-*ortho* (PCB-28, -66, -74, -118, -156) and di-*ortho* (PCB-99, -128, -138, -153, -170, -180, -183, -187, -194, -201, -203) substituted congeners detected in > 75% of breast milk samples from Canadian women. DDT, DDE and PCBs were included in the mixture according to the median concentrations in milk fat

MNU, *N*-methyl-*N*-nitrosourea; NS, not significant; PCB, polychlorinated biphenyl; TCDD, 2,3,7,8-tetrachlorodibenzo-*para*-dioxin; vs, versus

(Desaulniers *et al.*, 2001). The PCB-congeners in the mixture were those detected in more than 75% of samples of breast milk from Canadian women and were included in proportions determined by their median concentrations measured in milk fat. The PCBs were: non-*ortho* (PCB-77, PCB-126, PCB-169), mono-*ortho* (PCB-28, PCB-66, PCB-74, PCB-118, PCB-156), and di-*ortho* (PCB-99, PCB-128, PCB-138, PCB-153, PCB-170, PCB-180, PCB-183, PCB-187, PCB-194, PCB-201, PCB-203) substituted congeners. In this study, five groups of neonatal rats were exposed to the mixture composed of DDT, its major metabolite DDE, and PCBs at 0 (corn oil), 10, 100, or 1000 times their concentrations in breast milk, by gavage, starting at age 1, 5, 10, 15, or 20 days. For comparison purposes, an additional group was exposed by gavage at age 18 days to 2,3,7,8-tetrachlorodibenzo-*para*-dioxin (TCDD) at a concentration of 2.5 µg/kg bw. On day 21, all treatment groups (except for a control group that received corn oil only, and a group that received the 1000-times mixture) received a single intraperitoneal injection of MNU (30 mg/kg bw) in saline. Animals were observed up to 308 days. Seven to nine rats from the groups not exposed to MNU were killed between ages 55 and 62 days; the remaining rats were killed at 224 days. MNU-treated rats were killed when palpable tumours reached 1 cm, or by day 308 if no palpable tumour was detected. Sporadic incidences of lesions of the mammary gland were observed in the groups not treated with MNU (0 and 1000-times mixture). On the contrary, a large number of lesions of the mammary gland (including hyperplasia, the most common lesion observed) were seen in MNU-treated rats, and there was a significant effect of the 1000-times mixture ($P = 0.05$) on the median number of combined benign and malignant lesions of the mammary gland when compared to the MNU-only treated rats. There was no significant effect on the incidence of any specific tumour type, either benign or malignant, or the combined incidence of benign and malignant neoplasms. [Given that the mixture contained DDT and DDE, in addition to PCBs, the Working Group considered this study as a co-carcinogenicity study, and conclusions regarding the effect of PCBs alone could not be made.]

3.2.7 PCB metabolites: 4′-OH-PCB-30 and 4′-OH-PCB-61

See Table 3.12

Mouse

Neonatal female BALB/cCrg1 mice were exposed 16 hours after birth onwards to: 20 or 200 µg of 2′,4′,6′-trichloro-4-biphenylol [4′-OH-PCB-30]; 40 or 400 µg of 2′,3′,4′,5′-tetrachloro-4-biphenylol [4′-OH-PCB-61]; 10 µg of 4′-OH-PCB-30 plus 10 µg of 4′-OH-PCB-61, or 100 µg of 4′-OH-PCB-30 plus 100 µg of 4′-OH-PCB-61 (Martinez *et al.*, 2005). Exposure occurred via daily subcutaneous injections for 5 days and the mice were held for 20 months. [The neonatal mouse model has previously been used as a model for diethylstilbestrol-induced carcinogenesis after exposure in utero. The BALB/c mouse is known to be sensitive to the induction of cervicovaginal tumours by estrogens.] Significant treatment-related increases in the incidence of cervicovaginal tumours were observed for the groups treated with 4′-OH-PCB-30. Modest but statistically significant increases in the incidence of cervicovaginal tumours were also seen in both groups exposed to 4′-OH-PCB-61, and to the combination of 4′-OH-PCB-30 + 4′-OH-PCB-61 at the higher dose. There was also a significant effect of 4′-OH-PCB-61 at the lower dose on the incidence of carcinoma of the mammary gland.

Table 3.12 Study of carcinogenicity in mice exposed perinatally to 2′,4′,6′-trichloro-4-biphenylol (OH-PCB-30) and/or 2′,3′,4′,5′-tetrachloro-4-biphenylol (4′-OH-PCB-61)

Strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence of tumours	Significance	Comments
BALB/cCrgl (F) Up to 20 mo Martinez *et al.* (2005)	Daily subcutaneous injections of 20 µL for 5 days starting 16 hours after birth. Mice were weaned at age 21 days. Examination daily for premature vaginal opening for the first 35 days of life and checks monthly to detect concretions. When concretions were found, the mice were removed from the study. All mice that survived to age 20 mo were killed Groups were injected with: sesame oil vehicle (control); 20 µg OH-PCB-30; 200 µg OH-PCB-30; 40 µg OH-PCB-61; 400 µg OH-PCB-61; 10 µg OH-PCB-30 + 10 µg OH-PCB-61; or 100 µg OH-PCB-30 + 100 µg OH-PCB-61 Number/group, NR	Cervicovaginal tract carcinoma: 0/33, 2/33, 10/22**, 4/30*, 5/24*, 3/36, 8/21* Mammary gland carcinoma: 0/33, 5/33, 0/22, 4/30*, 1/24, 3/36, 0/21	*$P < 0.05$ (Fisher exact test) **$P < 0.01$ (Fisher exact test)	Purity, NR The BALB/c mouse is sensitive to the induction of cervicovaginal tumours by estrogens. The inbred BALB/cCrgl strain has a low incidence of tumours of the mammary gland. The neonatal mouse model has previously been used as a model for diethylstilbestrol-induced carcinogenesis after exposure in utero Carcinomas of the cervicovaginal tract were mainly squamous cell carcinomas and adenosquamous carcinomas

F, female; mo, month; NR, not reported; PCB, polychlorinated biphenyl

3.3 Initiation–promotion and co-carcinogenicity studies

See Table 3.13

3.3.1 Initiation–promotion studies

(a) PCB-153

A study was carried out to determine whether PCB-153 had promoting activity in NDEA-initiated tumours of the liver in male B6129SF2/J mice, and whether the deletion of the NF-κB p50 subunit influenced liver carcinogenesis (Glauert *et al.*, 2008). Four groups of 14–17 wildtype and transgenic mice were injected intraperitoneally with NDEA (90 mg/kg bw in saline) at 9 weeks of age. After a 2-week recovery period, both wildtype and NF-κB $p50^{-/-}$ mice were injected intraperitoneally with PCB-153 at a dose of 0 (corn oil) or 300 µmol/kg bw every 14 days for a total of 20 injections. Mice were then maintained for an additional 15 weeks before being killed. Hepatocellular tumours were mainly classified as hepatocellular carcinoma. The incidence of hepatocellular tumours was higher in wildtype mice treated with PCB-153 than in wildtype mice receiving corn oil only. The deletion of p50 decreased the incidence of hepatocellular tumours in mice treated with PCB-153 or corn oil only.

(b) Aroclor 1254

(i) Mouse

In a study to determine whether Aroclor 1254 promoted the induction of liver nodules after initiation with NDEA, groups of male CD-1 mice were first given drinking-water containing NDEA at a dose of 0 or 8 µg/g bw per day, for 8 weeks (Gans & Pintauro, 1986). After 2.5 weeks, mice were given Aroclor 1254 as an intraperitoneal dose at 0 (tricaprylin/corn oil, 1/4, v/v) or 100 µg/g bw, every second week for 8 (8 mice per group) or 16 (18–19 mice per group) weeks. Aroclor 1254 did not increase the incidence of liver nodules, which were made up of type I, type II, or more commonly a mixture of type I and type II tissues. [The Working Group noted that it was not clear whether the diagnosis referred to hyperplasia and adenoma, respectively.]

Diwan *et al.* (1994) examined whether Aroclor 1254 promoted NDEA-initiated tumours of the liver in groups of 30 male DBA/2NCr × C57BL/6NCr (D2B6F1) mice. At age 5 weeks, mice were injected intraperitoneally with NDEA at a dose of 0 (tricaprylin vehicle) or 90 mg/kg bw. At age 7 weeks, mice were fed Aroclor 1254 at a dietary concentration of 175 or 350 mg/kg. The authors estimated the dose to be 0.1 or 0.2 mmol/kg bw per day based on a diet consumption of 4.5 g/day. [It was not reported whether food intake was measured.] Mice were killed after 60 weeks. The incidence of hepatocellular adenoma or carcinoma (combined) was significantly increased in both groups receiving NDEA plus Aroclor 1254 (all tumours were carcinomas) compared with the group receiving NDEA only (all tumours were adenomas). The incidences of hepatoblastoma in the group receiving Aroclor 1254 at 175 mg/kg, and of metaplastic and neoplastic glandular lesions within hepatocellular neoplasms (cholangiocellular neoplasms) in the groups receiving Aroclor 1254 at 175 and 350 mg/kg were higher [$P < 0.01$] than in the group receiving NDEA only.

Beebe *et al.* (1995) examined the promoting activity of Aroclor 1254 in the lung and liver in three strains of male mice that differ in AhR responsiveness: C57BL/6, DBA/2NCr, and B6D2F1. At age 5 weeks, groups of 23–34 mice were injected intraperitoneally with NDEA at a dose of 0 (tricaprylin vehicle) or 90 mg/kg bw. At age 8 weeks, the mice were placed on a diet containing Aroclor 1254 at a concentration of 0 or 100 mg/kg for 20 weeks. They were then left untreated for 24 weeks until being killed at age 52 weeks. Tumours of the liver were classified as hepatocellular adenoma, hepatocellular

Table 3.13 Initiation–promotion and co-carcinogenicity studies with PCBs

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Initiation–promotion studies (initiator followed by PCB)					
PCB-153	Mouse, wildtype (WT) and NF-κB p50$^{-/-}$ B6129SF2/J mice (M) 55 wk Glauert *et al.* (2008)	Initiation: NDEA (90 mg/kg, i.p.) at age 9 wk Promotion: 2 wk later, PCB-153 (300 µmol/kg bw in corn oil) by i.p. injection, every 14 days; total of 20 injections; then maintained for an additional 15 wk. 14–17/group	*Hepatocellular tumours* Corn oil controls: WT, 11/15 NF-κB p50$^{-/-}$, 5/11 PCB-153: WT, 7/7* NF-κB p50$^{-/-}$, 6/9	*[$P < 0.05$] vs WT mice receiving corn oil	Hepatocellular tumours were mainly carcinomas
Aroclor 1254	Mouse, CD-1 (M) 8 or 16 wk Gans & Pintauro (1986)	*Initiation:* NDEA, 0 (control) or 8 µg/g bw per day, in drinking-water, for 8 wk *Promotion:* 2.5 wk later, Aroclor 1254 at 100 µg/g bw in tricaprylin/corn oil vehicle, i.p. every other wk for 8 (8/group) or 16 wk (18–19/group)	Liver nodules of types I and II *8 wk:* Control + vehicle: 0/8 NDEA + vehicle: 2/8 Control + Aroclor 1254: 0/8 NDEA + Aroclor 1254: 2/8 *16 wk:* Control + vehicle: 0/18 NDEA + vehicle: 9/19 Control + Aroclor 1254: 1/18 NDEA + Aroclor 1254: 10/18	NS (effect of Aroclor 1254)	It was uncertain whether liver nodules included hyperplasias and adenomas Types not further identified
Aroclor 1254	Mouse, D2B6F1 (M) 60 wk Diwan *et al.* (1994)	*Initiation*: NDEA (0 or 90 mg/kg bw in saline, i.p.) at age 5 wk *Promotion*: Aroclor 1254 at 0, 175 or 350 mg/kg diet, at age 7 wk 30/group	*Hepatocellular adenoma or carcinoma (combined)* NDEA: 12/30 (3.4), 24/24* (10.8), 23/23* (16.9) Saline: 7/30 (1.1), 12/29 (2.1), 25/25 (2.9) *Hepatoblastoma* NDEA: 1/30 (1), 8/24* (1.5), 2/23 (1) Saline: NR, 0/29, 0/25 *Cholangiocellular tumours:* NDEA: 0/30, 7/24*, 17/23* Saline: NR, 2/29, 10/25	*$P < 0.00001$ *[$P < 0.01$] *[$P < 0.01$]	In both NDEA + Aroclor 1254 groups all tumours were carcinomas whereas in the NDEA-only group all tumours were adenomas

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Aroclor 1254	Mouse, C57BL/6, DBA/2NCr, and B6D2F1 (M) 44 wk Beebe *et al.* (1995)	*Initiation*: NDEA (90 mg/kg bw, i.p.) or tricaprylin vehicle at age 5 wk *Promotion*: at age 8 wk, Aroclor 1254 (100 mg/kg diet) for 20 wk followed by no-exposure phase of 24 wk Group 1: Tricaprylin Group 2: NDEA Group 3: NDEA+Aroclor 1254 Group 4: Tricaprylin+Aroclor 1254 23–34/group	*C57BL/6* Liver tumours (all types): 0/27, 4/28, 19/32*, 2/27 Hepatocellular adenoma: 0/27, 4/28, 17/32**, 2/27 Hepatocellular carcinoma: 0/27, 3/28, 3/32, 0/27 Cholangioadenoma or cholangiocarcinoma (combined): 0/27, 0/28, 4/32, 0/27 Hepatoblastoma: 0/27, 0/28, 4/32, 0/27 Lung tumours (all): 1/27, 20/26, 20/25, 1/27 *B6D2F1* Liver tumours (all types): 0/34, 7/33, 8/33, 3/34 Hepatocellular adenoma: 0/34, 6/33, 6/33, 3/34 Hepatocellular carcinoma: 0/34, 0/33, 2/33, 0/34 Cholangioadenoma or cholangiocarcinoma (combined): 0/34, 0/33, 0/33, 0/34 Hepatoblastoma: 0/34, 1/33, 0/33, 0/34 Lung tumours (all): 0/31, 33/34, 31/34, 2/34	*$P < 0.05$ (group 3 vs group 2) **$P < 0.05$ (group 3 vs group 2 and group 3 vs group 4)	Purity, NR

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Aroclor 1254 (cont.)			*DBA/2* Liver tumours (all types): 0/23, 6/28, 6/31***, 0/24 Hepatocellular adenoma: 0/23, 5/28, 4/31, 0/24 Hepatocellular carcinoma: 0/23, 2/28, 2/31, 0/24 Cholangioadenoma or cholangiocarcinoma (combined): 0/23, 0/28, 0/31, 0/24 Hepatoblastoma: 0/23, 0/28, 0/31, 0/24 Lung tumours (all): 3/23, 24/28, 28/29, 1/24	***$P < 0.05$ (group 3 vs group 4)	
Aroclor 1254	Mouse, HRS/1 hairless (F) 20 wk Poland *et al.* (1982)	*Initiation*: MNNG (5 µmol in 50 µl of acetone) at age 8 wk *Promotion*: 1 mg Aroclor 1254 in 50 µL of acetone per mouse, twice weekly topically for 20 wk 20 mice in groups receiving Aroclor 1254; 26 in MNNG-only group	Skin papilloma: MNNG + vehicle, 0/23 Vehicle + Aroclor 1254, 0/19 MNNG + Aroclor 1254, 4/19	NS	Statistical test, NR

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Aroclor 1254	Mouse, Swiss (Cr:NIH) (M, F) 44 wk Beebe *et al.* (1993)	*Initiation:* For transplacental studies, pregnant mice were injected with NNK (100 mg/kg bw, i.p.) on days 15, 17, and 19 of gestation, or with NDMA (10 mg/kg bw, i.p.) on day 19 of gestation, or with saline vehicle on day 19 of gestation For neonatal studies, pups were injected with NDMA (5 mg/kg bw, i.p.), NNK (50 mg/kg bw, i.p.), or saline vehicle on PND 4 *Promotion:* Aroclor 1254 (500 mg/kg bw, p.o.) or olive oil vehicle on PND 56 *Transplacental initiation* Group 1: Saline/olive oil Group 2: Saline/Aroclor 1254 Group 3: NDMA/olive oil Group 4: NDMA/Aroclor 1254 Group 5: NNK/olive oil Group 6: NNK/Aroclor 1254 *Neonatal initiation* Group 7: NDMA/olive oil Group 8: NDMA/Aroclor 1254 Group 9: NNK/olive oil Group 10: NNK/Aroclor 1254 Animals/group, NR	*Transplacental initiation* Lung tumours (M): 2/27, 3/30, 0/29, 10/28*, 1/27, 8/29** Lung tumours (F): 1/29, 2/30, 3/30, 4/30, 4/30, 5/29 *Neonatal initiation* Lung tumours (M): 11/28, 22/30***, 8/30, 10/30 Lung tumours (F): 16/27, 19/27, 4/30, 11/29****	*$P < 0.001$ (group 4 vs group 3) **$P = 0.026$ (group 6 vs group 5) ***$P = 0.016$ (group 8 vs group 7) ****$P = 0.039$ (group 10 vs group 9)	Purity, NR The classification of lung tumours was not provided
Aroclor 1254	Rat, Sprague-Dawley (M) 18 wk Preston *et al.* (1981)	*Initiation*: NDEA at 66 µg/mL in drinking-water for 5 wk *Promotion*: Aroclor 1254 or Aroclor 1254 from which PCDFs were removed at 100 mg/kg diet, or control diet 40/group	Hepatocellular carcinoma: NDEA alone, 5/32 NDEA + Aroclor 1254, 21/33* NDEA + Aroclor 1254 with PCDFs removed, 27/32*	*$P < 0.05$, χ^2 analysis	
Aroclor 1254	Rat, Sprague-Dawley (M) 19 wk Vansell *et al.* (2004)	*Initiation*: DIPN (2.5 g/kg bw, s.c.) *Promotion*: 1 wk later, Aroclor 1254 at 100 mg/kg diet for 19 wk 24/group	*Thyroid* Cystic adenoma: 0/24, 2/22 Follicular adenoma: 5/24, 9/22 Follicular carcinoma: 1/24, 0/22 "Complete carcinoma": 0/24, 4/22*	*$P < 0.05$	Uncertainty in classification of one type of thyroid tumour as "complete carcinoma"

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Kanechlor 400	Rat, Donryu (F) 6 mo Kimura *et al.* (1976)	*Initiation*: MDAB (600 mg/kg diet) for 2 mo, rats aged 11–15 wk Treatment with Kanechlor 400 at 400 mg/kg diet before, during, or after MDAB Two groups were treated with Kanechlor 400 or MDAB only 25/group; 10 untreated controls	Hepatocellular carcinoma: MDAB alone, 2/15 MDAB followed by Kanechlor 400, 7/11* Kanechlor 400 followed by MDAB, 0/9 MDAB/Kanechlor 400 together, 0/11 Kanechlor 400 alone, 0/12 Untreated controls, 0/7	[*$P < 0.05$] vs MDAB-only group	The authors indicated that the incidence in the group receiving MDAB followed by Kanechlor 400 was significantly different from that in all other groups, using *t*-test, but the Working Group noted that this test cannot be used for binomial data
Kanechlor 500	Rat, Wistar (M) 40 or 52 wk Nishizumi (1979)	*Initiation*: NDEA at 50 mg/L in drinking-water for 2 wk *Promotion*: 1 wk later, 0.1 mL of 10% Kanechlor 500 in olive oil, by gavage, twice per week for 12 wk, then maintained until 40 or 52 wk after start of study 7–8/group per time-point	Hepatocellular tumours (mainly carcinomas): *40 wk:* NDEA + olive oil: 0/8 NDEA + Kanechlor 500: 6/7* (3.3 tumours/rat)** *52 wk:* NDEA + olive oil: 0/8 NDEA + Kanechlor 500: 8/8* (6.9 tumours/rat)**	*[$P < 0.05$] **$P < 0.01$	
Unspecified PCB mixture	Rat, F344 (M) 32 wk Hirose *et al.* (1981)	*Initiation*: 0.1% EHEN in drinking-water for 2 wk *Promotion*: 0 or 0.05% unspecified PCB mixture in diet for 32 wk UN 1 wk after starting PCBs 20–21/group	Hepatocellular carcinoma: EHEN only, 7/21 EHEN + PCB, 19/19 Renal cell tumours [benign]: EHEN, 18/21 EHEN + PCB, 12/19	$P < 0.001$ NS	PCB mixture: Kanegafuchi Chemical Co., Osaka, Japan No renal cell carcinomas were observed Statistical analysis, NR

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Unspecified PCB mixture	Rat F344 (M) 32 wk Arai *et al.* (1983)	*Initiation*: NDMA (0.04% in diet) for 2 wk *Promotion*: 2 wk later, 500 mg/kg diet PCB mixture (or a basal diet) for 28 wk; UN 1 wk after starting PCBs 20/group	*Liver* Hyperplastic or neoplastic nodules (combined): NDMA, 5/18 NDMA + UN, 7/20 NDMA + PCBs, 10/11* NDMA + PCBs + UN, 7/7* Hepatocellular carcinoma: NDMA, 0/18 NDMA + UN, 0/20 NDMA + PCBs, 3/11* NDMA + PCBs + UN, 1/7 *Kidney* Nephroblastoma: NDMA, 17/18 NDMA + UN, 18/20 NDMA + PCBs**, 4/11 NDMA + PCBs + UN**, 3/7	*[$P < 0.05$] vs control group **[$P < 0.05$] vs control group (decrease)	PCB mixture: Kanegafuchi Chemical Co., Osaka, Japan Statistical analysis, NR Significant mortality in some groups, especially in the group receiving NDMA + PCBs + UN
PCBs with other modifying agents					
Aroclor 1254	Mouse, C57BL/10ScSn and DBA/2 2, 4, 8, and 12 mo Smith *et al.* (1990)	Injection with Fe (Fe-dextran, 12 mL/kg; Fe, 600 mg/kg bw, s.c.) or dextran followed 7 days later by Aroclor 1254 at 100 mg/kg diet for 2 mo (5 mice/group), 4 mo (C57 only, 5 mice/group), 8 mo (10 mice/group for C57; 5–7 group for DBA), or 12 mo (C57 only, 15–19/group)	*Hepatocellular adenoma:* 4 mo: Aroclor 1254, 0/5 Aroclor 1254 + Fe, 1/5 8 mo (C57): Aroclor 1254, 0/10 Aroclor 1254 + Fe, 7/9* 12 mon: Aroclor 1254, 0/16 Aroclor 1254 + Fe, 15/18* *Hepatocellular carcinoma:* 12 mo only: Aroclor 1254, 1/16 Aroclor 1254 + Fe, 7/18*	*[$P < 0.05$]	Statistical analysis, NR No effects of iron and Aroclor 1254 in DBA/2 mice

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Aroclor 1254	Mouse, C57BL/10ScSn 8 and 12 mo Smith *et al.* (1995)	Injection with Fe-dextran (Fe, 600 mg/kg bw, s.c.) or dextran, followed 3 days or 1 wk later by Aroclor 1254 at 100 mg/kg diet; for 8 mo (10/group) or 12 mo (15–19/group) Group 1: Aroclor Group : Aroclor + Fe	*8 mo:* Group 1: 0/10 (hepatocellular tumours); Group 2: 7/9* (hepatocellular adenoma) *12 mo:* Group 1: 0/16 (hepatocellular tumours); Group 2: 15/18* (hepatocellular adenoma) and 7/18* (hepatocellular carcinoma)	*[$P < 0.05$]	Statistical analysis, NR
Aroclor 1254	Mouse, C57BL/6J (M), *Cyp1a2*$^{-/-}$ or $^{+/+}$ (wildtype) 57 wk Greaves *et al.* (2005)	Injection with Fe-dextran (Fe, 800 mg/kg bw; route NR) followed by Aroclor 1254 at 100 mg/kg diet for 57 wk Fe + Aroclor 1254, 10/group Fe-only, 5/group	Liver adenoma: Fe-only: *Cypla2*$^{+/+}$: 0/5 *Cypla2*$^{-/-}$: 0/5 Fe + Aroclor: *Cypla2*$^{+/+}$: 5/10* *Cypla2*$^{-/-}$: 0/10	*[NS]	Statistical analysis, NR
Kanechlor 400	Mouse, A/J (M) 24 wk Nakanishi *et al.* (2001)	Single dose of Kanechlor 400 (2.5 mg/kg bw, i.p.) or DMSO vehicle injected into mice aged 6 wk. Mice were then injected with 1-nitropyrene at 1575 mg/kg bw (total dose of all injections) or DMSO vehicle (i.p., 3×/wk), 17 injections. Mice killed 18 wk after final injection of 1-nitropyrene 8–20/group	*Bronchioloalveolar lesions* Incidence (average number): DMSO control: 0/8 (0) Kanechlor 400: 2/10 (0.4) 1-Nitropyrene: 16/20 (1.8) Kanechlor 400 + 1-nitropyrene: 13/13 (3.2)* Number: DMSO control: 0 Kanechlor 400: 2 hyperplasias, 2 adenomas; 1-Nitropyrene: 10 hyperplasias, 20 adenomas, 3 adenocarcinomas; 1-Nitropyrene + Kanechlor 400: 15 hyperplasias, 23 adenomas, 8 adenocarcinomas	*$P < 0.01$ compared with 1-nitropyrene group	Statistical analysis, NR for incidence and number of lesions

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Kanechlor 400 and Kanechlor 500	Mouse, dd (M) 24 wk Nagasaki *et al.* (1975)	Dietary administration for 24 wk: α-BHC (250 mg/kg) α-BHC (250 mg/kg) + Kanechlor 500 (250 mg/kg) α-BHC (250 mg/kg) + Kanechlor 400 (250 mg/kg) α-BHC (100 mg/kg) α-BHC (100 mg/kg) + Kanechlor 500 (250 mg/kg) α-BHC (100 mg/kg) + Kanechlor 500 (100 mg/kg) α-BHC (100 mg/kg) + Kanechlor 400 (250 mg/kg) α-BHC (100 mg/kg) + Kanechlor 400 (100 mg/kg) α-BHC (50 mg/kg) α-BHC (50 mg/kg) + Kanechlor 500 (250 mg/kg) α-BHC (50 mg/kg) + Kanechlor 500 (100 mg/kg) α-BBC (50 mg/kg) + Kanechlor 400 (250 mg/kg) α-BHC (50 mg/kg) + Kanechlor 400 (100 mg/kg) Kanechlor 500 (250 mg/kg) Kanechlor 500 (100 mg/kg) Kanechlor 400 (250 mg/kg) Kanechlor 400 (100 mg/kg) 20–38/group	*Liver* Nodular hyperplasia: 30/38, 16/20, 26/30, 0/20, 8/25, 3/24, 4/29, 0/27, 0/20, 9/30, 0/28, 0/28, 0/27, 0/20, 0/20, 0/20, 0/20 Hepatocellular carcinoma: 10/38, 11/20*, 15/30*, 0/20, 1/25, 0/24, 0/29, 0/27, 0/20, 2/30, 0/28, 0/28, 0/27, 0/20, 0/20, 0/20, 0/20	*[$P < 0.05$] compared with α-BHC (250 mg/kg) group	The chemical is erroneously reported as benzene hexachloride and is actually hexachlorocyclohexane Statistical analysis, NR

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
Kanechlor 500	Mouse, dd (M) 24 wk Ito *et al.* (1973)	BHC, (α, β, or γ isomers) (50, 100 or 250 mg/kg diet) for 24 wk ± Kanechlor 500 (250 mg/kg diet) for 24 wk 25–30/group	*Liver nodular hyperplasia* α-BHC: 0/28, 0/26, 23/30 α-BHC + Kanechlor 500: 9/30, 8/25*, 21/26 β-BHC: 0/28, 0/26, 0/26 β-BHC + Kanechlor 500: 0/29, 5/30*, 16/29* *Hepatocellular carcinoma* α-BHC: 0/28, 0/26, 8/30 α-BHC + Kanechlor 500: 2/30, 1/25, 15/26* β-BHC: 0/28, 0/26/, 0/26 β-BHC + Kanechlor 500: 0/29, 1/30, 6/29* γ-BHC (all doses) and γ-BHC (all doses) + Kanechlor 500: no tumours (0/26–30)	*[$P < 0.05$]	The chemical is erroneously reported as benzene hexachloride and is actually hexachlorocyclohexane Statistical analysis, NR
PCB-77	Rat, Sprague-Dawley (F) 10.5 wk Nesaretnam *et al.* (1998)	Single dose of DMBA at 10 mg by gavage in 0.5 mL corn oil at age 50 days PCB-77 treatment: single dose at 10 mg/kg bw by gavage at the same time as DMBA, then in the diet at 500 mg/kg for one additional wk ($n = 2 \times 20$); or DMBA only ($n = 2 \times 20$) Rats were then fed either a low-fat (5%) ($n = 2 \times 20$) or a high-fat (20%) diet ($n = 2 \times 20$) Total: 4 groups of 20 rats Group 1: DMBA+PCB-77 + low fat Group 2: DMBA+PCB-77 + high fat Group 3: DMBA + low fat Group 4: DMBA + high fat	Mammary gland tumours (mainly mammary ductal carcinoma)	Number of palpable tumours: $P < 0.005$ for group 2 vs group 4 and group 1 vs group 3 at 8, 9, and 10 wk Incidence at 10.5 wk: $P < 0.05$ for group 1 (60%) vs group 3 (15%)	It was unclear whether the rats not treated with PCB-77 were given the vehicle instead Data were presented graphically

Table 3.13 (continued)

PCB congener or mixture	Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%), and/or multiplicity of tumours	Significance	Comments
PCB-126, PeCDF, and TCDD	Rat, Harlan Sprague-Dawley (F) 104 wk NTP (2006e)	PCB-126, TCDD and PeCDF in corn oil : acetone (99 : 1) by gavage 5 days/wk for 104 wk at doses of: 0 ng TEQ/kg bw (controls); 10 ng TEQ/kg bw (3.3 ng/kg TCDD, 6.6 ng/kg PeCDF, 33.3 ng/kg PCB 126); 22 ng TEQ/kg bw (7.3 ng/kg TCDD, 14.5 ng/kg PeCDF, 73.3 ng/kg PCB 126); 46 ng TEQ/kg bw (15.2 ng/kg TCDD, 30.4 ng/kg PeCDF, 153 ng/kg PCB-126); and 100 ng TEQ/k bw (33 ng/kg TCDD, 66 ng/kg PeCDF, 333 ng/kg PCB 126) 81 rats/group Interim evaluations: up to 10 rats/group were evaluated at 14, 31, and 53 wk	*Liver* Hepatocellular adenoma: 0/53, 1/53, 1/53, 1/53, 11/51* Cholangiocarcinoma: 0/53, 0/53, 2/53, 7/53*, 9/51** *Lung* Cystic keratinizing epithelioma: 0/53, 0/53, 0/53, 2/53, 20/53*	*$P < 0.001$ $P < 0.001$ (trend) *$P = 0.011$ **$P < 0.001$ $P < 0.001$ (trend) *$P < 0.001$ $P < 0.001$ (trend)	*Non-neoplastic lesions* Liver: hepatocyte hypertrophy, multinucleated hepatocytes, pigmentation, inflammation, diffuse fatty change, bile duct hyperplasia, oval cell hyperplasia, nodular hyperplasia, eosinophilic focus, cholangiofibrosis, bile duct cysts, necrosis, portal fibrosis, mixed cell focus, and toxic hepatopathy Lung: squamous metaplasia

DIPN, *N*-nitroso diisopropanolamine; DMBA, 7,12-dimethylbenz[*a*]anthracene; EHEN, *N*-ethyl-*N*-hydroxyethylnitrosamine; i.p., intraperitoneal; MDAB, 3′-methyl-4-dimethylaminoazobenzene; MNNG, *N*-methyl-*N*'-nitrosoguanidine; mo, month; MNU, *N*-methyl-*N*-nitrosourea; NDEA, *N*-nitrosodiethylamine; NDMA, *N*-nitrosodimethylamine; NNK, 4-(*N*-nitrosomethylamino)-1-(3-pyridyl)-1-butanone; NR, not reported; NS, not significant; PCB, polychlorinated biphenyl; PCDF, polychlorinated dibenzofuran; PeCDF, 2,3,4,7,8-pentachlorodibenzofuran; s.c., subcutaneous; TCDD, 2,3,7,8-tetrachlorodibenzo-*para*-dioxin; TPA, 12-*O*-tetradecanoylphorbol-l3-acetate; UN, unilateral nephrectomy; wk, week

carcinoma, cholangioadenoma, cholangiocarcinoma, or hepatoblastoma. [The classification of tumours of the lung was not described.] In NDEA-treated DBA/2NCR mice and B6D2F1 mice, Aroclor 1254 did not affect the incidence or multiplicity of tumours of the liver (all or any of the various types) when compared with mice receiving NDEA only. In NDEA-treated C57BL/6 mice, Aroclor 1254 increased the incidences of tumours of the liver (all types combined) and of hepatocellular adenoma. The incidence or multiplicity of tumours of the lung was not affected by treatment with NDEA and Aroclor 1254 in any strain when compared with mice receiving NDEA only.

Poland *et al.* (1982) investigated whether Aroclor 1254 could promote *N*-methyl-*N*′-nitro-*N*-nitrosoguanidine (MNNG)-initiated skin papillomas in female HRS/1 hairless mice. At age 8 weeks, mice were given 5 µmol of MNNG (in 50 µl of acetone) or the vehicle topically. Mice were then given a topical application of 1 mg of Aroclor 1254 (in 50 µl of acetone) per mouse, twice per week, for 20 weeks. There were 20 mice in the groups receiving MNNG plus Aroclor 1254, or Aroclor 1254 only, and 26 in the MNNG only-treated group. Aroclor 1254 did not promote MNNG-initiated tumours, and there was no neoplastic effect of Aroclor 1254 in non-initiated mice. [The statistical test was not reported.]

Beebe *et al.* (1993) investigated whether Aroclor 1254 could promote tumours of the lung and liver initiated by NDMA or 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), either neonatally or transplacentally, in male and female Swiss (Cr:NIH) mice. For transplacental studies, pregnant mice were injected intraperitoneally with NNK at a dose of 0 (saline vehicle) or 100 mg/kg bw on days 15, 17, and 19 of gestation, or with NDMA at a dose of 0 (saline vehicle) or 10 mg/kg bw on day 19 of gestation. For the neonatal studies, infant mice were injected with NDMA (5 mg/kg bw), NNK (50 mg/kg bw), or saline vehicle on postnatal day 4. Mice were then given Aroclor 1254 by gavage (500 mg/kg bw) or olive oil vehicle for 44 weeks starting at age 56 days. There were 27–30 mice in all groups when the mice were killed at age 52 weeks. In females, transplacental exposure to NNK or NDMA plus Aroclor 1254 did not increase the incidence of tumours of the lung or liver compared with controls treated with NNK or NDMA only. In males, Aroclor 1254 increased the incidence of tumours of the lung (but not of the liver) initiated by either NDMA or NNK transplacentally. In females, Aroclor 1254 increased the incidence of tumours of the lung initiated neonatally by NNK, but not by NDMA. In males, Aroclor 1254 increased the incidence of tumours of the lung initiated neonatally by NDMA, but not by NNK. [The classification of tumours of the lung was not provided.]

(ii) Rat

Preston *et al.* (1981) investigated whether Aroclor 1254 promotes chemically-induced hepatocarcinogenesis in male Sprague-Dawley rats. Three groups of 40 rats were first given drinking-water containing NDEA at a concentration of 66 µg/mL for 5 weeks as an initiating agent. The rats were then fed an unrefined diet containing Aroclor 1254 at a concentration of 100 mg/kg, or Aroclor 1254 from which PCDFs (present as impurities) had been removed, or control diet. Rats were fed the diets for 18 weeks and then killed. Lesions of the liver were classified as foci of cellular alteration, neoplastic nodules, hepatocellular carcinoma, cholangioma, or cholangiocarcinoma. The administration of either Aroclor 1254, or Aroclor 1254 without PCDFs, significantly increased the incidences of NDEA-initiated hepatocellular carcinoma.

Vansell *et al.* (2004) studied whether Aroclor 1254 could promote tumours of the thyroid initiated by *N*-nitrosodiisopropanolamine (DIPN) in male Sprague-Dawley rats. Rats were first injected subcutaneously with DIPN at 0 (saline) or 2.5 g/kg bw. After a 1-week recovery period,

rats were fed a diet containing Aroclor 1254 at a concentration of 100 mg/kg for 19 weeks and then killed. Tumours were classified as thyroid cystic adenoma, thyroid follicular adenoma, thyroid follicular carcinoma, or "thyroid complete carcinoma." Aroclor 1254 only significantly increased the incidence of "thyroid complete carcinoma." [The Working Group noted the uncertainty of the classification of one type of thyroid tumour as "thyroid complete carcinoma."]

(c) Kanechlor 400 and Kanechlor 500

Rat

Kimura *et al.* (1976) gave female Donryu rats (age, 11–15 weeks) diets containing Kanechlor 400 or 3′-methyl-4-dimethylaminoazobenzene (MDAB) at a concentration of 400 or 600 mg/kg, respectively. Both agents were dissolved in olive oil before being added to the diet. There were five groups of 25 rats each. A first group was treated with Kanechlor 400 for 6 months, no treatment for 2 months, and then MDAB for 2 months; a second group was treated with MDAB for 2 months, no treatment for 2 months, then Kanechlor 400 for 6 months; a third group was treated with Kanechlor 400 for 6 months with MDAB given for the last 2 months, and no treatment for 4 months; a fourth group was treated with MDAB for 2 months and no treatment for 8 months; and a fifth group treated with Kanechlor 400 for 6 months and no treatment for 4 months. Additionally a sixth group of 10 rats was maintained for 10 months with no treatment. In all groups except that given MDAB only, body weight decreased markedly compared with untreated controls. Therefore, treatment with Kanechlor 400 was discontinued for 2 weeks after 3 months of treatment, and again for 4 weeks after the second 1 month of treatment. As for survival, 9, 11, 11, 15, 12 and 7 mice remained in groups 1 to 6, respectively. Only 2 out of 15 mice receiving MDAB only developed hepatocellular carcinoma compared with 7 out of 11 mice receiving MDAB followed by Kanechlor 400 [$P < 0.05$]. [The Working Group noted that the authors calculated the incidence in the group receiving MDAB then Kanechlor 400 compared to all other groups using a *t*-test, but it is not correct to use this test for binomial data.]

In a study to determine whether Kanechlor 500 could promote NDEA-initiated carcinogenesis, groups of 7–8 male Wistar rats were given drinking-water containing NDEA at a concentration of 50 mg/L for 2 weeks (Nishizumi, 1979). After a 1-week recovery period, the rats were given Kanechlor 500 (0.1 mL of 10% Kanechlor 500 in olive oil) by gavage twice per week for 12 weeks. Rats were then maintained without further treatment until being killed 40 and 52 weeks after the start of the experiment. Data were analysed using the Student *t*-test. The incidence [$P < 0.05$] and tumour multiplicity ($P < 0.01$) of hepatocellular tumours (mainly hepatocellular carcinomas) was significantly higher in rats given NDEA plus Kanechlor 500 than in rats given NDEA only, at both 40 and 52 weeks.

(d) Unspecified PCBs

Rat

In a study to examine the effect of an unspecified PCB mixture on hepatic and renal carcinogenesis induced by *N*-ethyl-*N*-hydroxyethylnitrosamine (EHEN), two groups of 20–21 male Fischer 344 rats were given drinking-water containing 0.1% EHEN for 2 weeks, or untreated drinking-water (Hirose *et al.*, 1981). After an unspecified time, rats were placed on a diet containing 0.05% PCBs [not further specified] for 32 weeks. One week after starting the experimental diet, the right kidney was removed (unilateral nephrectomy). All rats treated with EHEN plus PCBs (19 out of 19; $P < 0.001$) developed hepatocellular carcinoma, compared with one third (7 out of 21) of the rats treated with EHEN only. Treatment with PCBs had no effect on the incidence or number of EHEN-induced

tumours of the kidney (neoplastic nodules or renal cell tumours [all benign tumours]) compared with rats receiving EHEN only. No renal cell carcinoma was observed.

In a study to determine whether an unspecified PCB mixture could promote tumours of the liver and kidney induced by NDMA, four groups of 20 male Fischer 344 rats were fed a diet containing 0.04% NDMA for 2 weeks (Arai *et al.*, 1983). After a 2-week recovery period, rats were fed a diet containing PCBs [not further specified] at a concentration of 0 (basal diet) or 500 mg/kg for 28 weeks and then killed. In some groups, unilateral nephrectomy was performed at 5 weeks (1 week after starting the PCB containing diet). Tumours of the liver were classified as hyperplastic and neoplastic nodules, and hepatocellular carcinoma. Tumours of the kidney were classified as adenoma, adenocarcinoma, and nephroblastoma. In rats receiving NDMA plus PCBs, the incidences of liver hyperplastic or neoplastic nodules (combined) and of hepatocellular carcinoma (only in non-nephrectomized rats) were higher than in the respective controls. The administration of PCBs, either with or without nephrectomy, decreased the incidence of nephroblastoma. [The Working Group noted that no statistical analysis was reported and that there appeared to be significant mortality in some groups, especially in the group receiving NDMA plus PCBs plus unilateral nephrectomy.]

3.3.2 Studies with other modifying agents

(a) PCB-77

Rat

Nesaretnam *et al.* (1998) investigated whether dietary fat could influence the effect of PCB-77 on DMBA-induced tumours of the mammary gland in female Sprague-Dawley rats. Groups of 20 female rats were given DMBA (10 mg in 0.5 mL corn oil) by gavage at age 50 days. Two groups were also given a simultaneous dose of PCB-77 at 10 mg/kg bw by gavage, then a diet containing PCB-77 at a concentration of 500 µg/g corn oil for an additional week. Two groups were not exposed to PCB-77. [It was unclear whether these rats were given the vehicle instead of PCB-77.] The four groups (treated and not treated with PCB-77) were then fed either a low-fat (5%) or a high-fat (20%) purified diet. [Fat was substituted for dextrose on a weight basis rather than on a caloric basis.] Rats were palpated weekly for tumours of the mammary gland and were killed 10.5 weeks after administration of DMBA. Tumours at autopsy were mainly classified as mammary ductal carcinoma. The number of palpable tumours of the mammary gland was significantly higher in rats fed a high-fat diet plus PCB-77 than in rats fed a high-fat diet only, at 8, 9, and 10 weeks. Similarly, the incidence of tumours of the mammary gland was higher in rats fed a low-fat diet plus PCB-77 (~60%) than in rats fed a low-fat diet (~15%) only, at 10.5 weeks. [Data were presented graphically.]

(b) Aroclor 1254

Mouse

Smith *et al.* (1990) investigated whether iron (Fe) and/or Aroclor 1254 could influence liver carcinogenesis in male C57BL/10ScSn and DBA/2 mice. Mice (age 7–10 weeks) were first injected subcutaneously with Imferon, an Fe–dextran complex (12 mL/kg; dose of Fe, 600 mg/kg bw) or an equivalent volume of dextran C solution in water (200 mg/mL). After 7 days, mice were fed a diet mixed with 2% corn oil containing Aroclor 1254 at a concentration of 100 mg/kg for 2 (5 mice/group), 4 (C57 only, 5 mice/group), 8 (C57, 10 mice/group; DBA, 5–7 mice/group), or 12 months (C57 only, 15–19 mice/group) before being killed. Tumours were classified as hepatocellular adenoma or hepatocellular carcinoma. Higher incidences of hepatocellular tumour were observed in C57 mice receiving both Fe and Aroclor 1254 at 8 months (adenomas) and 12

months (adenomas and carcinomas) compared with those receiving Aroclor 1254 only. [No statistical analyses were reported.]

Smith *et al.* (1995) studied the influence of Fe and/or Aroclor 1254 on liver carcinogenesis in male C57BL/10ScSn mice [age of mice not reported]. Mice were subcutaneously injected a Fe–dextran solution (100 mg/mL Fe, and 100 mg/mL dextran; dose of Fe, 600 mg/kg bw) or the equivalent dextran solution only. After 3 days or 1 week, mice were fed a diet containing Aroclor 1254 (0.01% of diet) and corn oil (2%) for 8 months (10 mice/group) or 12 months (15–19 mice/group). Tumours were classified as nodules [hepatocellular adenoma] or hepatocellular carcinoma. Higher incidences of hepatocellular tumours were observed in mice receiving Aroclor 1254 plus Fe for 8 months (adenomas) and 12 months (adenomas and carcinomas) than in mice receiving Aroclor 1254 only. [No statistical analyses were reported.]

Greaves *et al.* (2005) studied the effects of deletion of the *Cyp1a2* gene on the induction of tumours of the liver by Aroclor 1254 and Fe in male C57BL/6J mice. *Cyp1a2* knockout $^{(-/-)}$ and wildtype $^{(+/+)}$ mice were given a Fe–dextran solution (Fe, 800 mg/kg bw) [route not reported], followed by a diet containing Aroclor 1254 at 100 mg/kg for 57 weeks or until death. There were 10 mice in the Aroclor 1254-treated groups and 5 mice in the control groups receiving Fe only. Liver tumours were classified as adenomas. No tumours were observed in *Cyp1a2* $^{(-/-)}$ mice or in *Cyp1a2* $^{(+/+)}$ wildtype mice not receiving Aroclor 1254. No tumours were seen in the 10 *Cyp1a2* $^{(-/-)}$ mice receiving Aroclor 1254, but 5 out of 10 [not significant] of the wildtype mice receiving Aroclor 1254 developed liver adenoma. [No statistical analyses were provided.]

(c) Kanechlor 400 and Kanechlor 500

Mouse

Nakanishi *et al.* (2001) examined the effects of Kanechlor 400 on lung tumorigenesis induced by 1-nitropyrene in male A/J mice. Mice (age, 6 weeks) were given a single intraperitoneal dose of Kanechlor 400 at 0 (corn oil vehicle) or 2.5 mg/kg bw. Mice were then given 1-nitropyrene or the DMSO vehicle, three times per week (17 intraperitoneal injections for a total dose of 1575 mg/kg bw). Mice were killed 18 weeks after the last injection of 1-nitropyrene. Numbers of mice per group were as follows: DMSO controls, 8; Kanechlor 400, 10; 1-nitropyrene, 20; 1-nitropyrene plus Kanechlor 400, 13. Lung lesions were classified as bronchioloalveolar hyperplasia, adenoma, or adenocarcinoma. The incidence of lesions of the lung was increased in both groups of mice receiving 1-nitropyrene. The average number of lesions, but not incidence, was significantly greater in the group receiving Kanechlor 400 plus 1-nitropyrene than in the group receiving 1-nitropyrene only.

Nagasaki *et al.* (1975) investigated whether co-administration of Kanechlor 400 or Kanechlor 500 and α-benzene hexachloride (α-BHC) [hexachlorocyclohexane] would affect the incidence of nodular hyperplasia of the liver and hepatocellular carcinoma in male dd mice. Mice were given diets containing α-BHC at a concentration of 50, 100, or 250 mg/kg, and/or Kanechlor 400 or Kanechlor 500 (100 or 250 mg/kg), for 24 weeks. Nodular hyperplasia and hepatocellular carcinoma were observed. The incidence of hepatocellular carcinoma was higher [$P < 0.05$] in mice receiving 250 mg/kg α-BHC and the higher dose of Kanechlor 400 or Kanechlor 500, than in mice receiving only α-BHC at 250 mg/kg. No tumours were induced by Kanechlor 400 or Kanechlor 500 only. [Statistical analyses were not reported.]

A study by Ito *et al.* (1973) examined the effects of co-administration of Kanechlor 500 and one isomer of benzene hexachloride (BHC)

[hexachlorocyclohexane] on the incidence of nodular hyperplasia of the liver and hepatocellular carcinoma. Groups of male dd mice (age, 8 weeks) were given diets containing α-, β-, or γ-BHC (50, 100 or 250 mg/kg) for 24 weeks, with or without Kanechlor 500 (250 mg/kg). In some groups, Kanechlor 500 promoted the incidence of nodular hyperplasia and hepatocellular carcinoma induced by α-BHC and β-BHC. [Statistical analyses were not reported.]

(d) PCB-126, PeCDF, and TCDD

Rat

In a study by the NTP, groups of 81 female Harlan Sprague-Dawley rats were given a mixture of TCDD, PeCDF, and PCB-126 by gavage, 5 days per week, for up to 2 years (NTP, 2006e). Up to 10 rats per group were evaluated after 14, 31, and 53 weeks. Doses were formulated by using the WHO TEF values of 1.0 for TCDD, 0.1 for PCB-126, and 0.5 for PeCDF. Specific target doses were: "10 ng TEQ/kg bw" (TCDD, 3.3 ng/kg; PeCDF, 6.6 ng/kg; PCB-126, 33.3 ng/kg), "22 ng TEQ/kg bw" (TCDD, 7.3 ng/kg; PeCDF, 14.5 ng/kg; PCB-126, 73.3 ng/kg), "46 ng TEQ/kg bw" (TCDD, 15.2 ng/kg; PeCDF, 30.4 ng/kg; PCB-126, 153 ng/kg), and "100 ng TEQ/kg bw" (TCDD, 33 ng/kg; PeCDF, 66 ng/kg; PCB-126, 333 ng/kg). Rats in the control group received the corn oil : acetone vehicle (99 : 1; 2.5 mL/kg bw) only. After 2 years, there were statistically significant increases ($P < 0.001$) in the incidences of cholangiocarcinoma, hepatocellular adenoma, and cystic keratinizing epithelioma of the lung in the group at 100 ng TEQ/kg bw. The incidence of cholangiocarcinoma was also significantly increased ($P = 0.011$) in the group at 46 ng TEQ/kg. In addition, there was a significant trend in the incidence of these three types of neoplasm with increasing dose.

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4. MECHANISTIC AND OTHER RELEVANT DATA

4.1 Absorption, distribution, metabolism, and excretion

In this Section, the most recent Ballschmiter & Zell (BZ) nomenclature was used throughout (see Mills *et al.*, 2007). For the full corresponding IUPAC nomenclature, the reader is referred to Section 1.1, Tables 1.1–1.3. For the methyl sulfonyl metabolites, and wherever the nomenclature reported is unclear, the name of the metabolite is given as reported in the article, followed by, where appropriate, the abbreviation as well as the structural name (Maervoet *et al.*, 2004; Grimm *et al.*, 2015).

4.1.1 Absorption

(a) Oral exposure

(i) Humans

The absorption of polychlorinated biphenyls (PCBs) was studied in four breastfed infants in Sweden by Dahl *et al.* (1995). Absorption was measured by comparing the estimated total intake and the excretion in faeces for 48 hours, at 1, 2, and 3 months postpartum. The concentrations of 56 congeners in maternal milk were determined. For tetrachlorosubstituted to octachlorosubstituted congeners, absorption was found to be close to 100%, while absorption of trichlorinated congeners was 60–98%, probably due to the low levels at which they were present and ensuing analytical difficulties in detection. [Another possible explanation could be metabolism of the trichlorinated congener.]

The gastrointestinal absorption of 10 congeners from food was investigated using a mass balance approach in seven individuals aged 24–81 years with different contaminant body burdens (Schlummer *et al.*, 1998). The difference between ingested and excreted amounts of the chlorinated compounds was defined as net absorption. Nearly complete net absorption was observed for PCB-28, PCB-52, PCB-77, PCB-101, and PCB-126. Absorption of PCB-105, PCB-138, PCB-153, and PCB-180 was > 60% in most volunteers, but limited absorption was observed in the three older subjects. In all cases, absorption of PCB-202 was < 52%.

(ii) Experimental systems

Several reports have been published on the dietary absorption of PCBs, mostly individual congeners. Gastrointestinal absorption of congeners with between one and six chlorine atoms has been investigated by monitoring faecal excretion in rats fed individual congeners at doses ranging from 5 to 100 mg/kg bw. Absorption of the administered dose was > 90% for all 20 congeners tested (Albro & Fishbein, 1972). Metabolic studies in rodents given oral doses of various radiolabelled PCBs with three to six chlorine atoms (i.e. PCB-31, PCB-47, PCB-85, PCB-101, and PCB-153) indicated that gastrointestinal

absorption was highest for the trichlorobiphenyl congener (about 94% of the administered dose), and lowest for the hexachlorobiphenyl PCB-153 (28%) (Bergman *et al.*, 1982). In a study by Tanabe *et al.* (1981), absorption efficiency was 95% for dichlorobiphenyls, but only 75% for octachlorobiphenyls. These data suggested that, in rats, absorption of PCBs decreases as the number of chlorine atoms increases.

(b) Inhalation

(i) Humans

There is indirect evidence for absorption of PCBs via inhalation in humans; several congeners have been detected in body fluids of people exposed in occupational settings or frequenting contaminated buildings, such as schools, where air concentrations of PCBs have also been measured (Wolff, 1985; Wolff *et al.*, 1992; Schwenk *et al.*, 2002; Liebl *et al.*, 2004).

(ii) Experimental systems

Hu *et al.* (2010) used a nose-only exposure system to assess the time course of PCB vapour uptake from commercial products in animals. Rats (average weight, 188 g) were exposed to vapours of Aroclor 1242 (PCB concentration, 2.4 mg/m^3; total amount, 40 µg) for a total of 2 hours, with a 1-hour break, and killed at 0, 1, 3, 6, and 12 hours after exposure. Congeners detected in tissues included mostly PCBs with mono- or di-*ortho*-substitution, ranging from mono- to pentachlorobiphenyls, with the majority being tri- and tetrachlorobiphenyls. PCB-20 + PCB-28 co-elution was most abundant in every tissue. When compared with the air mixture, most of the material retained in the tissues had shifted from mono- and dichlorinated PCBs to tri- and tetra- or even more highly chlorinated biphenyls. The amount of PCBs measured in the five tissues collected (liver, lung, blood, adipose tissue, and brain) was 5 µg per rat. The measured body burden (i.e. the sum of PCBs loaded at the end of exposure) was 33 µg per rat, suggesting pulmonary absorption of close to 100%. Casey *et al.* (1999) found that uptake of PCBs was greater by inhalation than by ingestion in a comparison of rats exposed to Aroclor 1254 for 30 days via inhalation (0.9 µg/m^3) or in the diet (0.436 µg/g).

(c) Dermal exposure

(i) Humans

Studies on exposure of capacitor workers to PCBs suggested that these compounds are well absorbed by skin contact (Wolff, 1985). Skin samples collected from human cadavers and exposed in vitro to [^{14}C]-labelled Aroclor 1254 and Aroclor 1242 retained 43–44% of the administered dose over a 24-hour period when the mixtures were formulated in water (Wester *et al.*, 1990, 1993). A lower retention was observed when PCBs were formulated in mineral oil or adsorbed on contaminated soil.

(ii) Experimental systems

In rhesus monkeys, percutaneous absorption in vivo of [^{14}C]-labelled Aroclor 1242 and Aroclor 1254 formulated in mineral oil was 20.4 ± 8.5% and 20.8 ± 8.3% of the administered dose, respectively, as determined by urinary and faecal excretion of radiolabel for 30 days after topical application (Wester *et al.*, 1990).

In rats given selected mono-, di-, tetra- and hexachlorobiphenyls as a single dermal dose (0.4 mg/kg bw), dermal penetration varied inversely with the degree of chlorination Garner & Matthews (1998). At 48 hours, dermal penetration ranged from about 100% for the monochlorobiphenyl to about 30% for the hexachlorobiphenyl.

In rats given a topical dose of [^{14}C]-labelled PCB-77 or PCB-153, absorption at 24 hours after dosing ranged from 5% to 8% for both compounds (Hughes *et al.*, 1992). Skin retention was 3–31% for PCB-77 and 3–12% for PCB-153. Dermal absorption was similar for all application forms (solid, aqueous paste, aqueous suspension, dissolved in ethanol). For PCB-153, absorption

was significantly higher when PCB-153 was applied as a solid compared with in ethanol.

Male F344 rats were given single doses (0.4 mg/kg bw) of [^{14}C]-labelled mono-, di-, tetra- and hexachlorobiphenyls applied to 1 cm^2 areas of the dorsal skin (Garner *et al.*, 2006). The more highly chlorinated PCBs were slowly absorbed and accumulated in the adipose tissue and skin. Excretion of absorbed radiolabel varied with chlorine content, ranging from 27% to about 100% at 2 weeks after dosing (Garner *et al.*, 2006).

4.1.2 Distribution

The distribution of PCBs is dependent on the structure and the physicochemical characteristics of the individual congeners, and also on dose.

(a) Humans

No studies of quantitative distribution in humans after controlled exposure to PCBs were available to the Working Group. However, some information existed regarding the concentration of PCBs in human tissues and biological fluids after occupational or dietary exposure. PCBs distribute preferentially to adipose tissue and concentrate in human breast milk due to its high fat content. The pattern of congeners observed in tissues does not correspond with the profiles of commercial PCB mixtures.

The most commonly detected PCBs in plasma and in adipose tissue of occupationally exposed individuals are the hexa- and heptachlorobiphenyls. PCB congeners with chlorine atoms in the 4 and 4′ positions were generally found at relatively high concentrations, while PCBs with nonsubstituted 3,4-positions on at least one ring were present at lower concentrations (ATSDR, 2000).

In Greenlanders exposed through high consumption of fat from sea mammals, the most abundant PCB congeners found in adipose tissue, plasma, and liver were PCB-138, PCB-153, and PCB-180 (Dewailly *et al.*, 1999).

Some studies focused on transplacental transfer of PCBs, as determined by measurement of PCB concentrations and congener profiles in maternal blood, placenta and cord blood. Tsukimori *et al.* (2013) investigated concentrations of four non-*ortho* PCBs (PCB-77, PCB-81, PCB-126, PCB-169) in maternal blood, placenta, and cord blood in 19 pregnant women from Fukuoka City, Japan. Mean concentrations were 3.95, 0.87, and 1.08 pg toxic equivalency (TEQ)/g lipid in maternal blood, placenta, and cord blood, respectively. Among specific congeners, PCB-126 showed the highest ratio for cord blood to maternal blood (0.3). PCBs are able to cross the placental barrier in humans, with PCB concentration in cord blood being 25–50% of that in maternal blood.

A study of 360 second-grade schoolchildren (a subgroup of the cohort in Hesse, Germany) in 1995 (Karmaus *et al.*, 2001a, b) found a significant dose-dependent relationship between the duration of breastfeeding (0, 1–4 weeks, 5–8 weeks, 9–12 weeks, > 12 weeks) and blood concentrations of all organochlorine compounds, including PCBs. Breastfeeding for more than 12 weeks was associated with a doubling of concentrations of organochlorine compounds in the children's blood.

Scheele *et al.* (1992) measured the concentrations of PCB-138, PCB-153, and PCB-180 in 38 children with leukaemia and 15 children in a control group. The PCB concentrations in bone marrow were higher by two- to threefold than those in fat tissue; however, there was no significant difference between PCB concentrations in bone marrow of children with leukaemia and of children in the control group.

PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, and PCB-180 were analysed in six post-mortem samples of human lung (Rallis *et al.*, 2012). The limit of quantification (LOQ) varied from 1.7–4.5 ng/g tissue. PCB-153 (detected in two cases), PCB-138 and PCB-180 (detected in three cases) were found at highest concentrations, ranging from < LOQ to 6.3 ng/g.

In 107 post-mortem samples of human brain (Mitchell *et al.*, 2012), eight congeners (PCB-28, PCB-95, PCB-105, PCB-118, PCB-138, PCB-153, PCB-170, and PCB-180) were analysed. PCB-138, PCB-153, and PCB-180 were most frequently detected, at average concentrations of 5.5–8 ng/g lipid. PCB-95 was mainly detected in samples from individuals with neurodevelopmental disorders with a known genetic basis, compared with neurologically typical controls.

In addition to the parent PCBs, hydroxylated metabolites have been detected in human serum and adipose tissue (Fernandez *et al.*, 2008). The concentrations of hydroxylated PCBs (OH-PCBs; 14 congeners), methylsulfonyl PCBs ($MeSO_2$-PCBs; 24 congeners), and parent PCBs (17 congeners) in five paired samples of human liver and adipose tissue were reported by Guvenius *et al.* (2002). The sum of OH-PCB congeners was higher in liver (7–175 ng/g lipid) than in adipose tissue (0.3–9 ng/g lipid), with 3′-OH-PCB-138 and 4′-OH-PCB-130 as the predominant OH-PCB metabolites. The sum of $MeSO_2$-PCBs was of the same order of magnitude as OH-PCB congeners in the same samples: 12–358 ng/g lipid and 2–9 ng/g lipid in liver and adipose tissue, respectively. The concentrations of parent PCBs were similar in liver and adipose tissue, at 459–2085 ng/g lipid and 561–2343 ng/g lipid, respectively.

Concentrations and congener profiles of PCBs and OH-PCBs in placenta samples from a population in Madrid, Spain, were reported by Gómara *et al.* (2012). The sum of PCB concentrations in placenta samples ranged from 943–4331 pg/g fresh weight, and their hydroxylated metabolites showed a 20-times lower concentration (53–261 pg/g fresh weight). PCB-52 and PCB-101 accounted for more than 44% of the total amount of PCBs. The OH-PCB profiles were dominated by 4-OH-PCB-187 and 4-OH-PCB-146, representing > 50% of the sum concentration of OH-PCBs in the placenta samples.

The concentration of OH-PCBs may comprise 10–20% of total PCBs in human serum, and as many as 38 different OH-PCBs were structurally identified in human plasma, pooled from 10 randomly selected male donors. Only a few of these make up the major proportion of the OH-PCBs present in human blood (Hovander *et al.*, 2002).

$MeSO_2$ metabolites of PCBs were investigated in serum samples from pregnant women from Slovakia and in a selected number of paired samples of cord blood (Linderholm *et al.*, 2007). The major methylsulfone in most samples was a non-identified $MeSO_2$-hexachlorinated biphenyl, followed by 4′-$MeSO_2$-PCB-101, 4′-$MeSO_2$-PCB-87, and 4-$MeSO_2$-PCB-149. The concentrations of $MeSO_2$-PCBs in maternal serum were about 1.5 times higher than in the corresponding cord serum on a lipid-weight basis. In samples of human adipose tissue, 4-$MeSO_2$-PCB-49 [4-$MeSO_2$-2,2′,4′,5-tetraCB; 4′-$MeSO_2$-PCB-49], 4-$MeSO_2$-PCB-101 [4′-$MeSO_2$-PCB-101; 4-$MeSO_2$-2,2′,4′,5,5′-pentaCB], and 3-$MeSO_2$-PCB-110 [5-$MeSO_2$-PCB-110; 3-$MeSO_2$-2,3′,4′,5,6- pentaCB] were the predominant $MeSO_2$ metabolites (Karásek *et al.*, 2007).

(b) Experimental systems

(i) PCB mixtures

Adult rhesus monkeys were given Aroclor 1248 as a single dose at 1.5 or 3.0 g per kg bw by gastric intubation, and killed after 4 days (Allen *et al.*, 1974). At the lowest dose tested, average concentrations found in liver, kidney, and brain were 25, 12, and 17 µg/g tissue, respectively. In another study, two groups of eight adult rhesus monkeys were exposed to diets containing Aroclor 1248 at 2.5 ppm (Allen & Barsotti, 1976). After 6 months of exposure, the monkeys were successfully bred. After 2 months, milk samples after birth were obtained from four lactating mothers exposed at 2.5 ppm. Concentrations of PCBs ranged from 0.154 to 0.397 µg per g milk in

three samples of milk fat, and reached 16.44 µg per g in milk fat in the fourth sample.

PCBs were analysed in blood, adipose tissue, liver, kidney and brain from female rhesus monkeys fed Aroclor 1254 at a daily dose of 0, 5, 20, 40, or 80 µg/kg bw for approximately 6 years (16 animals per group) (Mes *et al.*, 1995a). Offspring were nursed for 22 weeks and fed no additional PCBs until necropsy at approximately 120 weeks after birth. PCB concentrations in all tissues of the adult monkeys (mothers and offspring) increased with increasing dose. Mes *et al.* (1994) reported that for groups exposed to higher doses (≥ 40 µg/kg bw), tissues of infants from dosed dams contained higher concentrations of PCBs than tissues of infants from control dams. The PCB distribution pattern in tissues from a dosed mother/infant pair differed considerably. A larger percentage of heptachlorobiphenyls was found in the infants than in their dams.

In rats given a single dose of Aroclor 1254 at 500 mg/kg bw by gavage, the highest PCB concentrations were found in adipose tissue (996 µg/g wet weight), liver (116 µg/g wet weight), and brain (40 µg/g wet weight), indicating that PCBs are able to cross the blood–brain barrier (Grant *et al.*, 1971). The relative amounts of PCBs in the brain, liver, spleen, blood, testes, heart, kidney, and adipose tissue of rats killed 3 weeks after treatment were 10%, 16%, 20%, 21%, 22%, 24%, 36%, and 67%, respectively, of those found in animals killed after 2 days. In a subsequent long-term study, Grant *et al.* (1974) fed rats with Aroclor 1254 at a dietary concentration of 0, 2, 20, or 100 mg/kg feed and found highest concentrations of PCBs after 246 days in adipose tissue, with concentrations reaching 26.1 ± 2.9 µg/g wet tissue at the lowest contamination tested (2 mg/kg feed). Levels of PCBs in all tissues analysed were dose-related, and generally, the tissue concentrations did not increase significantly after 64 days of exposure. The residues present in the adipose tissue, liver, and brain had decreased by 64%, 75%, and 10% respectively, 182 days after removal of Aroclor 1254 at 2 mg/kg from the diet. Part of the decrease observed in the adipose tissue and the liver resulted from a dilution effect due to weight increase in these tissues.

The analysis of individual congeners in tissues of rats fed diets containing Aroclor 1254 for 84 days demonstrated a limited accumulation of PCB congeners with a low level of chlorine substitution (tri- and tetrachlorobiphenyls) (Nims *et al.*, 1994). In these rats, time- and dose-dependent increases in the relative concentrations of PCB-138 and PCB-153 were detected in the liver and adipose tissue. Increases in PCB-99 concentrations in hepatic and adipose tissues, and in PCB-156 in adipose tissue, were also observed.

Aroclor 1254 was given to pregnant rats once daily on days 7–15 of gestation (Curley *et al.*, 1973). The concentrations of PCBs found in fetuses were higher by twofold in the group at 50 mg/kg bw compared with the group at 10 mg/kg bw. The mean concentrations of PCB-derived components found in brain, liver, and kidney in weanlings aged 21 days (27 days after the last dose was given to the mother in the group at 10 mg/kg bw) were approximately 2, 4, and 2 µg/g wet tissue, respectively. Concentrations in milk sampled from the same group were between 16 and 25 µg/g.

Samples of brain, adipose tissue, and liver from rat pups and dams exposed to Aroclor 1254 were analysed by Shain *et al.* (1986). In adipose tissue, most congeners were detected at concentrations close to the feed concentration, but the following congeners accumulated to tissue concentrations 10-fold those in the feed: PCB-176, PCB-146, PCB-138 + PCB-168 + PCB 178 (co-eluted), and PCB-177. In the liver and the brain, the congeners present at the highest concentrations were PCB-85 and PCB-179 + PCB-188 (co-eluted). Bioaccumulation of congeners in the milk closely resembled that observed in fat samples from the dams. The chromatographic pattern

of bioaccumulated congeners in pup liver was different from that observed in the dams. The congener found at the highest concentration in samples of newborn rat brain was PCB-85. Shain *et al.* (1986) estimated that the transfer of PCBs through the mammary gland and milk in rats may be 100 times higher than the transfer across the placenta, resulting in a higher accumulation during lactation than during pregnancy.

Kodavanti *et al.* (1998) investigated the congener-specific distribution of PCBs in blood, brain, liver, and adipose tissue of adult rats given repeated doses of Aroclor 1254 (30 mg/kg bw per day; once per day, 5 days per week for 4 weeks). Total PCB congeners in control rat brain were < 0.02 µg/g tissue. Mean concentrations of total PCBs in treated rats in the frontal cortex, cerebellum, and striatum were 15.1, 13.1, and 8.2 µg/g tissue, respectively; those in the blood, liver, and adipose tissue were 1.6, 38.3, and 552 µg/g tissue, respectively. In addition to differential total uptake between tissues, there was differential accumulation of PCBs with respect to number of chlorine substituents. In all tissues, heavily (hexa- to nona-) chlorinated congeners were present in higher proportions than in the parent mixture, Aroclor 1254, while less highly (tetra- and penta-) chlorinated congeners were present to a lesser degree than their respective proportions in Aroclor 1254. This shift towards accumulation of heavily chlorinated congeners appeared to be more pronounced in the brain than in liver and fat.

In rats exposed via inhalation to vapour-phase PCBs generated from Aroclor 1242 for 10 days, much higher amounts of PCBs (× 400) were found in liver and lung than in blood (Hu *et al.*, 2010). PCB-20 + PCB-28 (co-eluted), PCB-49 + PCB-69 (co-eluted), PCB-52, PCB-60, PCB-61 + PCB-70 + PCB-74 + PCB-76 (co-eluted), PCB-66, PCB-83 + PCB-99 + PCB-112 (co-eluted), PCB-85 + PCB-116 + PCB-117 (co-eluted), PCB-90 + PCB-101 + PCB-113 (co-eluted), PCB-105, and PCB-118 were the major congeners in these tissues.

The presence of $MeSO_2$-PCB atropisomers was determined in liver, lung, and adipose tissues of rats orally exposed to Clophen A50. In all tissues analysed, especially lung, *para*-$MeSO_2$ PCBs were more abundant than *meta*-derivatives. An excess of the atropism 2(A_2) of 4-$MeSO_2$-PCB-149 – (*R*)-3-$MeSO_2$-PCB-149 – in lung extracts was observed (Larsson *et al.*, 2002). The enantiomeric enrichment of PCB atropoisomers was reported in selected tissues from rats exposed to Aroclor 1254 (Kania-Korwel *et al.*, 2006). Both PCB-95 and PCB-149 were enantiomerically enriched to a significant extent in adipose tissue, liver, and skin.

A few studies on complex mixtures such as Aroclor 1254 mention substantial retention of certain congeners in lung of treated mice (Anderson *et al.*, 1993).

In mice exposed to contaminated soil (retrieved from a Superfund site before remediation) through their bedding for 4 weeks, total PCB residues in skin and fat declined about 80% during the 4-week recovery period. PCB residues were detected in the ear skin (total PCBs, 208 mg/kg of tissue), trunk skin (total PCBs, 129 mg/kg of tissue), and in body fat (total PCBs, 370 mg/kg), confirming these tissues as important PCB reservoirs (Imsilp & Hansen, 2005).

(ii) Individual congeners

Several experiments carried out in mammals, including non-human primates, confirm the data obtained with complex mixtures. The congeners investigated were unlabelled or labelled PCB-3, PCB-5, PCB-15, PCB-30, PCB-31, PCB-47, PCB-65, PCB-77, PCB-101, PCB-116, PCB-118, PCB-126, PCB-153, and PCB-196 (Goto *et al.*, 1974a, b; Matthews & Anderson, 1975a, b; Abdel-Hamid *et al.*, 1981; Beran *et al.*, 1983; Shimada & Sawabe, 1984; Koga *et al.*, 1990; van Birgelen *et al.*, 1996; Pereg *et al.*, 2001; NTP, 2006a, b, 2010). In some cases, mixtures of individual congeners were used (Öberg *et al.*, 2002; NTP, 2006c, d). Taken together, the data indicated that an oral

dose of PCBs results in an initially high concentration in liver and serum, followed by a decrease in concentrations in the liver, and a concomitant increase in adipose tissue and lipid-rich tissues. This redistribution generally occurred during the first week after dosing, and the differences between the congeners were mainly dependent on the number of chlorine atoms (ATSDR, 2000). In rodents, the hepatic retention/accumulation of non-*ortho*-substituted PCBs such as PCB-126 may occur to a higher extent than in adipose tissue, including after long-term exposure (NTP, 2006a). This was not the case for congeners with chlorine atoms in *ortho* positions, such as PCB-153 (van Birgelen *et al.*, 1996; NTP, 2006a, b).

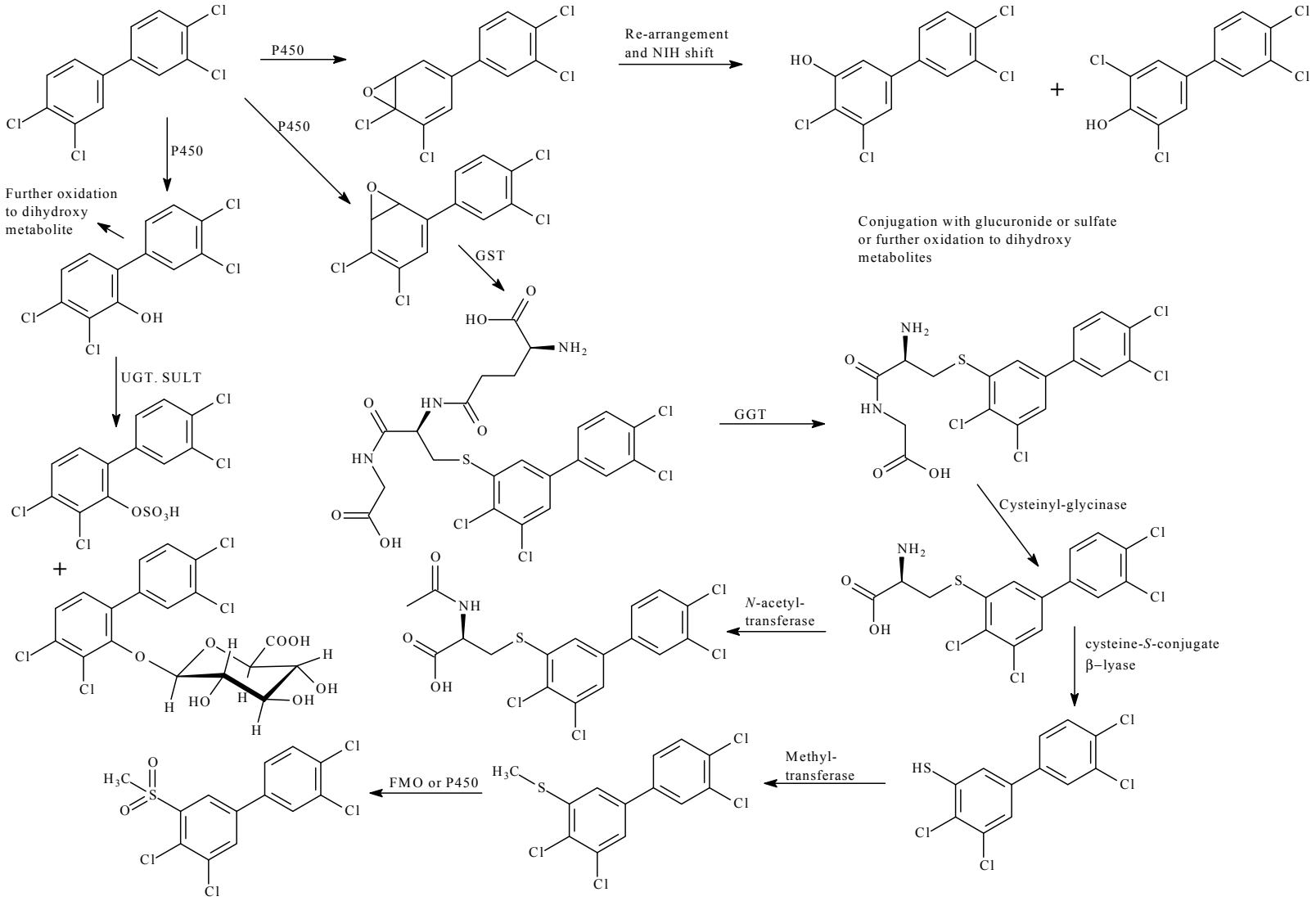
4.1.3 Metabolism

There is evidence that most known PCBs are subject to biotransformation (metabolism) in humans and other animals through enzymatic processes (Safe, 1993). Biotransformation is important for the eventual elimination of PCBs from the body, as most (but not all) of the metabolites are more water-soluble than the parent compound. As well as serving as substrates for biotransformation enzymes, some PCBs and PCB metabolites can interact with several drug-metabolizing enzymes as inducers or inhibitors, as discussed further below.

The first step in metabolism targets the biphenyl ring carbons, and is catalysed by cytochrome P450 (CYP) monooxygenase enzymes. Subsequent metabolism involves one or more of several other possible enzymatic pathways (James, 2001). Some of the major pathways of PCB metabolism are illustrated in Fig. 4.1, with PCB-77 as an example. Fig 4.2 shows structures of representative PCB metabolites. The rate and extent of biotransformation of a particular PCB congener depend upon its chlorination pattern, the number of chlorine substituents, the species, age, and sometimes sex of the animal, and in some cases whether or not the exposure is continuous or a single exposure. The number of chlorine substituents and substitution pattern determine how well a particular PCB congener binds to and can be metabolized by the biotransformation enzyme (Matthews & Dedrick, 1984). In general, congeners with more than four chlorine substituents are more slowly metabolized than those with four or fewer chlorines, and congeners with unsubstituted 3,4-positions in one or both rings are more readily metabolized than those without such substitution patterns (Hansen, 2001). Biotransformation enzymes with similar functions often differ between animal species in properties of substrate recognition and binding, which contributes to species differences in metabolism. Very young animals often have lower levels of several biotransformation enzymes than adults, resulting in age-related differences in metabolism (Hines, 2008). In rodents, sex affects the expression of several important biotransformation enzymes, particularly CYP, which can lead to sex-specific differences in PCB metabolism. The reason that continuous exposure to certain PCB congeners can affect rate and extent of metabolism is that such exposure can result in upregulation of expression of enzymes that biotransform PCBs, through receptor-mediated processes. PCBs that bind the aryl hydrocarbon receptor (AhR) (see Section 4.3.1) are known to induce CYP isoforms in the 1 family (CYP1A1, CYP1A2 and CYP1B1) as well as epoxide hydrolase, some isoforms of uridine diphosphate-glucuronosyltransferase (UGT) and glutathione *S*-transferase (GST) (Parkinson *et al.*, 1980; 1983). PCBs that bind the nuclears receptors, the pregnane-X receptor (PXR) and the constitutive androstane receptor (CAR) have been shown to induce CYP3A4 and CYP2B isoforms (Petersen *et al.*, 2007; Al-Salman & Plant, 2012).

In the context of carcinogenesis, biotransformation to electrophilic metabolites that are more chemically reactive than the parent PCB is likely to be an important component. Being more biotransformed, the metabolized congeners are more likely to undergo bioactivation.

Fig. 4.1 Metabolic pathways for polychlorinated biphenyls, showing PCB-77 (3,3′,4,4′-tetrachlorobiphenyl) as an example

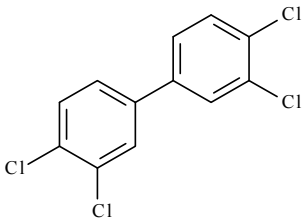


P450, cytochrome P450; UGT, UDP-glucuronosyltransferase; SULT, 3′-phosphoadenosine-5′-phosphosulfate-sulfotransferase; GST, glutathione-*S*-transferase; GGT, gamma-glutamyl transpeptidase; FMO, flavin monooxygenase

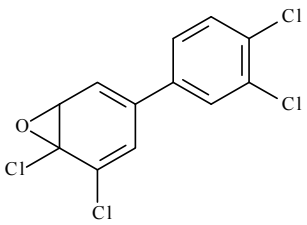
The NIH shift causes non-enzymatic migration of chlorine atoms to an adjacent carbon.

Compiled by the Working Group

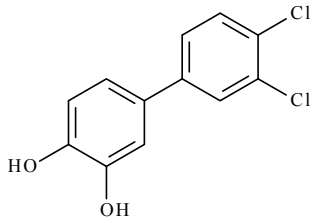
Fig. 4.2 Representative metabolites derived from PCB-77 (3,3′,4,4′-tetrachlorobiphenyl)



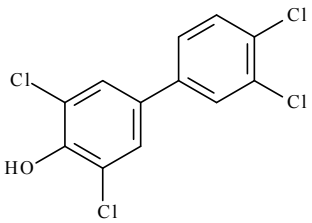
3,3',4,4'-tetrachlorobiphenyl (parent)



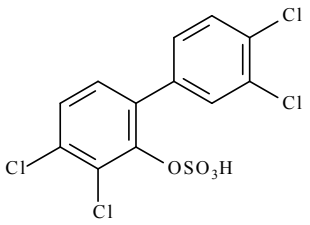
4,5-arene oxide of 3,3',4,4'-tetrachlorobiphenyl



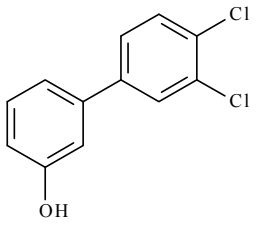
3',4'-dihydroxy-3,4-dichlorobiphenyl
(catechol metabolite)



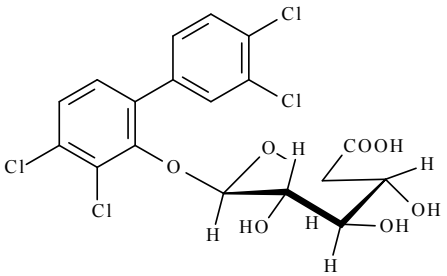
4'-hydroxy-,3,3',4,5'-tetrachlorobiphenyl



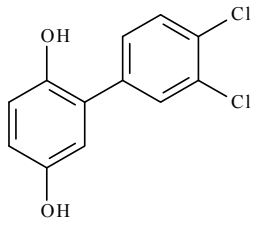
2'-hydroxy-3,3',4,4'-tetrachlorobiphenyl-2'-sulfate



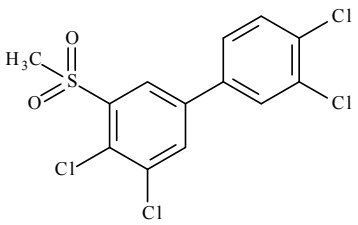
3'-hydroxy-3,4-dichlorobiphenyl



2'-hydroxy-3,3',4 ,4'-tetrachlorobiphenyl-2'-glucuronide



2',5'-dihydroxy-3,4-dichlorobiphenyl
(semiquinone metabolite)



5-methyl-sulfonyl-3,3',4,4'-tetrachlorobiphenyl
(methyl sulfone metabolite)

The following sections describe the different enzymatic pathways known to be involved in PCB metabolism.

(a) CYP

The first step in biotransformation of PCBs is introduction of oxygen, catalysed by one or more members of the CYP superfamily of monooxygenase enzymes (Guengerich, 2008). Two mechanisms are known, H• radical abstraction and recombination of the short-lived chlorobiphenyl radical with an OH• radical from the active site of CYP to give a hydroxylated (phenolic) metabolite, and formation of an arene oxide by addition of oxygen across an aromatic bond in the biphenyl ring. The arene oxide is an electrophilic metabolite that can rearrange non-enzymatically to form a phenolic metabolite. If one of the carbons that forms part of the arene oxide is substituted with chlorine then, during the non-enzymatic rearrangement, that chlorine can migrate to the adjacent non-chlorine-substituted carbon, while the phenolic hydroxy group attaches to the carbon previously substituted with chlorine, a mechanism known as the NIH shift (shown in Fig. 4.1). Alternatively, the arene oxide may undergo further metabolism by epoxide hydrolase or GST, or may bind with a nucleophilic site on DNA, such as the N7 of guanine, to form an adduct.

As noted above, the chlorine substitution pattern, number of chlorine substituents and presence or absence of unsubstituted 3,4-positions are important factors in determining how readily a particular congener is metabolized by CYP. There are more than 50 isoforms of CYP in humans, and a similar number in experimental animals (Guengerich, 2008). Studies to date have shown that several human isoforms can biotransform one or more PCB congeners; these include CYP1A1, CYP1A2, CYP2A6, CYP2B6, CYP2C8, CY2C9, CYP2C19, and CYP3A4 (Ariyoshi *et al.*, 1995; McGraw & Waller 2006, 2009; Warner *et al.*, 2009; Yamazaki *et al.*, 2011). Related isoforms usually metabolize the same congeners in rat (Morse *et al.*, 1995; Warner *et al.*, 2009; Wu *et al.*, 2011), mouse (Curran *et al.*, 2011), and other species such as fish (Schlezinger *et al.*, 2000). Studies have suggested that congeners with one or no *ortho*-chlorine substitutent are more likely to be metabolized by CYP1 family isoforms. Although it has not been explicitly demonstrated, CYP1B1 metabolizes many of the same substrates as CYP1A1 and CYP1A2 (Shimada *et al.*, 1997) and may also metabolize congeners with one or no *ortho* chlorine. However, CYP1B1 protein is not constitutively expressed in liver, the major drug-metabolizing tissue, and is generally very low in normal tissues (Murray *et al.*, 2001). Congeners with two or more *ortho*-chlorine substituents are usually metabolized by CYP2A, CYP2B, CYP2C and CYP3A subfamily isoforms. It is not well understood which isoforms are involved in monooxygenation of each known PCB congener. This is partly because of difficulties in studying monooxygenation of some of the congeners in vitro with hepatic microsomes or expressed recombinant individual CYP isoforms. The less chlorinated congeners, which tend to be readily metabolized by CYP, are easily studied in vitro; however, until recently they attracted much less attention than the more highly chlorinated congeners (Espandiari *et al.*, 2004). The difficulty in studying highly chlorinated congeners is that they are very slowly metabolized, and conditions for incubation in vitro are difficult to set up to produce sufficient hydroxylated metabolite for analysis. With increasingly sensitive analytical techniques, this problem can be overcome (Yamazaki *et al.*, 2011). While some early publications claimed that certain congeners did not produce metabolites in particular species (Murk *et al.*, 1994), these congeners were later shown through studies in vivo to produce hydroxylated metabolites (Buckman *et al.*, 2007).

An important determinant of the activity of CYP is whether or not the isoform that metabolizes a particular congener is subject to induction, either through exposure to PCBs or through

exposure to other agents known to induce that form of CYP. For example, many congeners with no or one *ortho* chlorine are metabolized by CYP1A1 or CYP1A2 (Curran *et al.*, 2011), and these congeners, like dioxin, polycyclic aromatic hydrocarbons and some components of tobacco smoke, induce CYP1A1 and CYP1A2 by binding to and activating AhR (Parkinson *et al.*, 1983; Safe, 1993). CYP1B1 is also induced by compounds that bind and activate AhR (Murray *et al.*, 2001). A study in which wildtype and knockout mouse strains were exposed in utero and by lactation to a complex mixture of PCBs showed that mice with poor-affinity AhR and lacking CYP1A2 (*Cyp1a2*$^{(-/-)}$ knockout) had higher concentrations of congeners with no or one *ortho* chlorine in tissues than mice with high-affinity AhR and CYP1A2 (*Cyp1a2*$^{(+/+)}$ wild-type), consistent with low metabolism of these PCB congeners in the knockout mice (Curran *et al.*, 2011). PCBs with two or more *ortho* chlorines and at least one *para* chlorine interact with rat and human CAR and induce CYP2B family isoforms, including CYP2B1 and CYP2B6 in a similar manner to the classic inducer, phenobarbital (Parkinson *et al.*, 1980; Al-Salman & Plant, 2012). Some PCBs with two or more *ortho* chlorines have been shown to bind to the human and rat PXR and to human CAR, resulting in upregulation of CYP3A4 (Waller *et al.*, 1996; Petersen *et al.*, 2007; Al-Salman & Plant 2012). CYP3A4 converts PCB-101 and PCB-118 to hydroxylated metabolites (McGraw & Waller, 2009). Activation of CAR also results in upregulation of CYP2B isoforms, several of which have been shown to metabolize PCBs with two or more *ortho* chlorines. For example, human CYP2B6 and the related enzyme, canine CYP2B11, were shown to convert PCB-153 to the 3-hydroxylated metabolite, albeit very slowly (Ariyoshi *et al.*, 1995).

An interesting subgroup of PCBs comprises the 19 chiral PCB congeners, all of which have three or more *ortho* chlorines, which limit rotation around the biphenyl bond. There was evidence that these compounds are enantioselectively metabolized, resulting in depletion of one enantiomer through metabolism, while the form that is resistant to metabolism accumulates (Kania-Korwel *et al.*, 2008; Lehmler *et al.*, 2010). Forms of CYP identified as metabolizing chiral PCBs are rat CYP2B1 and human CYP2B6. For example, there was evidence that PCB-45, PCB-84, PCB-91, PCB-95, PCB-132 and PCB-136 were enantioselectively metabolized to hydroxylated metabolites in vitro by purified rat CYP2B1 and human CYP2B6, leading to alterations in the enantiomeric fractions of the parent congeners (Warner *et al.*, 2009). The positions of hydroxylation were not identified. In a separate study, rat liver microsomes preferentially metabolized (+)-2,2′,3,3′,6,6′-hexachlorobiphenyl (PCB-136) to 5-hydroxy-PCB-136 (5-OH-PCB-136), and treatment of rats with phenobarbital, which induces CYP2B1, further increased the formation of 5-OH-PCB-136 from (+)-PCB-136, compared with untreated rats, thereby leaving an excess of the less readily metabolized (–)-PCB-136 (enantiomeric enrichment) (Wu *et al.*, 2011). There was also a slight increase in 5-OH-PCB-136 formation in dexamethasone-treated rats, which have induced CYP3A, compared with controls. The minor metabolites, 4-OH-PCB-136 and 4,5-dihydroxy-PCB-136 were also formed preferentially by microsomes from phenobarbital-treated rats compared with controls. Since the ryanodine receptor is sensitized only by (–)-PCB-136, more rapid metabolism of (+)-PCB-136 means that the more toxic enantiomer is preferentially retained in the body.

Once formed, OH-PCBs are sometimes further hydroxylated by CYP and perhaps other oxygenases to dihydroxy-PCBs (McLean *et al.*, 1996a; Garner *et al.*, 1999; Wu *et al.*, 2011). If the two OH groups are *ortho* to each other, the metabolites are termed catechols (Garner *et al.*, 1999), and if the two OH groups are *para* to each other, the metabolites are termed hydroquinones (or semiquinones) (Fig. 4.2).

(b) Other oxidative enzymes

PCB catechols and hydroquinones can undergo oxidation to PCB quinones, which are electrophilic, potentially reactive, metabolites. One pathway for quinone formation is through the action of prostaglandin endoperoxide H synthase, an enzyme expressed in extrahepatic tissues, including prostate, ovary, and breast (Wangpradit *et al.*, 2009). Hydroquinones can also be converted to quinones by peroxidases such as horseradish peroxidase, myeloperoxidase, and lactoperoxidase (Srinivasan *et al.*, 2002).

(c) Epoxide hydrolase

If not quickly rearranged to form a phenolic metabolite, an arene oxide metabolite can be converted to a dihydrodiol by addition of water, in a reaction catalysed by epoxide hydrolase (Ota & Hammock, 1980). The dihydrodiol metabolite is generally non-toxic and readily eliminated as the dihydrodiol or as a glucuronide conjugate. It has been suggested that dihydrodiol metabolites of PCBs may be oxidized by dihydrodiol dehydrogenase to the corresponding catechol metabolite, thereby restoring aromaticity to the ring (Garner *et al.*, 1999). Furthermore, catechols could be converted to the *ortho* quinone, which is chemically reactive and can bind to protein and DNA (Zhao *et al.*, 2004).

(d) PCB oxygenation and formation of ROS

PCB biotransformation by CYP can sometimes give rise to formation of reactive oxygen species (ROS) of PCBs, through uncoupling of the CYP cycle. Formation of ROS during PCB monooxygenation by CYP most likely occurs if the congener binds to the CYP substrate-binding site in an orientation that is not favourable for rapid monooxygenation: this has been demonstrated for PCB-77 biotransformation by CYP1A from fish and other vertebrates (Schlezinger *et al.*, 1999, 2000). PCB-77 has been shown to inhibit ethoxyresorufin *O*-deethylase (EROD) activity at high concentrations, perhaps by competitive inhibition of CYP1A by PCB-77 (Hahn *et al.*, 1993). Formation of ROS through uncoupling of the CYP1A cycle has been demonstrated with two other non-*ortho*-substituted PCB congeners, PCB-126 and PCB-169 (Schlezinger *et al.*, 2006). PCB-126 and PCB-169 were also shown to uncouple human CYP1B1 and produce ROS (Green *et al.*, 2008). Since CYP1B1 is expressed and inducible in tissues that are frequent targets for cancer, including colon, breast, lung, endometrium, ovary, and prostate, formation of ROS in these tissues could result in genotoxicity.

PCB metabolism by peroxidases and prostaglandin H synthase (also called cyclooxygenase; COX) can also give rise to ROS (Gonçalves *et al.*, 2009). Another pathway leading to ROS production during PCB metabolism occurs when quinone metabolites are formed. Quinones undergo redox cycling through reaction with glutathione (GSH) to form adducts through Michael addition. The quinone-GSH-adduct can be converted back to the semi-quinone or catechol and recycled through this pathway (Amaro *et al.* 1996; Oakley *et al.*, 1996a). This cycling results in depletion of the important cellular antioxidant, GSH, which can cause oxidative stress to the cell and formation of ROS. Redox cycling of the 2′,5′-dihydroxy metabolite of PCB-12 has been shown to result in DNA adducts through formation of ROS (Oakley *et al.*, 1996a).

(e) GST

Arene oxide metabolites of PCBs are potential substrates for GSTs, as shown in Fig. 4.1. After initial formation of a conjugate with GSH, the two terminal amino acids of the tripeptide are enzymatically removed, leaving a cysteine conjugate of the PCB. This metabolite may be converted to a mercapturic acid, which is readily excreted in urine or bile (Bakke *et al.*, 1982). Alternatively, the cysteine conjugate may be a substrate for cysteine conjugate β-lyase, which converts the cysteine conjugate to a thiol. The

thiol metabolite of the PCB can then be methylated by methyltransferase and oxidized by flavin monooxygenase or CYP to yield the $MeSO_2$-PCB (Mio & Sumino 1985). Depending on its structure, the $MeSO_2$-PCB metabolite may not be readily excreted and may accumulate in tissues, particularly liver, lung, and adipose tissue (Haraguchi *et al.*, 1997a, b; Guvenius *et al.*, 2002; Hovander *et al.*, 2006; Karásek *et al.*, 2007). Chiral PCBs were shown, by analysis of the $MeSO_2$-PCBs present in human adipose tissue, seal blubber and pelican muscle, to form $MeSO_2$-PCBs in an enantioselective manner (Karásek *et al.*, 2007). Tissue accumulation can occur in fatty tissues because the $MeSO_2$-PCBs are very lipid soluble, especially those that are highly chlorinated. Accumulation in lung appears to be due to specific binding of the $MeSO_2$-PCBs to an uteroglobin-like protein/PCB-binding protein, a protein that is synthesized in non-ciliated bronchiolar Clara cells of the lung epithelium (Nordlund-Möller *et al.*, 1990; Anderson *et al.*, 1993). Formation of $MeSO_2$-PCBs and their retention in tissues are of concern because several of these metabolites have been shown to interact with the glucocorticoid receptor (Johansson *et al.*, 1998), and to be antiestrogenic (Letcher *et al.*, 2002). Some $MeSO_2$-PCBs such as 3-$MeSO_2$-2,2′,4′,5-tetrachlorobiphenyl [3′-$MeSO_2$-PCB-49] and 3-$MeSO_2$-2,2′,4′,5,5′-pentachlorobiphenyl [3′-$MeSO_2$-PCB-101] were potent inducers of CYP2B1 and CYP2B2 in rats (Kato *et al.*, 1997).

(f) Glucuronosyltransferase and sulfotransferase

OH-PCBs may be expected to be conjugated with glucuronic acid or sulfate to form non-toxic, readily excreted metabolites, in reactions catalysed by uridine 5′-diphosphate-(UDP)-glucuronosyl transferase (UGT) or 3′-phosphoadenosine-5′-phosphosulfate (PAPS)-sulfotransferase (SULT). Glucuronide and sulfate conjugates of a hydroxylated metabolite of PCB-3 were identified in urine of rabbits given 1 g by gavage (Block & Cornish, 1959).

Studies of glucuronidation have not been conducted with human liver microsomes or UGTs; however, two studies demonstrated formation of glucuronide conjugates with several OH-PCB metabolites, using rat liver microsomes and expressed rat UGTs in yeast strain AH22 (Tampal *et al.*, 2002; Daidoji *et al.*, 2005). In a further study of OH-PCB glucuronidation, it was noted that rates of conjugation varied with the particular OH-PCB substitution pattern, in catfish as well as in rats (Sacco *et al.*, 2008). Those OH-PCBs with only one chlorine flanking a 4-OH group exhibited a higher V_{max} for glucuronidation than OH-PCBs with a 4-OH-3,5-dichloro substitution pattern. The more slowly glucuronidated 4-OH-3,5-dichloro-substituted OH-PCBs were shown to be more potent inhibitors of human estrogen sulfotransferase (human SULT1E1) than those lacking two flanking chlorine atoms (Kester *et al.*, 2000).

In addition to a study in rabbits, which were shown to excrete glucuronide and sulfate conjugates of OH-PCB-3 (Block & Cornish, 1959), further evidence that OH-PCBs are sulfonated in vivo was provided by a study of the fate of PCB-3 in male rats given a dose of 112 mg/kg bw by intraperitoneal injection (Dhakal *et al.*, 2012). The major metabolite was the 4′-sulfate of PCB-3, with little evidence for the glucuronide conjugate; 4′-OH-PCB-3 was converted to the 4′-sulfate conjugate by rat SULT1A1 (Liu *et al.*, 2009). The 4′-OH-PCB-3 was also a substrate for human hepatic cytosolic SULT1A1 (Wang *et al.*, 2006). Other OH-PCBs tested were very poor substrates for human SULT1A1 (Wang *et al.*, 2006), human SULT2A1 (Liu *et al.*, 2006; Ekuase *et al.*, 2011), rat liver SULT1A1, or rat liver SULT2A3 (Liu *et al.*, 2009).

As noted above, OH-PCBs with a 4-OH-3,5-dichloro- structural motif are potent inhibitors of human SULT1E1, with 17-β-estradiol as substrate (Kester *et al.*, 2000). Recent studies showed that some OH-PCBs inhibit sulfonation of dehydroepiandrosterone (DHEA), catalysed by human SULT2A1 or rat SULT2A3 (Liu

et al., 2006, 2009; Ekuase et al., 2011). As well as inhibiting sulfonation of estradiol and DHEA, OH-PCBs inhibit sulfonation and glucuronidation of xenobiotic substrates. Several OH-PCBs were potent inhibitors (low μM values for IC_{50}, the concentration producing 50% inhibition) of sulfonation of 3-hydroxy-benzo[*a*]pyrene catalysed by human liver cytosol, human SULT1A1 and human SULT1E1, but were very weak inhibitors or did not inhibit SULT1A3 (Wang et al., 2005).

(g) Sites of metabolism

For most xenobiotics, including PCBs, the liver is the major organ of metabolism, as most of the drug-metabolizing enzymes are expressed in liver in high concentrations. This is true for several isoforms of CYP, epoxide hydrolase, GST, glucuronosyltransferase, and sulfotransferase; however, the liver is not the only site where these enzymes are expressed. The intestine expresses many of the same enzymes as the liver. The liver is able to convert PCBs to reactive metabolites, and to respond to PCBs that interact with AhR, but the role of metabolism in other tissues is not always clear. Tissues where there are associations between PCB exposure and cancer include the liver, lung, oral mucosa, uterus, thyroid, pancreas, adrenal, breast, skin, blood and lymphatic system, and these effects in some instances may be due to tissue distribution of PCBs or metabolite. As noted above, CYP1B1 is inducible by AhR agonists and has been shown to be expressed in colon, breast, lung, endometrium, ovary and prostate (Schmidt & Bradfield, 1996; Green et al., 2008). Prostaglandin endoperoxide H synthase, implicated in formation of quinone metabolites from OH-PCBs, is expressed in high concentrations in the prostate gland, and is also found in ovary and breast (Wangpradit et al., 2009). The skin contains inducible CYP1A, as well as other drug-metabolizing enzymes (Costa et al., 2010).

4.1.4 Excretion

(a) Humans

Two well designed studies (taking into account ongoing exposure and body weight changes, and not limited by small sample size or short sampling interval) showed that highly chlorinated congeners persist in the body, with half-lives averaging about 8–15 years, while less chlorinated PCBs clearly have shorter half-lives (Table 4.1; Grandjean et al., 2008; Ritter et al., 2011).

Few studies on the faecal (Schlummer et al., 1998; Moser & McLachlan, 2001), or urinary excretion (Price et al., 1972; ATSDR, 2000) of PCBs in humans have been published. A substantial part of absorbed or retained PCBs may be eliminated via breast milk (see Section 1.4 in this *Monograph*). Concentrations varying from 9 to 1915 ng/g lipid have been reported in the general population. Not only parent compounds, but also OH-PCBs were detected in breast milk. Traces of OH-PCBs (median of the sum of 12 congeners, 3 pg/g milk) were found in milk samples collected in 2000–2001 from 15 mothers living in Stockholm; the ratio of total PCBs to total OH-PCBs was approximately 1400, and the major metabolite was an unresolved mixture of 4-OH-CB-107 [4-OH-2,3,3′,4′,5-pentaCB; 4-OH-PCB-109] and 4′-OH-CB-108 [4′-OH-2,3,3′,4,5′-pentaCB; 4-OH-PCB-107] (Guvenius et al., 2003). Adenugba et al. (2009) analysed 15 samples of human bile, collected endoscopically, for seven PCB congeners (PCB-28, PCB-52, PCB-101, PCB-118, PCB-153, PCB-138, and PCB-180). Total PCB concentrations in bile ranged from 6 to 49 ng/mL, and PCB-28 was the predominant congener.

(b) Experimental systems

Elimination half-lives have been estimated in different animal species. In rats, elimination half-lives vary from days (di- and trichlorobiphenyl) to more than 3 months (penta- and

Table 4.1 Estimated human elimination half-lives for nine PCB congeners at background concentrations

Age group	Elimination half-life (years)								
	PCB-28	PCB-52	PCB-105	PCB-118	PCB-138	PCB-153	PCB-170	PCB-180	PCB-187
Children[a]	NR	NR	5.4	5.7	3.7	8.4	7.6	9.1	8
Adults[b]	5.5	2.6	5.2	9.3	10.8	14.4	15.5	11.5	10.5

[a] Grandjean *et al.* (2008), $n = 200$
[b] Ritter *et al.* (2011), $n = 229$
NR, not reported
Adapted from Ritter *et al.* (2011)

hexachlorobiphenyl), while a half-life of approximately 10 months was estimated for Aroclor 1254 in weanling pigs (ATSDR, 2000). Half-lives of a group of congeners (PCB-105, PCB-118, PCB-128, PCB-138, PCB-153, PCB-156, PCB-157, PCB-180, PCB-183) were estimated in monkeys dosed with Aroclor 1254. On average, half-lives varied from 0.4 years (PCB-105) to 1.9 years (PCB-128); however, a wide range of estimates (0.42–7.58 years, depending on individuals) was reported for PCB-128 (Mes *et al.*, 1995b). [These data indicated that PCB half-lives vary according to species, and that PCB half-lives are longer in humans than in experimental animals, including monkeys.]

In rodents, PCBs administered by different routes are mainly excreted in the faeces, with urine usually representing a minor route of excretion.

PCB metabolites that have been identified in urine are mentioned in Section 4.1.3. In addition to OH-PCBs and dihydroxylated PCBs and corresponding glucuronides also observed in other studies, the elimination in urine of sulfated metabolites of PCB-3, PCB-3 2′-sulfate, PCB-3 3′-sulfate, and PCB-3 4′-sulfate after a single intraperitoneal dose of PCB-3 (112 mg/kg bw) was reported. In rats, approximately 3% of the administered dose was excreted in the urine as sulfates over 36 hours, with peak excretion occurring 10–20 hours after exposure (Dhakal *et al.*, 2012). Mercapturic acid of [^{14}C]-2,4′,5-trichlorobiphenyl (PCB-31) was isolated from the urine of rats treated with this congener (Bakke *et al.*, 1982). This metabolite represented 0.3% of the administered dose of 4 mg per rat. About 57% of the administered dose was excreted in the bile, and 30–35% was present as metabolites in the mercapturic acid pathway.

Lactation is also a major route of excretion of PCBs in animals. In monkeys exposed to different doses of Aroclor 1254 in long-term studies, approximately 4% of the intake was eliminated in milk (Mes *et al.*, 1994). The transfer of [^{14}C]-labelled congeners PCB-77, PCB-126, PCB-169, and PCB-105 to milk has been investigated in mice (Sinjari *et al.*, 1996). These compounds were administered intraperitoneally to lactating mice at a single dose of 2.0 µmol/kg bw each on postnatal day 11. Concentrations of PCB-126, PCB-169 and PCB-105 in milk 1 day after administration were higher (1450–2520 pmol/mL) than concentrations of PCB-77 (580 pmol/mL).

In addition to these routes of elimination, other minor pathways have been reported. Studies by Yoshimura & Yamamoto (1975) on PCB-66 in rats have suggested that excretion of unmetabolized PCB through the small intestinal wall may occur. In other experiments with rats, PCBs were excreted unchanged in hair and through the skin (Matthews *et al.*, 1976).

4.2 Genetic and related effects

Since the first *IARC Monograph* on PCBs (IARC, 1978), the genetic and related effects of PCBs have been studied in several experimental systems and in humans (for details and references, see Tables 4.2, 4.3, 4.4, 4.5, 4.6), and summarized in numerous reviews (Safe, 1989; Silberhorn *et al.*, 1990; ATSDR, 2000; Ludewig, 2001).

4.2.1 Exposed humans

Several studies have used cytogenetic effects (structural chromosome aberration, sister-chromatid exchange, and DNA adducts) in cells from body fluids (blood and semen) as biomarkers in humans occupationally or environmentally exposed to PCBs (see Table 4.2).

(a) Genotoxicity and cytogenicity from occupational exposure

Peripheral lymphocytes from 32 workers exposed occupationally to commercial PCB mixtures (DELOR 103 and 106) for up to 25 years were examined for cytogenetic changes. All workers with PCB exposure were smokers and moderate drinkers, and control groups were chosen accordingly: control group 1 consisted of 20 people working outside the PCB-production unit, and control group 2 consisted of 20 employees from administrative offices and the research department (Kalina *et al.*, 1991). Workers with PCB exposure were also exposed to formaldehyde and benzene, but at levels not exceeding national exposure limits. Occupational exposure to PCB mixtures led to an increase in PCB plasma concentrations of more than 100-fold (305–487 µg/L), when compared with the control groups (1.5–3 µg/L). A significant increase in the frequency of chromosomal aberration and sister-chromatid exchange was observed in workers exposed to PCBs for at least 11 years; however, no dose–response effect was observed between cytogenetic effects and PCB blood concentrations. [The Working Group was not able to determine how the PCB plasma concentrations were measured. No quantitative data were provided on the exposure of the workers to benzene and formaldehyde, or on whether all three groups were similarly exposed to benzene and formaldehyde. The choice of control group used for the *t*-test analysis was not clearly indicated].

An increase in structural chromosomal aberration in lymphocytes was also observed in workers occupationally exposed to PCBs when compared with a non-exposed control group; however, no information on PCB blood concentrations or confounders was available (Joksić & Marković, 1992).

Peripheral blood lymphocytes from male workers (n = 21) exposed occupationally to PCBs for 2–5 years at a factory decontaminating industrial transformers and capacitors and from workers in an industrial control group (87; 53 men and 34 women) were analysed for structural and numerical chromosomal aberrations. Significant increases of twofold in the frequency of structural chromosomal aberration and four- to sixfold in the frequency of premature centromere division in mitotic chromosomes were observed in the PCB-exposed group (Jakab *et al.*, 1995; Major *et al.*, 1999). [The Working Group noted that PCB concentrations in blood and/or air were not monitored, the industrial control group was not further specified, and no adjustment for confounders was made.]

Two studies of occupational exposure examined workers exposed to PCBs after a fire at an electric station (Elo *et al.*, 1985; Melino *et al.*, 1992). In one study, maximum blood PCB concentrations (median, 14 µg/L) were reached 3 days after exposure and declined over the course of 1 month to background levels (≤ 2 µg/L). No exposure-related increases in the frequency of structural chromosomal aberration and sister-chromatid exchange in 15 PCB-exposed workers were observed for

Table 4.2 Genetic effects and markers of oxidative DNA damage in humans exposed to PCBs

Target tissue	End-point	Result	Comments	Reference
Occupational exposure				
Peripheral blood lymphocytes	Chromosomal aberration Sister-chromatid exchange	– –	Exposed, 15; unexposed, not defined No details on individual numbers and statistical analysis	Elo *et al.* (1985)
Peripheral blood lymphocytes	Chromosomal aberration	–	Exposed, 45 (29 men, 12 women, 4 children) living within 2 km from capacitor-manufacturing plant (24 workers, 21 residents); unexposed; pre-employment test from workers Heavy smokers excluded; no statistical analysis; no correlation with PCB concentrations (11 congeners) in blood and adipose tissue [no details on PCB concentrations were given]	Tretjak *et al.* (1990)
Peripheral blood lymphocytes	Chromosomal aberration Sister-chromatid exchange	+ ($P < 0.01$) + ($P < 0.05$)	Exposed to technical PCB mixture, 32; unexposed group 1 (working outside production unit), 20; unexposed group 2 (administration and research), 20. Positive correlation with duration of exposure but not blood PCB levels	Kalina *et al.* (1991)
Peripheral blood lymphocytes	Chromosomal aberration Micronucleus formation Sister-chromatid exchange	? + +	Exposed, 48; unexposed, 15 No statistical analysis performed	Joksić & Marković (1992)
Peripheral blood lymphocytes	Chromosomal aberration Sister-chromatid exchange	– –	Exposed, 12; unexposed, 19 No serum PCB concentrations; both groups contained moderate smokers; no confounder taken into account	Melino *et al.* (1992)
Peripheral blood lymphocytes	Chromosomal aberration Premature centromere division	+ ($P < 0.01$) + ($P < 0.01$)	Exposed, 21 (men); unexposed, 87 (53 men,34 women) Heavy smokers (> 20 cigarettes/day); heavy drinkers (> 100 g alcohol/day); donors with neoplasia	Jakab *et al.* (1995), Major *et al.* (1999)
Urine	Oxidative DNA damage (8-OHdG)	–	Study cohort: 64; pre- and post-shift workplace exposure	Wen *et al.* (2008)
Environmental exposure				
Peripheral blood lymphocytes	Chromosomal aberration	–	Exposed, 36 (Yucheng; 17 men, 19 women); unexposed 10 (5 men, 5 women) Sampling of exposed group occurred 3 years after exposure; chromosomal aberrations included breaks, exchanges, acentric fragments, and gaps.	Wuu & Wong (1985)
Peripheral blood lymphocytes	Chromosomal aberration Sister-chromatid exchange *After exposure of lymphocytes to α-naphthoflavone* in vitro*:* Chromosomal aberration Sister-chromatid exchange	– – – + ($P < 0.001$)	Exposed, 35 women (Yucheng victims); unexposed, 24 Blood samples of exposed individuals were taken in 1985 or 5 years after the exposure had occurred; unexposed women were from the same county; all participants were nonsmokers	Lundgren *et al.* (1988)

Table 4.2 (continued)

Target tissue	End-point	Result	Comments	Reference
Peripheral blood lymphocytes	Sister-chromatid exchange	–	Exposed, 16 Yusho patients; unexposed, 39	Nagayama *et al.* (2001)
	Sister-chromatid exchange after exposure of lymphocytes to α-naphthoflavone	–		
Peripheral blood lymphocytes	Micronucleus formation	+ ($P < 0.01$; PCB-118)	Study cohort: up to 1583; age 50–65 years; confounder: age, sex, smoking, lifestyle, body mass index	De Coster *et al.* (2008)
	DNA damage (comet assay)	+ ($P < 0.05$; PCB-118)		
Blood serum	Prostate specific antigen	–		
	Carcinoembryogenic antigen	–		
	TP53	+ ($P < 0.05$; sum of PCB-138, PCB-153, PCB-180)		
Leukocytes	DNA adduct	–	Study cohort: 103 Inuits, categorized into low (1.7–20 µg/L; $n = 54$), medium (21–40 µg/L; $n = 21$) and high (41–143 µg/L; $n = 28$) PCB exposure	Ravoori *et al.* (2008)
Leukocytes	DNA adduct	Negative correlation with PCB ($P < 0.0001$)	Study cohort: 83 Inuits: 56 women, 27 men Effect of age, sex, smoking status, PCB and selenium concentrations on DNA adduct accumulation taken into account	Ravoori *et al.* (2010)
	DNA adduct and 8-OHdG	Negative correlation in the high selenium/PCB ratio group ($P < 0.01$ and $P = 0.014$; respectively)		
Sperm	XY disomy	+ ($P < 0.001$)	Study cohort: 192 men from subfertile couples	McAuliffe *et al.* (2012)
	Total sex-chromosome disomy	+ ($P < 0.001$)		
	XX disomy	Negative correlation ($P < 0.001$)		
Sperm	Sperm chromatid structure	+ ($P < 0.01$)	Study cohort: 176 adult men (Swedish)	Rignell-Hydbom *et al.* (2005)
Sperm	Sperm chromatid structure (DNA fragmentation)	+	Study cohort: 707 adult men (193 Greenland Inuits, 178 Swedish, 141 Polish, and 195 Ukrainian) Statistically positive association for Ukranian and Swedish cohorts, and for European cohorts combined (Sweden, Poland, Ukraine)	Spanò *et al.* (2005)
Sperm	Sperm chromatid structure (DNA fragmentation)	+ ($P < 0.05$)	Study cohort: 652 adult men (200 Greenland Inuits; 166 Swedish, 134 Polish, and 152 Ukrainian) Significant association only for European cohorts combined (Sweden, Poland, Ukraine)	Stronati *et al.* (2006)
Urine	Oxidative DNA damage (8-OHdG)	–	Study cohort: up to 1583; age 50–65 years; confounder: age, sex, smoking, lifestyle, body mass index	De Coster *et al.* (2008)

8-OHdG, 8-hydroxy-2′-deoxyguanosine

Table 4.3 Genetic and related effects of commercial PCB mixtures in experimental systems in vitro

Agent	Test system	Results[a]		Dose[b] (LED or HID), µg/mL	Reference
		Without exogenous metabolic system	With exogenous metabolic system		
Non-mammalian systems					
Aroclor 1221	*Salmonella typhimurium* TA1538, reverse mutation	–	(+)	200	Wyndham *et al.* (1976)
	Saccharomyces cerevisiae, strain RS112, interchromosomal recombination	+	+	10 000	Schiestl *et al.* (1997)
	Salmonella typhimurium TA98, TA1538, reverse mutation	–		5000 µg/plate	Shahin *et al.* (1979)
Aroclor 1254	*Salmonella typhimurium* C3076, D3052, G46, TA98, TA1000, TA1535, TA1537, TA1538, and *Escherichia coli* WP2 *uvrA*, reverse mutation	–	–	NR	Probst *et al.* (1981)
	Salmonella typhimurium TA98, TA100, TA1535, TA1537, reverse mutation	–	–	500	Bruce & Heddle (1979) Schoeny *et al.* (1979)
	Salmonella typhimurium TA98, TA100, TA1535, TA1537, TA1538, and *Escherichia coli* WP2 *uvrA*, reverse mutation	–	–	333	Dunkel *et al.* (1984)
	Salmonella typhimurium TA1538, reverse mutation	–	–	200	Wyndham *et al.* (1976)
	Salmonella typhimurium TA98, TA100, *Escherichia coli* WP2 *uvrA*, reverse mutation	–	–	200 µg/plate	Evandri *et al.* (2003)
	Saccharomyces cerevisae, heterozygous transgenic for human MS32 minisatellite, length mutation	+	NR	6000	Appelgren *et al.* (1999)
Aroclor 1260	*Saccharomyces cerevisiae*, strain RS112, interchromosomal recombination	+	+	15 000	Schiestl *et al.* (1997)
Aroclor 1268	*Salmonella typhimurium* TA1538, reverse mutation	–	–	200	Wyndham *et al.* (1976)
Kanechlor 300	*Salmonella typhimurium* TA1535, TA1536, TA1537, TA1538, TA98, TA100, reverse mutation	–	–	NR	Odashima (1976)
	Salmonella typhimurium TA98, TA100, *Escherichia coli* WP2, reverse mutation	–	–	NR	Sugimura *et al.* (1976)
Kanechlor 500	*Salmonella typhimurium* TA1535, TA1536, TA1537, TA1538, TA98, TA100, reverse mutation	–	–	NR	Odashima (1976)
	Salmonella typhimurium TA98, TA100, *Escherichia coli* WP2, reverse mutation	–	–	NR	Sugimura *et al.* (1976)
Clophen 30	*Drosophila melanogaster*, genetic crossing-over, sex-chromosome loss	–		250	Nilsson & Ramel (1974)
Clophen 50	*Drosophila melanogaster*, genetic crossing-over, sex-chromosome loss	–		200	Nilsson & Ramel (1974)
Mammalian cells in vitro					
Aroclor 1221	Intrachromosomal (non-homologous) recombination at *Hprt* locus, Chinese hamster lung Sp5/V79 cells	–		30	Helleday *et al.* (1998)
	Intrachromosomal (homologous) recombination *Hprt* locus, Chinese hamster lung SPD8/V79 cells	+		20	

Table 4.3 (continued)

Agent	Test system	Results[a]		Dose[b] (LED or HID), µg/mL	Reference
		Without exogenous metabolic system	With exogenous metabolic system		
Aroclor 1221 (cont.)	Intrachromosomal recombination by deletion in *HPRT* locus, human lymphoblastoid GM6804 cells	+		5	Aubrecht *et al.* (1995)
Aroclor 1016	DNA adducts ^{32}P-postlabelling, primary human hepatocytes (three donors)	(+)		23	Borlak *et al.* (2003)
Aroclor 1242	Gene mutation (ouabain resistance), Chinese hamster fibroblast V79 cells	–		150	Hattula (1985)
	Chromosomal aberrations, chicken embryo (*Gallus domesticus*)	–		20	Blazak & Marcum (1975)
Aroclor 1254	DNA single-strand breaks, alkaline elution, rat hepatocytes	+		100	Sina *et al.* (1983)
	DNA strand breaks (comet assay), rat primary prostate cells	+		1	Cillo *et al.* (2007)
	Unscheduled DNA synthesis, primary rat hepatocytes	+		20 (MED)	Althaus *et al.* (1982)
	Unscheduled DNA synthesis, primary F344 rat hepatocytes	–		[16] 50 µM	Probst *et al.* (1981)
	DNA adducts ^{32}P-postlabelling, primary fetal rat hepatocytes		–	[16] 50 µM	Dubois *et al.* (1995)
	DNA adducts ^{32}P-postlabelling, human hepatocarcinoma HepG2 cells	–		[16] 50 µM	Dubois *et al.* (1995)
	DNA adducts ^{32}P-postlabelling, primary human hepatocytes (three donors)	(+)		[20] 60 µM	Borlak *et al.* (2003)
	Detection of repairable adducts by growth inhibition (DRAG) assay in wildtype and DNA repair-deficient Chinese hamster ovary cells	–		135/114, 127, 132[c]	Johansson *et al.* (2004)
	Micronucleus formation, human keratinocytes	–		3	van Pelt *et al.* (1991)
	Chromosomal aberrations, human lymphocytes (five donors)	+		0.1	Sargent *et al.* (1989)
	Cell transformation, Syrian hamster embryo cells	–		50	Pienta (1980)
Clophen A60	Gene mutation (ouabain resistance), Chinese hamster fibroblast V79 cells	–		150	Hattula (1985)
Kanechlor 500 + 600 (plus PCDD/PCDF/ PCB-77, PCB-126, PCB-169 as 0.5% wt)	Sister-chromatid exchange, human lymphocytes	+	+	[0.4 ng WHO-TEQ/g; 0.25 ng WHO-TEQ/g]	Nagayama *et al.* (1994)

[a] +, considered to be positive; (+), considered to be weakly positive in an inadequate study; –, considered to be negative; ?, considered to be inconclusive (variable responses in several experiments within an inadequate study)

[b] Approximately minimal lethal dose not reported.

[c] Dose 135 µg/mL is the IC_{50} concentration inhibiting growth of wildtype CHO cells (AA8) by 50%; doses 114, 127 and 132 µg/mL are the IC_{50} for repair-deficient CHO cells EM9, UV4 and UV5, respectively.

HID, highest effective dose; LED, lowest effective dose; MED, maximum effective dose; PCDD/PCDF, polychlorinated dibenzodioxin/polychlorinated dibenzofuran; TEQ, toxic equivalency

Table 4.4 Genetic and related effects of commercial PCB mixtures in experimental animals in vivo

Agent	Test system	Results[a]	Dose[b] (LED or HID)	Reference
Aroclor 1221	Intrachromosomal recombination by DNA deletion, homozygous C57BL/6J p^{un}/p^{un} mouse	+	1000 ip × 1	Schiestl *et al.* (1997)
Aroclor 1242	DNA adducts ^{32}P-postlabelling, and 8-OHdG, HPLC/ECD-analysis, male Lewis rat liver, thymus, glandular stomach, spleen, testes, seminal vesicles and prostate gland	–	20 po × 1	Schilderman *et al.* (2000)
	Chromosomal aberrations (structural), male Osborne-Mendel rat bone-marrow and spermatogonial cells	–	5000 po × 1	Green *et al.* (1975a)
		–	500 po × 4	
	Dominant lethality, Osborne-Mendel rat	–	2500 po × 1	Green *et al.* (1975b)
		–	250 po × 5	
Aroclor 1254	DNA adducts (I-compounds only) ^{32}P-postlabelling, male Sprague-Dawley rat liver, kidney, lung	–	500 ip × 2	Nath *et al.* (1991)
	DNA adducts ^{32}P-postlabelling, male F344 rat liver	–	25 po × 35	Chadwick *et al.* (1993)
	Unscheduled DNA synthesis, Sprague-Dawley rat, primary hepatocytes	–	300 ip × 1	Kornbrust & Dietz (1985)
	Unscheduled DNA synthesis, rat, primary hepatocytes	–	500 ip × 1	Shaddock *et al.* (1989)
	Unscheduled DNA synthesis, male cynomolgus monkey, primary hepatocytes	–	50 ip × 1	Hamilton *et al.* (1997)
		–	50 ip × 2	
	Micronucleus formation, fish (*C. carpio*), erythrocytes	+	50 ip × 1	Al-Sabti (1986)
	Micronucleus formation, B6C3F$_1$ mouse, bone-marrow cells	–	15 000 ip × 5	Bruce & Heddle (1979)
	Chromosomal aberrations (structural), fish (*C. carpio; T. tinica; C. idella*), kidney cells	+	50 ip × 1	Al-Sabti (1985)
	Chromosomal aberrations (structural), Sprague-Dawley rat, spermatogonial cells	–	50 po × 7	Dikshith *et al.* (1975)
	Chromosomal aberrations (structural), male Osborne-Mendel rat, bone-marrow cells	–	300 po × 5	Green *et al.* (1975a)
	Chromosomal aberrations (structural), male Holtzman rat, bone-marrow and spermatogonial cells	–	500 ppm, 5 weeks	Garthoff *et al.* (1977)
	Sperm morphology, B6C3F$_1$ mice	–	7500 ip × 5	Bruce & Heddle (1979)
	Germline length mutation PC-1 minisatellite, male C57B1/6 mouse, liver	+	100 ip × 1	Hedenskog *et al.* (1997)
	Germline length mutation PC-2 minisatellite, male C57B1/6 mouse, liver	–	100 ip × 1	Hedenskog *et al.* (1997)
	Dominant lethal mutation, Osborne-Mendel rats	–	300 po × 5	Green *et al.* (1975b)
	Gene mutation, transgenic male BigBlue™ mice	(+)	100 ppm in diet, 7 weeks	Davies *et al.* (2000)
Aroclor 1260	Intrachromosomal recombination by DNA deletion, homozygous C57BL/6J p^{un}/p^{un} mouse	+	500 ip × 1	Schiestl *et al.* (1997)
	DNA adducts ^{32}P-postlabelling, male and female B6C3F$_1$ mouse, liver	–	50 po × 1	Whysner *et al.* (1998)
		–	200 ppm × 2 weeks	

Table 4.4 (continued)

Agent	Test system	Results[a]	Dose[b] (LED or HID)	Reference
Kaneclor 300	Chromosomal aberrations, mouse, bone-marrow cells	–	NR[c]	Odashima (1976)
	Chromosomal aberrations, rat, bone-marrow cells	–	NR[c]	Odashima (1976)
Kaneclor 500	Micronucleus formation, male ddY mice, bone-marrow cells	(+)	100 po × 6	Watanabe *et al.* (1982)
		–	100 sc × 6	
	Chromosomal aberrations, mouse, bone-marrow cells	+	NR	Odashima (1976)
	Chromosomal aberrations, rat, bone-marrow cells	–	NR	Odashima (1976)
Kanechlor [no further specification given]	DNA strand breaks (comet assay), ddY male mouse (stomach, colon, liver, kidney, urinary bladder, lung, brain, bone marrow)	–	1000 po × 1	Sasaki *et al.* (2000)
PCB_3[c]	Micronucleus formation, fish (*Misgurnus anguillicaudatus*), erythrocytes	+	0.5 mg/L × 7 d	Chu *et al.* (1996a)
		+	1 mg/L × 2 d	
		–	10 ppm × 12 mo	

[a] +, considered to be positive; (+), considered to be weakly positive in an inadequate study; –, considered to be negative; ?, considered to be inconclusive (variable responses in several experiments within an inadequate study)

[b] In-vivo tests, mg/kg bw

[c] Commercial PCB mixture manufactured in China, the composition of which was similar to that of Aroclor 1242 (see Section 1.1, Table 1.8)

CB, chlorobiphenyl; d, day; HID, highest effective dose; HPLC/ECD, high-performance liquid chromatography electrochemical detection; ip, intraperitoneal; LED, lowest effective dose; mo, month; NR, not reported; 8-OHdG, 8-hydroxy-2'-deoxyguanosine; po, oral administration; TEQ, toxic equivalency

Table 4.5 Genetic and related effects of PCB congeners and their metabolites in experimental systems in vitro

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]		Dose[c] (LED or HID), µg/mL	Reference
			Without exogenous metabolic system	With exogenous metabolic system		
Non-mammalian systems						
2-MonoCB	PCB-1	*Salmonella typhimurium* C3076, D3052, G46, TA98, TA1000, TA1535, TA1537, TA1538, reverse mutation	–	–	1000	McMahon et al. (1979)
4-MonoCB	PCB-3	*Salmonella typhimurium* C3076, D3052, G46, TA98, TA1000, TA1535, TA1537, TA1538, reverse mutation	–	–	1000	McMahon et al. (1979)
4-MonoCB	PCB-3	*Salmonella typhimurium* TA1538, reverse mutation	?	+	50 µg/plate	Wyndham et al. (1976)
4-MonoCB	PCB-3	*Salmonella typhimurium* TA98, TA100, TA1535, TA1537, reverse mutation	–	–	200	Schoeny (1982)
4,4-DiCB	PCB-15	*Salmonella typhimurium* TA98, TA100, reverse mutation	–	–	100	Butterworth et al. (1995)
4,4'-DiCB	PCB-15	*Drosophila melanogaster*, somatic mutation and recombination, eye mosaic test	+	+	223	Butterworth et al. (1995)
2,2',4,4'-TetraCB	PCB-47	*Salmonella typhimurium* TA98, TA100, reverse mutation	–	–	200	Schoeny (1982)
2,2',5,5'-TetraCB	PCB-52	*Salmonella typhimurium* TA1538, reverse mutation	–	–	200 µg/plate	Wyndham et al. (1976)
2,2',5,5'-TetraCB	PCB-52	*Salmonella typhimurium* TA98, TA100, TA1535, TA1537, reverse mutation	NT	–	200 µg/plate	Hsia et al. (1978)
4-OH-2,2',5,5'-TetraCB	4-OH-PCB-52	*Salmonella typhimurium* TA98, TA100, TA1535, TA1537, reverse mutation	NT	–	20 µg/plate	Hsia et al. (1978)
3,4-Epoxy-2,2',5,5'-tetraCB	3,4-Epoxy-PCB-52	*Salmonella typhimurium* TA98, TA100, TA1535, TA1537, reverse mutation	NT	–	200 µg/plate	Hsia et al. (1978)
3,3',4,4'-TetraCB	PCB-77	*Salmonella typhimurium* TA98, TA100, reverse mutation	–	–	200	Schoeny (1982)
2,2',4,4',6,6'-HexaCB	PCB-155	*Salmonella typhimurium* TA98, TA100, reverse mutation	–	–	200	Schoeny (1982)
2,2',3,3',4,4',5,5',6,6'-DecaCB	PCB-209	*Salmonella typhimurium* TA98, TA100, TA1535, TA1537, *Escherichia coli* WP2 *uvrA*, reverse mutation	–	–	5000	Han et al. (2009)

Table 4.5 (continued)

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]		Dose[c] (LED or HID), µg/mL	Reference
			Without exogenous metabolic system	With exogenous metabolic system		
Mammalian cells in vitro						
2',5'-HQ-2-MonoCB	Metabolite of PCB-1	Polyploidy, Chinese hamster lung V79 cells	–		4.4	Flor & Ludewig (2010)
2',5'-HQ-2-MonoCB	Metabolite of PCB-1	Sister-chromatid exchange, Chinese hamster lung V79 cells	–		4.4	Flor & Ludewig (2010)
2',5'-HQ-3-MonoCB	Metabolite of PCB-2	Polyploidy, Chinese hamster lung V79 cells	+		1.1	Flor & Ludewig (2010)
2',5'-HQ-3-MonoCB	Metabolite of PCB-2	Sister-chromatid exchange, Chinese hamster lung V79 cells	–		2.2	Flor & Ludewig (2010)
4-MonoCB	PCB-3	Binding (covalent) to DNA, RNA or protein, Chinese hamster ovary cells	+		2	Wong et al. (1979)
4-MonoCB	PCB-3	Unscheduled DNA synthesis, Chinese hamster ovary cells	(+)		2	Wong et al. (1979)
4-MonoCB	PCB-3	DNA adducts (^{32}P-postlabelling), primary human hepatocytes (three donors)	+		43	Borlak et al. (2003)
4-MonoCB	PCB-3	Gene mutation, Chinese hamster lung V79 cells, *Hprt* locus	–		56	Zettner et al. (2007)
2'-OH-4-MonoCB	Metabolite of PCB-3		–		20	
3'-OH-4-MonoCB	Metabolite of PCB-3		–		20	
4'-OH-4-MonoCB	Metabolite of PCB-3		–		20	
2',5'-HQ-4-MonoCB	Metabolite of PCB-3		–		1.7	
3',4'-HQ-4-MonoCB	Metabolite of PCB-3		–		5.5	
2',5'-Q-4-MonoCB	Metabolite of PCB-3		+		0.1	
3',4'-Q-4-MonoCB	Metabolite of PCB-3		+		0.1	
4-MonoCB	PCB-3	Micronucleus formation, Chinese hamster lung V79 cells	–		38	Zettner et al. (2007)
2'-OH-4-MonoCB	Metabolite of PCB-3		+		10	
3'-OH-4-MonoCB	Metabolite of PCB-3		+		20	
4'-OH-4-MonoCB	Metabolite of PCB-3		+		15	
2',5'-HQ-4-MonoCB	Metabolite of PCB-3		+		0.6	
3',4'-HQ-4-MonoCB	Metabolite of PCB-3		+		3.3	
2',5'-Q4-MonoCB	Metabolite of PCB-3		+		0.1	
3',4'-Q-4-MonoCB	Metabolite of PCB-3		+		0.5	

Table 4.5 (continued)

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]		Dose[c] (LED or HID), µg/mL	Reference
			Without exogenous metabolic system	With exogenous metabolic system		
4-MonoCB	PCB-3	Aneuploidy, Chinese hamster lung V79 cells	–		38	Zettner et al. (2007)
2'-OH-4-MonoCB	Metabolite of PCB-3		+		10	
3'-OH-4-MonoCB	Metabolite of PCB-3		+		20	
4'-OH-4-MonoCB	Metabolite of PCB-3		+		15	
2',5'-HQ-4-MonoCB	Metabolite of PCB-3		+		0.6	
3',4'-HQ-4-MonoCB	Metabolite of PCB-3		+		3.3	
2',5'-Q-4-MonoCB	Metabolite of PCB-3		+		0.5	
3',4'-Q-4-MonoCB	Metabolite of PCB-3		+		1.1	
2',5'-HQ-4-MonoCB	Metabolite of PCB-3	Polyploidy, Chinese hamster lung V79 cells	+		1.1	Flor & Ludewig (2010)
3',4'-HQ-4- MonoCB	Metabolite of PCB-3	Polyploidy, Chinese hamster lung V79 cells	–		2.2	Flor & Ludewig (2010)
2',5'-HQ-4-MonoCB	Metabolite of PCB-3	Sister-chromatid exchange, Chinese hamster lung V79 cells	–		2.2	Flor & Ludewig (2010)
3',4'-HQ-4-MonoCB	Metabolite of PCB-3	Sister-chromatid exchange, Chinese hamster lung V79 cells	+		1.1	Flor & Ludewig (2010)
2',5'-Q-4-MonoCB	Metabolite of PCB-3	Micronucleus formation, human breast epithelial MCF-10A cells	+		0.1	Venkatesha et al. (2008)
2',5'-Q-4-MonoCB	Metabolite of PCB-3	Micronucleus formation, Chinese hamster lung V79 cells	+		0.6	Jacobus et al. (2008)
2,2',5,5'-TetraCB	PCB-52	DNA strand breaks (alkaline sedimentation), mouse fibroblast L-929 cells	+		20	Stadnicki et al. (1979)
4-OH-/3-OH-2,2',5,5'-TetraCB (4 : 1)	Metabolites of PCB-52	DNA strand breaks (alkaline sedimentation), mouse fibroblast L-929 cells	+		20	Stadnicki et al. (1979)
3,4-Epoxy-2,2',5,5'-TetraCB	Metabolite of PCB-52	DNA strand breaks (alkaline sedimentation), mouse fibroblast L-929 cells	+		10	Stadnicki et al. (1979)
2,2',5,5'-TetraCB	PCB-52	DNA strand breaks (comet assay), human lymphocytes (six donors)	(+)		0.3	Sandal et al. (2008)
2,2',5,5'-TetraCB	PCB-52	Sister-chromatid exchange, human lymphocytes (four donors)	–		1	Sargent et al. (1989)
2,2',5,5'-TetraCB	PCB-52	Chromosomal aberrations, human lymphocytes (5–9 donors)	–		1	Sargent et al. (1989)

Table 4.5 (continued)

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]		Dose[c] (LED or HID), µg/mL	Reference
			Without exogenous metabolic system	With exogenous metabolic system		
2,2',5,5'-TetraCB and 3,3',4,4'-tetraCB	PCB-52 + PCB-77	Chromosomal aberrations, human lymphocytes (5–9 donors)	+		$1 + 10^{-5}$	Sargent et al. (1989)
2,2',5,5'-TetraCB and 3,3',4,4'-tetraCB	PCB-52 + PCB-77	Sister-chromatid exchange, human lymphocytes (four donors) in vitro	–		$1 + 10^{-5}$	Sargent et al. (1989)
3-$MeSO_2$-2',3',4,5-TetraCB	5-$MeSO_2$-PCB-56	Sister-chromatid exchange, human lymphocytes	–		7.1	Nagayama et al. (1999)
3-$MeSO_2$-2',3',4,5-TetraCB	5-$MeSO_2$-PCB-56	Micronucleus formation, human lymphocytes	–		7.1	Nagayama et al. (1995)
3,3',4,4'-TetraCB	PCB-77	DNA strand breaks (comet assay), human lymphocytes (three donors)	–		25	Belpaeme et al. (1996a)
3,3',4,4'-TetraCB	PCB-77	DNA strand breaks (comet assay), human lymphocytes (six donors)	(+)		3	Sandal et al. (2008)
3,3',4,4'-TetraCB	PCB-77	DNA adducts ^{32}P-postlabelling, human hepatocarcinoma HepG2 cells	+		15	Dubois et al. (1995)
3,3',4,4'-TetraCB	PCB-77	DNA adducts ^{32}P-postlabelling, primary fetal rat hepatocytes		+	15	Dubois et al. (1995)
3,3',4,4'-TetraCB	PCB-77	Sister-chromatid exchange, human lymphocytes (four donors) in vitro	–		0.1	Sargent et al. (1989)
2,2',5,5'-TetraCB and 3,3',4,4'-tetraCB	PCB-52 + PCB-77	Sister-chromatid exchange, human lymphocytes (four donors) in vitro	–		$1 + 10^{-5}$	Sargent et al. (1989)
3,3',4,4'-TetraCB	PCB-77	Micronucleus formation, human lymphocytes (two donors)	–		500	Belpaeme et al. (1996a)
3,3',4,4'-TetraCB	PCB-77	Chromosomal aberrations (structural), human lymphocytes (5–9 donors)	+		0.01	Sargent et al. (1989)
3-$MeSO_2$-3',4,4',5-TetraCB	5-$MeSO_2$-PCB-77	Sister-chromatid exchange, human lymphocytes	–		6.8	Nagayama et al. (1999)
3-$MeSO_2$-3',4,4',5-TetraCB	5-$MeSO_2$-PCB-77	Micronucleus formation, human lymphocytes	–		7.8	Nagayama et al. (1995)
4,4'-$(OH)_2$-3,3',5,5'-TetraCB	Metabolite of PCB-80	Detection of repairable adducts by growth inhibition (DRAG) assay in wildtype and DNA repair-deficient Chinese hamster ovary cells	(+)		140/102, 92, 91[d]	Johansson et al. (2004)
4-$MeSO_2$-2,2',3',4',5-PentaCB	4'-$MeSO_2$-PCB-87	Sister-chromatid exchange, human lymphocytes	+		5.8	Nagayama et al. (1999)

Table 4.5 (continued)

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]		Dose[c] (LED or HID), µg/mL	Reference
			Without exogenous metabolic system	With exogenous metabolic system		
4-$MeSO_2$-2,2',3',4',5-PentaCB	4'-$MeSO_2$-PCB-87	Micronucleus formation, human lymphocytes	–		5.8	Nagayama et al. (1995)
2,2',4,5,5'-PentaCB	PCB-101	DNA strand breaks (comet assay), fish fibroblast RTG-2 cells	+		16	Marabini et al. (2011)
2,2',4,5,5'-PentaCB	PCB-101	Micronucleus formation, fish fibroblast RTG-2 cells	+		16	Marabini et al. (2011)
3-$MeSO_2$-2,2',4',5,5'-PentaCB	3'-$MeSO_2$-PCB-101	Sister-chromatid exchange, human lymphocytes	+		5.2	Nagayama et al. (1999)
3-$MeSO_2$-2,2',4',5,5'-PentaCB	3'-$MeSO_2$-PCB-101	Micronucleus formation, human lymphocytes	–		5.2	Nagayama et al. (1995)
4-OH-2,3,3',4',5-PentaCB	Metabolite of PCB-109	Detection of repairable adducts by growth inhibition (DRAG) assay in wildtype and DNA repair-deficient Chinese hamster ovary cells	–			Johansson et al. (2004)
2,3',4,4',5-PentaCB	PCB-118	DNA strand breaks (comet assay), fish fibroblast RTG-2 cells	+		10	Marabini et al. (2011)
2,3',4,4',5-PentaCB	PCB-118	Micronucleus formation, fish fibroblast RTG-2 cells	+		10	Marabini et al. (2011)
3,3',4,4',5-PentaCB	PCB-126	Micronucleus formation, human hepatoma HepG2 cells in vitro	–		0.003	Wei et al. (2009b)
2,2',3,4,4',5'-HexaCB	PCB-138	DNA strand breaks (comet assay), fish fibroblast RTG-2 cells	+		25	Marabini et al. (2011)
2,2',3,4,4',5'-HexaCB	PCB-138	Micronucleus formation, fish fibroblast RTG-2 cells	–		25	Marabini et al. (2011)
4-$MeSO_2$-2,2',3',5,5',6'-HexaCB	4'-$MeSO_2$-PCB-151	Sister-chromatid exchange, human lymphocytes	–		9.6	Nagayama et al. (1999)
4-$MeSO_2$-2,2',3',5,5',6'-HexaCB	4'-$MeSO_2$-PCB-151	Micronucleus formation, human lymphocytes	–		9.6	Nagayama et al. (1995)
2,2',4,4'5,5'-HexaCB	PCB-153	Chromosomal aberrations (structural), human lymphocytes (5–9 donors)	+		1	Sargent et al. (1989)

Table 4.5 (continued)

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]		Dose[c] (LED or HID), µg/mL	Reference
			Without exogenous metabolic system	With exogenous metabolic system		
2,2',4,4',5,5'-HexaCB	PCB-153	Micronucleus formation, human breast epithelial MCF-10A cells	+		0.4	Venkatesha et al. (2008)
2,2',4,4',5,5'-HexaCB	PCB-153	Micronucleus formation, human hepatoma HepG2 cells	+		36	Wei et al. (2009a)
2,2',4,4',5,5'-HexaCB	PCB-153	DNA strand breaks (comet assay), fish fibroblast RTG-2 cells	+		11	Marabini et al. (2011)
2,2',4,4',5,5'-HexaCB	PCB-153	Micronucleus formation, fish fibroblast RTG-2 cells	+		11	Marabini et al. (2011)
4-OH-2,2',3,4',5,5',6-HeptaCB	Metabolite of PCB-187	Detection of repairable adducts by growth inhibition (DRAG) assay in wildtype and DNA repair-deficient Chinese hamster ovary cells	–		23	Johansson et al. (2004)
2,2',3,3',4,4',5,5',6,6'-DecaCB	PCB-209	Gene mutation, mouse lymphoma L5178Y cells, *Tk*$^{+/-}$ locus	–	–	150	Han et al. (2009)

[a] BZ nomenclature as listed in Table 1.1, Section 1

[b] +, considered to be positive; (+), considered to be weakly positive in an inadequate study; –, considered to be negative;?, considered to be inconclusive (variable responses in several experiments within an inadequate study); 0, not tested.

[c] Approximately minimal lethal dose not reported.

[d] Dose 140 µg/mL is the IC_{50} concentration inhibiting growth of wildtype CHO cells (AA8) by 50%; 102, 92 and 91 are the IC_{50} for repair-deficient CHO cells EM9, UV4 and UV5, respectively.

CB, chlorobiphenyl; HID, highest effective dose; HQ, hydroquinone; LED, lowest effective dose; MED, maximum effective dose; $MeSO_2$, methyl sulfonyl; OH, hydroxyl

For the nomenclature of PCB metabolites, the reader is referred to the review by Grimm *et al.* (2015).

Table 4.6 Genetic and related effects of PCB congeners and their metabolites in experimental animals in vivo

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]	Dose[c] (LED or HID)	Reference
4-MonoCB 4′-OH-4-MonoCB	PCB-3 Metabolite of PCB-3	Gene mutation, transgenic male BigBlue® rat, liver	+ –	113 ip × 4 82 ip × 4	Lehmann *et al.* (2007)
4-MonoCB 4′-OH-4-MonoCB	PCB-3 Metabolite of PCB-3	Gene mutation, transgenic male BigBlue® rat, lung	(+) (+)	113 ip × 4 (1/week) 82 ip × 4 (1/week)	Maddox *et al.* (2008)
4-MonoCB 4′-OH-4-MonoCB	PCB-3 Metabolite of PCB-3	Gene mutation, transgenic female BigBlue® rat, liver	– –	113 ip × 4 82 ip × 4	Jacobus *et al.* (2010)
2,2′,5,5′-TetraCB	PCB-52	Chromosomal aberrations (numerical and structural), female Sprague-Dawley rat, 70% hepatectomy, bone-marrow cells	–	10 ppm, 1 year	Meisner *et al.* (1992)
2,2′,5,5′-TetraCB	PCB-52	Chromosomal aberrations (numerical), female Sprague-Dawley rat, liver cells after 70% hepatectomy	– –	10 ppm × 7 mo 10 ppm × 12 mo	Sargent *et al.* (1992)
3,3′,4,4′-TetraCB	PCB-77	Chromosomal aberrations (numerical & structural), female Sprague-Dawley rat, 70% hepatectomy, bone-marrow cells	–	0.1 ppm, 1 year	Meisner *et al.* (1992)
3,3′,4,4′-TetraCB	PCB-77	Chromosomal aberrations (numerical), female Sprague-Dawley rat liver cells after 70% hepatectomy	 –	0.1 ppm × 7 mo 0.1 ppm × 12 mo	Sargent *et al.* (1992)
3,3′,4,4′-TetraCB	PCB-77	DNA strand breaks (comet assay) and micronucleus formation, fish (*Salmo trutta fario*) erythrocytes	–	0.9 µg/mL	Belpaeme *et al.* (1996b)
3,3′,4,4′-TetraCB and 2,2′,5,5′-tetraCB	PCB-77 + PCB-52	Chromosomal aberrations (numerical & structural), female Sprague-Dawley rat, 70% hepatectomy, bone marrow cells	+	0.1 + 10 for 1 year	Meisner *et al.* (1992)
3,3′,4,4′ -TetraCB + 2,2′,5,5′-tetraCB	PCB-77 + PCB-52	Chromosomal aberrations (numerical), female Sprague-Dawley rat liver cells after 70% hepatectomy	–	0.1 + 10 ppm for 7 mo	Sargent *et al.* (1992)
3,3′,4,4′,5-PentaCB	PCB-126	Gene mutation, transgenic Muta™Mouse fetus, day 18 of gestation, after exposure on day 10, in utero	–	0.5 po × 1	Inomata *et al.* (2009)
3,3′,4,4′,5-PentaCB	PCB-126	DNA adducts, M_1dG secondary oxidative DNA lesion, LC-MS/MS female Sprague-Dawley rat, liver	+	0.001 po × 5 per week for 53 weeks	Jeong *et al.* (2008)
2,2′,4,4′,5,5′-HexaCB	PCB-153	DNA adducts, M_1dG secondary oxidative DNA lesion, LC-MS/MS female Sprague-Dawley rat, liver	–	1 po × 5 per week for 53 weeks	Jeong *et al.* (2008)
2,2′,4,4′,5,5′-HexaCB	PCB-153	DNA adducts, M_1dG secondary oxidative DNA lesion, LC-MS/MS female Sprague-Dawley rat, brain	–	1 po × 5/week for 53 weeks	Jeong *et al.* (2008)

Table 4.6 (continued)

PCB congener Structural name	BZ nomenclature[a]	Test system	Results[b]	Dose[c] (LED or HID)	Reference
3,3′,4,4′,5-PentaCB and 2,2′,4,4′,5,5′-hexaCB	PCB-126 + PCB-153	DNA adducts, M_1dG secondary oxidative DNA lesion, LC-MS/MS female Sprague-Dawley rat, liver	+	0.0003 + 3 po × 5/ week for 53 weeks	Jeong *et al.* (2008)
3,3′,4,4′,5-pentaCB and 2,2′,4,4′,5,5′-hexaCB	PCB-126 + PCB-153	DNA adducts, M_1dG secondary oxidative DNA lesion, LC-MS/MS female Sprague-Dawley rat, brain	–	0.001 + 1 po × 5/week for 53 weeks	Jeong *et al.* (2008)
2,2′,3,3′,4,4′,5,5′,6,6′-DecaCB	PCB-209	Micronucleus formation, male and female Crl:CD1 mice bone-marrow cells	–	2000 po × 1	Han *et al.* (2009)
1 : 2 : 3 : 2 Mixture of 2,3′,4,4′,5-pentaCB, 2,2′,3,4,4′,5′-hexaCB, 2,2′,4,4′,5,5′-hexaCB, and 2,2′,3,4,4′,5,5′-heptaCB	PCB-118, PCB-138, PCB-153, PCB-180	DNA adducts, M_1dG secondary oxidative DNA lesion, LC-MS/MS female C57BL/6J mouse, liver	–	10 ng TEQ/kg bw ip × 1	Jeong *et al.* (2008)

[a] BZ nomenclature as listed in Table 1.1, Section 1.

[b] +, considered to be positive; (+), considered to be weakly positive in an inadequate study; –, considered to be negative; ?, considered to be inconclusive (variable responses in several experiments within an inadequate study)

[c] In-vivo tests, mg/kg bw

CB, chlorobiphenyl; HID, highest effective dose; ip, intraperitoneal; mo, month; LED, lowest effective dose; po, oral administration; TEQ, toxic equivalency; 8-OHdG, 8-hydroxy-2′-deoxyguanosine; HPLC/ECD, high-performance liquid chromatography electrochemical detection; I-compounds, take from Table 4.3 or Table 4.4

7 months (Elo *et al.*, 1985). [The Working Group noted that the control group was not defined. The data on observed chromosomal aberration and sister chromatid exchange, and the statistical method used, were not provided.] The other study reported a non-significant increase by fourfold in the frequency of chromosomal aberration, but no increase in the frequency of sister-chromatid exchange in a group of 12 workers (Melino *et al.*, 1992).

In another report, the study group consisted of 45 randomly selected people (workers, residents, or children) living within 2 km of a capacitor-producing factory known to cause occupational and environmental exposure to PCBs, in Semic, Slovenia, and was compared to workers that had pre-employment tests. An abnormally high frequency of structural chromosome aberration (55%) was observed in peripheral lymphocytes from workers and residents when compared with the control group (Tretjak *et al.*, 1990). However, these findings were not correlated to environmental or blood PCB concentrations. [The Working Group noted that no PCB concentrations in blood were reported. No matched control group was available and no statistical analysis was performed. Heavy smokers and people who had had recent X-ray examinations were excluded from the study].

Men working in Chinese electrical and electronic equipment waste-dismantling factories were shown to be exposed occupationally to PCBs, tetrachlorodibenzo-*p*-dioxins and dibenzofurans (TCDD/Fs) and polybrominated diphenyl ethers (PBDEs). Urine concentrations of 8-hydroxydeoxyguanosine (8-OHdG), a product of oxidative DNA damage, were significantly increased in workers after their working shift when compared with levels before the working shift. However, no correlation could be drawn between the observed increase in urinary 8-OHdG concentrations and occupational exposure to any of the organochlorine compounds (Wen *et al.*, 2008).

(b) Genotoxicity and cytogenicity from non-occupational exposure

Three years after accidental contamination of cooking oil with PCBs in Taiwan, China (see Section 1.4.8), blood samples were taken from 36 patients with Yucheng ("oil disease"); lymphocytes were analysed for chromosomal aberrations and compared with lymphocytes from age- and sex-matched laboratory staff (n = 10). Blood PCB concentrations ranged from 6.4 to 101.8 μg/L. A high frequency of chromosomal aberration was observed in 19 out of 36 (53%) PCB-exposed patients, while none was seen in the control group. The findings could not be correlated with the blood PCB levels (Wuu & Wong, 1985). [The Working Group noted that no details on the statistical evaluation or adjustment for confounders were given.]

The frequencies of chromosomal aberration and sister-chromatid exchange in peripheral lymphocytes from 35 nonsmoking women from Taiwan, China, exposed to PCBs through contaminated rice oil ("Yucheng") were similar to those from matched controls. However, when peripheral blood lymphocytes were treated with α-naphthoflavone in vitro [to increase sensitivity], a small (20%) but significant increase in frequency of sister-chromatid exchange, but not chromosomal aberration, was observed (Lundgren *et al.*, 1987, 1988).

Similarly, 27 years after exposure to high concentrations of PCBs, the frequency of sister-chromatid exchange in lymphocytes of 16 victims of the "Yusho" food poisoning incident (see Section 1.4.8 in this *Monograph*) were not significantly different from those of a non-exposed control group, despite persistently elevated blood PCB concentrations in these patients (281 pg/g fat versus 41 pg/g fat in the control group). Addition of α-naphthoflavone did not increase the frequency of sister-chromatid exchange (Nagayama *et al.*, 2001).

Blood concentrations of cadmium, lead, *p,p′*-dichlorodiphenyldichloroethylene (DDE), hexachlorobenzene, PCBs (PCB-99, PCB-118, PCB-170, PCB-138, PCB-153, PCB-180), and dioxin-like activity (Calux assay) were analysed in 1583 residents in nine different industrialized regions in Belgium (De Coster *et al.*, 2008). Also analysed as effect biomarkers were the percentage of cells with micronucleus formation, DNA damage (comet assay) in peripheral blood cells, and 8-OHdG in urine. Overall significant differences between the different regions were found for micronucleus formation, DNA damage, and urinary 8-OHdG concentrations. Among these, positive correlations were reported between PCB-118 concentrations and both micronucleus formation and DNA damage.

In a group of 103 Inuit people from Northern Canada exposed to high dietary concentrations of PCBs and selenium, plasma PCB concentrations and DNA adduct profiles in leukocytes were determined (Ravoori *et al.*, 2008). The ^{32}P-postlabelling technique used allowed for differentiation between polar and lipophilic adducts. Plasma PCB concentrations were significantly correlated with increasing age [$P < 0.01$]. The most abundant PCB congeners in the plasma were PCB-138, PCB-153, and PCB-180. The most abundant adduct was 8-OHdG, which accounted for 51–57% of the total adduct burden. No correlation between adduct levels and specific PCB congeners, smoking status, or sex were observed.

In a follow-up study in 83 subjects, Ravoori *et al.* (2010) reported 30–800-fold interindividual variability in levels of unidentified polar DNA adducts (indicative of oxidative stress) in leukocytes. Negative associations were observed between total DNA adduct levels and selenium, and PCB concentrations, the latter being significant. After grouping the individuals according to selenium/PCB ratio as high-ratio (ratio, > 33; mean, 75.5; $n = 41$), or low-ratio (ratio, ≤ 33; mean, 18; $n = 42$), levels of 8-OHdG and total DNA adducts were significantly negatively correlated with the high-ratio group ($P = 0.014$ and $P < 0.01$, respectively), while there was no correlation with the low-ratio group, indicating a mitigating effect of selenium on the toxicity of PCBs.

(c) *Sperm DNA damage*

Sex-chromosome disomy in sperm nuclei was determined in 192 men from subfertile couples. A positive association with YY, XY, and total sex-chromosome disomy and an inverse association with XX disomy were observed with higher serum concentrations of four PCBs (PCB-118, PCB-138, PCB-153, and PCB-180) (McAuliffe *et al.*, 2012). Other environmental organochlorine pollutants may also have contributed to sex-chromosome aneuploidy, since plasma DDE concentrations were positively associated with increased rates of XX, XY, and total sex-chromosome disomy.

In a group of 176 Swedish fishermen with low or high consumption of fatty fish, the DNA fragmentation index in sperm was compared with serum PCB concentrations (Rignell-Hydbom *et al.*, 2005). Plasma concentration of PCB-153 was statistically significantly associated with an increase in DNA fragmentation ($P < 0.001$); however, when adjusted for age, which was strongly associated with percentage DNA fragmentation index, this association was no longer significant ($P = 0.28$). When PCB-153 concentrations were categorized into quintiles, the lowest-exposure quintile had significantly lower levels of DNA fragmentation than the other quintiles ($P < 0.001$), even after adjustment for age ($P = 0.006$). The association between DNA fragmentation and DDE concentrations was not significant (Rignell-Hydbom *et al.*, 2005).

In sperm samples from 707 adult men (193 Inuits from Greenland, 178 Swedish fishermen, 141 men from Poland, and 195 men from Ukraine), DNA fragmentation was correlated with serum PCB-153 concentrations (Spanò *et al.*, 2005). After adjustment for age, period of sexual abstinence, and serum PCB-153 concentration,

levels of DNA fragmentation betwen men in the three European groups did not differ considerably, but were significantly higher than those found in Inuit men. While DNA fragmentation in sperm was unrelated to PCB-153 concentration among the Inuits (very high PCB concentrations) and Polish men (very low PCB concentrations), increasing serum PCB-153 concentrations were significantly associated with increased DNA fragmentation in the Swedish (P = 0.001), and Ukranian cohorts (P = 0.027), and in the three European groups combined (P < 0.0001). No correlation between DNA fragmentation index and serum DDE concentrations was seen.

Similar results were observed in a subsequent study with a largely overlapping study population (Stronati *et al.*, 2006).

(d) Gene mutation

A possible correlation between PCB exposure and cancer of the pancreas has been discussed earlier (see Section 2.3.5). An analysis of blood organochlorine concentrations and *KRAS* mutations in tissue from pancreatic cancer found a significant correlation between tumours harbouring *KRAS* mutations and PCB-138, and PCB-153, and between the two most common mutations in *KRAS* and PCB-138 concentrations (Porta *et al.*, 2009). The dose–response pattern was approximately linear only for PCB-138.

Another study analysed post-mortem samples of brain from patients with neurodevelopmental disorders with a known genetic basis (n = 32), autism of unknown etiology (n = 32), and controls (n = 43) for eight PCBs (PCB-28, PCB-95, PCB-105, PCB-118, PCB-138, PCB-153, PCB-170, and PCB-180) (Mitchell *et al.*, 2012). The concentration of PCB-95 was significantly higher in the group with genetic neurodevelopmental diseases. In fact, PCB-95 was detected nearly exclusively in the brain of patients from mothers with a specific duplication in the long arm of chromosome 15 (dup15q11–q13) or deletions in the same chromosome 15q11–q13 in patients with Prader-Willie syndrome. Five out of six patients with dup15q11–q13, which is related to autism spectrum disorder, were born after 1976.

(e) Epigenetic effects

In the study by Mitchell *et al.* (2012) cited above, samples of brain showing dup15q also showed a lower level of methylation in regions of repetitive DNA, suggesting that PCBs may have caused hypomethylation in these regions, resulting in chromosome instability and a higher risk of duplication.

Rusiecki and coworkers used pyrosequencing to estimate global DNA methylation via repetitive elements *Alu* and (long interspersed nucleotide element) LINE-1 assays of bisulfite-treated DNA in 70 samples from Inuit people in Greenland to examine epigenetic effects of high PCB contamination (Rusiecki *et al.*, 2008). They observed significant inverse correlations between percentages of methylcytosine and plasma concentrations of DDT, DDE, β-hexachlorocyclohexane, oxychlordane, α-chlordane, mirex, sum of PCBs, and sum of all persistent organic pollutants, after adjusting for age and cigarette smoking.

(f) Changes in gene expression

In samples taken in 2007 from 139 daughters of members of a cohort of fish-consumers in Michigan, there was no correlation between serum concentrations of PCB, PBDE, or DDE, and expression of four genes encoding 17-α-hydroxylase (CYP17A1), aromatase (CYP19A1), and estrogen receptor α and β (ESR1 and ESR2) (Karmaus *et al.*, 2011). In contrast, maternal concentrations of serum PCB (prenatal PCB concentration), measured in 1973–1991, were highly significantly associated with decreased expression of the steroid synthesis genes *CYP17* and *CYP19* in blood lymphocytes. Other persistent organic pollutants were not correlated.

4.2.2 Experimental systems

(a) Commercial PCB mixtures

Table 4.3 and Table 4.4 summarize data with commercial PCB mixtures in in-vitro and in-vivo studies respectively. For each category of test (non-mammalian systems, mammalian cells in vitro, and in-vivo assays), the data are presented by commercial PCB mixture in increasing order of chlorination, and for each commercial mixture, by end-point.

(i) Non-mammalian systems

All PCB mixtures tested for their ability to induce gene mutation in bacteria, i.e. PCB mixtures with chlorination levels ranging from ~20% (e.g. Aroclor 1221) to ~70% (e.g. Aroclor 1268) were not mutagenic in different strains of *Salmonella typhimurium* and *Escherichia coli* in the absence or presence of an exogenous metabolic activation system comprising induced and non-induced liver microsomes (Table 4.3). However, only Aroclor 1254 was tested up to the recommended limit dose for hazard assessment of 5000 µg/plate (Shahin *et al.*, 1979), not all strains typically used in the Ames test battery (*S. typhimurium* TA98, TA100, TA1535, TA1537) or *E. coli* WP2 *uvrA* were tested, and an exogenous metabolic system was not always included.

In contrast, Aroclor 1221 and Aroclor 1260 did induce intrachromosomal recombination in *Saccharomyces cerevisiae* cells in the absence and presence of exogenous metabolic activation. Since Aroclor 1221 was effective at lower concentrations than Aroclor 1260, chlorination level seemed to be inversely correlated to mutagenicity of PCBs in this test system (Schiestl *et al.*, 1997).

Additionally, Aroclor 1254 induced mutations in the number of tandem repeats in *S. cerevisiae* transgenic for the human MS32 minisatellite (Appelgren *et al.*, 1999).

Clophen mixtures did not induce somatic mutation in the fruit fly *Drosophila melanogaster* (Nilsson & Ramel, 1974).

(ii) Mammalian cells in vitro

Aroclor 1254 caused DNA strand breaks (detected by alkaline filter elution) in primary rat hepatocytes (Sina *et al.*, 1983) and in primary rat prostate cells (comet assay; Cillo *et al.*, 2007), while evidence for induction of unscheduled DNA synthesis in primary rat hepatocytes was equivocal (Probst *et al.*, 1981; Althaus *et al.*, 1982). An increase in the frequency of DNA adducts (detected by ^{32}P-postlabelling) was observed in primary human hepatocytes from three different donors (Borlak *et al.*, 2003), but not in cultured human hepatocarcinoma HepG2 cells or dexamethasone-treated primary rat fetal hepatocytes (Dubois *et al.*, 1995). A dose-dependent increase in structural chromosomal aberration starting at concentrations of less than 1 µg/mL was seen in cultured human lymphocytes (Sargent *et al.*, 1989).

Aroclor 1221 caused intrachromosomal recombination at the *Hprt* locus in a mutant Chinese hamster V79 cell line (Helleday *et al.*, 1998), and in human lymphoblastoid cells (Aubrecht *et al.*, 1995). Aroclor 1016 enhanced DNA-adduct formation in primary human lymphocytes (Borlak *et al.*, 2003); no increase in the frequency of chromosomal aberration was seen in chicken embryos and ouabain-resistant colonies in Chinese hamster V79 cells treated with Aroclor 1242 (Blazak & Marcum, 1975; Hattula, 1985).

(iii) In-vivo assays

Repeated doses of Aroclor 1254 did not alter hepatic levels of DNA adducts (as measured by ^{32}P-postlabelling) in male Sprague-Dawley (given two intraperitoneal doses of 500 mg/kg bw) or male Fischer 344 rats (given 35 oral doses of 25 mg/kg bw) compared with controls (Nath *et al.*, 1991; Chadwick *et al.*, 1993).

When used for hepatic enzyme induction, a single intraperitoneal application of Aroclor 1254 of up to 500 mg/kg bw in rats (Kornbrust & Dietz, 1985; Shaddock *et al.*, 1989) and 50 mg/kg bw

in cynomolgus monkeys (Hamilton *et al.*, 1997) did not enhance unscheduled DNA synthesis in isolated primary hepatocytes.

Dietary exposure of male C57BL/6 (Big Blue®) mice transgenic for bacterial *lacI* to Aroclor 1254 at 100 ppm (0.01%) for 7 weeks caused a significant, but less than twofold, increase in the frequency of mutation in the liver (Davies *et al.*, 2000).

No increase in the frequency of structural chromosomal aberration in bone marrow and spermatogonial cells was observed in rats given repeated doses of Aroclor 1254 by gavage (300 mg/kg bw for five consecutive days or 50 mg/kg bw for seven consecutive days) or in the diet (500 ppm for 5 weeks) (Dikshith *et al.*, 1975; Green *et al.*, 1975a; Garthoff *et al.*, 1977). Aroclor 1254 did not increase the frequency of micronucleus formation in bone marrow of $B6C3F_1$ mice given Aroclor 1254 as intraperitoneal injections of 15 000 mg/kg bw on five consecutive days (Bruce & Heddle, 1979).

In contrast to the observations in rodents, a single intraperitoneal injection of Aroclor 1254 induced a dose-dependent increase in the frequency of micronucleus formation in fish (*Cyprinus carpio*) erythrocytes (Al-Sabti, 1986), and aberrant metaphases and structural chromosomal aberration in fish kidney cells (*Cyprinus carpio*, *Tinca tinica*, *Ctenopharyngodon idella*), from the starting dose of 50 mg/kg bw (Al-Sabti, 1985). In addition, Aroclor 1254 induced germline length mutation in the PC-1 but not PC-2 minisatellite region in male C57B1/6 mice given a single intraperitoneal dose of Aroclor 1254 at 100 mg/kg bw (Hedenskog *et al.*, 1997).

Kanechlor 500 (which has a similar level of chlorination as Arochlor 1254) caused a weak (less than twofold) increase in the frequency of micronucleus formation in bone-marrow cells in male ddY mice given an oral dose at 100 mg/kg bw for 6 days, but not when applied subcutaneously at the same dose (Watanabe *et al.*, 1982).

A single dose of Aroclor 1242 did not enhance levels of DNA adducts (as measured by ^{32}P-postlabelling) or 8-OHdG formation (as measured by high-performance liquid chromatography/electrochemical detection) in liver, glandular stomach, spleen, thymus, prostate, testes, and seminal vesicles of male Lewis rats, nor did Aroclor 1242 induce structural chromosomal aberrations in bone marrow and spermatogonial cells of Osborne-Mendel rats given a single oral dose of 5000 mg/kg bw, or repeated doses of 500 mg/kg bw for 4 days (Green *et al.*, 1975a; Schilderman *et al.*, 2000).

Aroclor 1242, like Aroclor 1254, did not reduce the number of mitotic spermatogonial cells in Osborne-Mendel rats at the highest doses tested (Green *et al.*, 1975a), and had no effect on the number of dominant lethals (Green *et al.*, 1975b).

A study by Desaulniers *et al.* (2009) examined the effects of PCB and organochlorine pesticide mixtures on DNA methylation in the liver of exposed rats. The PCB mixture, but not the organochlorine pesticide mixture, reduced the mRNA abundance of DNA methyltransferase-1, -3a, and -3b, reduced the abundance of the methyl donor *S*-adenosylmethionine, and decreased the methylation of CpG sites in the promoter region of the tumour suppressor gene $p16^{INK4a}$.

Another group analysed histone post-translational modifications in chromatids from liver of rats exposed to PCBs in early life (Casati *et al.*, 2012). There was a decrease in levels of histone H4K16Ac and histone H3K4me3, and an increase in the expression of *SirtT1* and *Jarid1b*, genes encoding two chromatid-modifying enzymes (histone demethylases). A decrease in the abundance of mRNA of androgen receptor, a histone enzyme coregulator, was also reported.

Ghosh *et al.* (2011) applied the tools of global gene expression and Ingenuity biological functions analysis to peripheral blood mononuclear cells (PBMC) exposed in vitro to PCB-138 (0.87 ng/mL) or PCB-153 (1.42 ng/mL) for 48

hours. The expression of several biologically significant genes was highly modulated in vitro, in general by downregulation, and differential gene expression was specific to the PCB used. Exposure to PCB-153 identified genes involved in three Ingenuity Pathway Analysis (IPA) networks involved in cellular movement, development and function of the haematological system, immune cell trafficking, molecular transport, and cancer. Exposure to PCB-138 resulted in significant expression of several genes including tumour necrosis factor-associated protein 1 (*TRAP1*), contactin 5 (human neuronal NB-2 gene) (*CNTN5*), glial cell line-derived neurotrophic factor family receptor α-1 (*GFRA1*), von Willebrand factor D and EGF domains (*VWDE*), and *CYP1A2*. Notable among these are the upregulated genes *TRAP1*, *CNTN5*, *GFRA1*, which are important in the activation of *TRAP-1*.

Using the same genomic methods, Hochstenbach *et al.* (2010) reported alterations indicative of exposure to immunotoxicants in whole genome gene-expression profiles (transcriptomic changes) in human PBMC from two healthy donors exposed in vitro to a range of immunotoxic chemicals including PCB-153.

Wens *et al.* (2013) studied gene-expression profiles in PBMC exposed in vitro to a dioxin-like polychlorinated biphenyl, PCB-126 (1 μM), or a non dioxin-like polychlorinated biphenyl, PCB-153 (10 μM). Hierarchal cluster analysis created distinct clustered gene groups for samples exposed to PCB-126 or PCB-153. The number of differentially expressed genes varied with the compound used and ranged from 60 to 192. As expected, exposure to PCB-126 caused induction of the AhR signalling pathway. Exposure to PCB-153, which is known to disrupt thyroid metabolism, resulted in expression of the nuclear estrogen receptor *ESR2*.

(b) Individual congeners and their metabolites

In this section, the data in the text are presented first for non-mammalian systems and then combined for cell culture tests and in-vivo assays, by PCB congener and corresponding metabolite(s) (Table 4.5 and Table 4.6). Data in the table are presented first for non-mammalian systems and cell culture tests (Table 4.5), and then for in-vitro assays (Table 4.6).

(i) Non-mammalian systems

In tests for gene mutation in bacteria, the PCB congeners PCB-1, PCB-3, PCB-15, PCB-47, PCB-52, PCB-77, PCB-155, and PCB-209 were not mutagenic in various strains of *Salmonella typhimurium* and *Escherichia coli* in the absence or presence of exogenous metabolic activation (induced and non-induced liver microsomes), except in one study with PCB-3 in *S. typhimurium* TA1538 in the presence of rabbit liver microsomes (Wyndham *et al.*, 1976). Only PCB-209 was tested up to the recommended limit dose of 5000 μg/plate and in all strains typically used in the Ames test battery, i.e. *S. typhimurium* TA98, TA100, TA1535, TA1537, and in *E. coli* WP2 *uvrA* (Han *et al.*, 2009).

The less chlorinated congener PCB-15 was reported to induce somatic mutation in *Drosophila melanogaster* (Butterworth *et al.*, 1995).

(ii) Cell culture tests and in-vivo assays

Several studies have shown in vitro or in non-humans in vivo that PCB congeners with one to four chlorine atoms are bioactivated to DNA- and protein-binding intermediates in vitro and in vivo. Each congener produced multiple different DNA adducts, particularly with guanine. The most prominent ultimate DNA-binding intermediates were quinone metabolites, but some binding of epoxide intermediates was suggested. Rodent and human liver microsomes produced similar or different adduct patterns depending on the PCB congener used, indicating that

species differences exist. Reactive intermediates can bind to cellular macromolecules, including DNA and DNA-maintenance proteins, and such adducts can be detected in multiple organs (Morales & Matthews, 1979; Amaro *et al.*, 1996; McLean *et al.*, 1996b; Oakley *et al.*, 1996a, 1996b; Lin *et al.*, 2000; Pereg *et al.*, 2001, 2002; Srinivasan *et al.*, 2002; Arif *et al.*, 2003; Zhao *et al.*, 2004; Bender *et al.*, 2006; Bender & Osheroff, 2007).

PCB-1, PCB-2, PCB-3 and metabolites

Without exogenous metabolic activation, tritium-labelled PCB-3 was reported to bind to DNA, RNA, and cellular proteins in cultured Chinese hamster ovary cells (Wong *et al.*, 1979). PCB-3 also enhanced unscheduled DNA synthesis by 1.6-fold in the same cell line (Wong *et al.*, 1979), and increased DNA-adduct formation dose-dependently in primary human hepatocytes, as determined by ^{32}P-postlabelling (Borlak *et al.*, 2003). Maximum adduct levels were observed 24 hours after exposure and declined to control levels within 48 hours (Borlak *et al.*, 2003).

The mutagenicity of PCB-3, its mono- and dihydroxylated metabolites, and its 3′,4′- and 2′,5′-quinones was investigated in cultured Chinese hamster V79 cells (Zettner *et al.*, 2007). Induction of gene mutations at the *Hprt* locus was determined by 6-thioguanine resistance. Induction of chromosomal and genomic mutation was assessed by micronucleus formation and immunochemical differentiation of micronuclei containing whole chromosomes (kinetochore-positive) or DNA fragments (kinetochore-negative). Both quinones, but not the PCB-3 itself or its mono- or dihydroxylated metabolites, caused a dose-dependent increase in the frequency of 6-thioguanine-resistant colonies at non-cytotoxic concentrations, and an increase in chromosomal and genomic mutation was observed at higher, cytotoxic concentrations.

In addition, the 2′,5′-dihydroxylated metabolites of PCB-3 and PCB-2, but not of PCB-1, or the 3′,4′-dihydroxy-PCB-3 induced polyploidy in V79 cells; of these dihydroxylated metabolites, only 3′,4′-dihydroxy-PCB-3 increased the levels of sister-chromatid exchange (Flor & Ludewig, 2010).

As in V79 cells, PCB-3–2′,5′-quinone caused a dose-dependent increase in the frequency of micronucleus formation in human breast epithelial MCF-10A cells (Venkatesha *et al.*, 2008). At the concentrations tested, electron paramagnetic resonance showed an increase in steady-state levels of ROS, and detected the presence of a semiquinone radical, suggesting redox cycling of the 2′,5′-quinone. Furthermore, the increase in number of micronucleated cells observed with PCB-3–2′,5′-quinone and also with PCB-153 was consistent with an increase in levels of phosphorylated histone protein γ-H2AX (Venkatesha *et al.*, 2008). The 2′,5′-quinone of PCB-3 also caused significant and dose-dependent shortening of the telomeres in human keratinocyte HaCaT cells after 11 weeks of exposure, and an increase in frequency of micronucleus formation in V79 cells (Jacobus *et al.*, 2008).

Induction of gene mutation in vivo by PCB-3 and its monohydroxylated metabolite 4′-OH-PCB-3 was investigated in male and female transgenic Fischer 344 (Big Blue®) rats given four intraperitoneal injections of PCB-3 at 113 mg/kg bw and 4′-OH-PCB-3 at 82 mg/kg bw over 4 weeks. Seventeen days after the last injection, the frequency and spectrum of mutation in the *lacI* gene were determined in the liver (Lehmann *et al.*, 2007) and lung (Maddox *et al.*, 2008) of males, and in the liver of females (Jacobus *et al.*, 2010). Both PCB-3 and its 4′-OH-metabolite caused a similar, more than twofold, increase in mutation frequency in the liver of male rats; however, only the increase observed with PCB-3 was statistically significant. Although the mutation spectrum induced by PCB-3 was different from that in control rats, and similar to that induced by the positive control, 3-methylcholanthrene, only the proportion of transitions was statistically different from that in control

rats. In contrast, the mutation spectrum for 4′-OH-PCB-3 differed only slightly from that in the control group (Lehmann *et al.*, 2007). In the liver of female rats treated with PCB-3 and its 4′-OH-metabolite, mutation frequencies and mutation spectra were not significantly different from those observed in control rats (Jacobus *et al.*, 2010). PCB-3 and its 4′-OH-metabolite caused a twofold, but not statistically significant, increase in mutation frequency in the lungs of treated males. However, a shift in the mutation spectra, especially with PCB-3, and an increase in the frequency of mutation outside of the hotspot region for spontaneous mutation of *lacI* (base pairs 1–400) were observed (Maddox *et al.*, 2008). The genotoxicity profile of metabolites of PCB-3 is summarized in Table 4.7.

PCB-28, PCB-52, PCB-77

PCB-52 enhanced the frequency of DNA strand breaks in human lymphocytes (comet assay) and mouse fibroblast L-929 cells (alkaline sedimentation), but had no effect on the level of sister-chromatid exchange and structural chromosomal aberration in human lymphocytes (Stadnicki & Allen, 1979; Stadnicki *et al.*, 1979; Sargent *et al.*, 1989; Sandal *et al.*, 2008). However, the addition of PCB-77 at non-genotoxic concentrations led to a threefold increase in the frequency of chromatid breaks compared with that in control cells (Sargent *et al.*, 1989).

PCB-28, PCB-52, and a synthetic mixture of PCBs similar to that present in air in Chicago, USA, at equimolar concentrations all caused a 30–40% reduction in telomerase activity in human skin HaCaT keratinocytes, but the effect on telomere length differed, with shortening effects caused by PCB-28, PCB-52, and the Chicago air mixture of about 10%, 40%, and 5%, respectively, compared with controls after 6 weeks of exposure (Senthilkumar *et al.*, 2011).

PCB-77 caused DNA-adduct formation in human hepatocarcinoma HepG2 cells and in dexamethasone-treated primary rat fetal hepatocytes. In human lymphocytes, PCB-77 induced structural chromosomal aberration, but no increase in the frequency of micronucleated cells and sister-chromatid exchange was observed (Sargent *et al.*, 1989; Dubois *et al.*, 1995; Belpaeme *et al.*, 1996b).

Long-term dietary exposure of female hepatectomized Sprague-Dawley rats to PCB-52 at 10 ppm for 7 months, or PCB-77 at 0.1 ppm for 1 year, did not enhance the frequency of structural or numerical chromosomal aberration in liver and bone-marrow cells (Meisner *et al.*, 1992). However, coexposure to PCB-52 and PCB-77 at the doses given above for 1 year increased the frequency of polyploidy and structural chromosome aberration in bone-marrow cells. Although the frequency of numerical and structural chromosomal aberration in primary hepatocytes remained unaffected after coexposure to PCB-52 and PCB-77 for 7 months, the liver became more susceptible to diethylnitrosamine-induced genotoxicity (Sargent *et al.*, 1992).

PCB-101, PCB-118, PCB-138

PCB-101, PCB-118, and PCB-138 were able to induce DNA strand breaks (comet assay) and micronucleus formation (except PCB-138) in fish fibroblast RTG-2 cells [usually not used for genotoxicity testing], in a single dose experiment. However, the time course of markers for oxidative stress (carboxy-dichlorofluorescein oxidation, intracellular GSH, lipid peroxidation, and superoxide dismutase activity) did not correspond with the observed genotoxicity (Marabini *et al.*, 2011).

PCB-126

PCB-126 did not increase the frequency of micronucleus formation in human hepatoma HepG2 cells, but did cause a significant, but not dose-dependent, increase in levels of the DNA repair protein XPA (Western blot), whereas XPC protein levels were unaffected (Wei *et al.*, 2009b).

Table 4.7 Genotoxicity profile of metabolites of PCB-3

Compound	Lowest effective dose (μM)						
	Gene mutation (thioguanine resistance)[a]	Micronucleus (clastogenic effect)[a]	Micronucleus (aneuploidy: chromosomal loss)[a]	SCE[b]	Polyploidy[b]	DNA damage (comet assay)[c]	ROS[c]
PCB-3	-	-	-	-	-	-	-
2-OH-PCB-3	-	-	50	-	-	-	-
3-OH-PCB-3	-	-	100	-	-	-	-
4-OH-PCB-3	-	75	75	-	-	-	-
3,4-dihydroxy-PCB-3	-	25	15	5	-	-	-
3,4-*ortho*-quinone	0.6	15	5	-	-	-	-
2,5-hydroquinone	-	5	2.5	-	7.5	10 (at 37°C, not 6°C, in HL-60 cells; not in Jurkat cells at 37°C)	5 (ROS increased in HL-60 cells at 37°C, not at 6°C; no effect on ROS in Jurkat cells)
2,5-*para*-quinone	0.5	1	2.5	-	-	5 (at 37°C or 6°C in HL-60 cells; at 37°C in Jurkat cells)	2.5 (ROS increased in HL-60 cells and in Jurkat cells)

[a] From Zettner *et al.* (2007)
[b] From Flor & Ludewig (2010)
[c] From Xie *et al.* (2010)
PCB, polychlorinated biphenyl; ROS, reactive oxygen species; SCE, sister-chromatid exchange
Adapted from Robertson & Ludewig (2011)

PCB-126 did not increase the frequency of mutation in fetuses of the transgenic Muta™Mouse on day 18 of gestation after a single maternal oral dose of 0.5 mg/kg bw on day 10 of gestation (Inomata *et al.*, 2009).

PCB-126 and PCB-153

The role of oxidative DNA damage in carcinogenesis caused by PCB-126, PCB-153, and a combination thereof, was investigated by measuring in treated animals the accumulation of a DNA adduct, namely 3-(2′-deoxy-β-D-erythro-pentafuranosyl)-pyrimido[1,2-α]-purin-10-one (M1dG) (the pyrimidopurinone of deoxyguanosine) (Dedon *et al.*, 1998), which can be formed by reaction of lipid-peroxidation derived malonedialdehyde or by oxidation of deoxyribose-derived DNA base propenal and deoxyguanosine. Accumulation of M1dG adducts was assessed in the liver of female C57BL/6J mice given a single dose and in Sprague-Dawley rats exposed for 1 year. A single dose of a mixture consisting of four dioxin-like compounds (including PCB-126), or a mixture consisting of four non-dioxin-like PCBs (PCB 118, 138, 153, 180), did not increase M1dG accumulation in the mouse liver. In female Sprague-Dawley rats exposed to PCB-126, PCB-153, or a combination of both for 1 year (see Section 3; NTP, 2006a, b, c), an increase in hepatic levels of M1dG was observed in rats treated with PCB-126, and in rats treated with a combination of PCB-126 + PCB-153. In female rats coexposed to PCB-126 + PCB-153, the observed levels of M1dG adducts correlated with the observed incidence of liver tumours (Jeong *et al.*, 2008).

PCB-153

PCB-153 induced structural chromosomal aberration in human lymphocytes (Sargent *et al.*, 1989) and a statistically significant dose-dependent increase in the frequency of micronucleus formation in human breast epithelial MCF-10A cells (Venkatesha *et al.*, 2008). PCB-153 also induced a significant and dose-dependent twofold increase in the frequency of micronucleation in human hepatocarcinoma HepG2 cells. Coexposure to PCB-153 and benzo[*a*]pyrene significantly and dose-dependently increased the frequency of micronucleus formation by 60%. When α-naphthoflavone (an inhibitor of CYP1A1) was added to cultures exposed to PCB-153 and PCB-153 + benzo[*a*]pyrene, the frequency of micronucleation decreased almost to control levels (Wei *et al.*, 2009a).

PCB-153 was able to induce DNA strand breaks and micronucleus formation in fish fibroblast RTG-2 cells (Marabini *et al.*, 2011; see above for comments).

Treatment of immortal human skin HaCaT keratinocytes with PCB-153 at a single concentration resulted in a decrease in telomerase activity (~20% after 1 week to ~40% after 7 weeks of exposure) and telomeres were shortened by about 40% (Senthilkumar *et al.*, 2012). Shortening of telomeres was also observed in normal human foreskin keratinocytes exposed to PCB-153 in culture, but the difference compared with the control cells was not statistically significant on any of the days analysed.

PCB-209

PCB-209 did not induce mutation at the thymidine kinase locus in mouse lymphoma L5178Y/$T^{+/-}$ cells, and did not cause an increase in micronucleus formation in bone-marrow cells of male and female Crl:CD1 mice given a single oral dose at 2000 mg/kg bw (Han *et al.*, 2009).

$MeSO_2$-PCB metabolites

$MeSO_2$-PCBs did not induce micronucleus formation in cultured human lymphocytes, but some, namely 3-$MeSO_2$-2,5,2′,4′,5′-pentaCB [3′-$MeSO_2$-PCB-101;3-$MeSO_2$-2,2′,4′,5,5′-pentaCB] and 4-$MeSO_2$-2,5,2′,3′,4′-pentaCB [4’-$MeSO_2$-PCB-87; 4-$MeSO_2$-2,2′,3′,4′,5- pentaCB], enhanced levels of sister-chromatid exchange (Nagayama *et al.*, 1995, 1999).

(c) Summary

Numerous cell-based test systems, and animal models, have been used to investigate the genotoxic potential of commercial PCB mixtures. However, only 13 individual congeners have been examined so far in studies of genotoxicity and related effects. Seven congeners (PCB-3, PCB-52, PCB-77, PCB-118, PCB-138, PCB-153, PCB-209) have been investigated in both cellular systems and animals. An additional four congeners (PCB-15, PCB-47, PCB-101, and PCB-155) were tested only in cellular systems, and two congeners (PCB-126 and PCB-180) have been tested only in cellular systems or animals, respectively.

Studies on induction of gene mutation in bacteria exposed to PCB mixtures, or to the few individual congeners tested, gave negative results. However, these data were of limited value for assessing this end-point because the doses applied were usually < 1000 µg/plate and/or where this was not the case, testing with an exogenous metabolic system was omitted. Studies with PCB-209 were not subject to the aforementioned limitations.

When high concentrations of commercial PCB mixtures were tested in *Saccharomyces cerevisiae*, genotoxicity was observed with Arochlor 1254, Aroclor 1221, and Aroclor 1260. In mammalian cells in vitro, Aroclor 1254 was reported to produce DNA adducts, unscheduled DNA synthesis, DNA strand breaks and, to some extent, chromosomal aberration. Although these end-points were negative when tested in rodents in vivo, Aroclor 1254 did increase chromosomal aberration and micronucleation in fish, and mutation frequency in the liver of transgenic Big Blue® mice. Aroclor 1254 induced cell transformation in cultured Syrian hamster embryo cells.

As for the individual congeners, the most comprehensive data on genetic effects were available for PCB-3 and its metabolites. PCB-3 did not induce gene mutation in bacteria at doses up to 1000 µg/plate in the presence or absence of an exogenous metabolic system, except for one study in strain TA1538 in the presence of rabbit liver microsomes (see Table 4.4). However, PCB-3 was reported to bind to DNA and to cause an increase in levels of DNA adducts in primary human hepatocytes.

The cell lines commonly used for mutagenicity testing (Chinese hamster lung fibroblast V79, Chinese hamster ovary fibroblast, and mouse lymphoma L5178Y cells) have no or only very limited biotransformation capability, a problem for test compounds that require metabolic activation. Using instead a series of synthetic PCB-3 metabolites in the V79 gene mutation assay, the *ortho* (3,4-) and *para* (2,5-) quinones were shown to efficiently induce mutation at the *Hprt* locus at non-cytotoxic concentrations, while none of the tested mono- or dihydroxylated metabolites or PCB-3 itself induced mutation (see Table 4.4). In addition, an increase in chromosomal and genomic mutation was observed for all tested PCB-3 metabolites at higher, cytotoxic concentrations. Also, the 2′,5′-dihydroxylated metabolites of PCB-3 and PCB-2, but not metabolites of PCB-1 or the 3′,4′-dihydroxylated PCB-3, induced polyploidy in V79 cells, indicating strict structure–activity requirements for this type of DNA damage. The 2′,5′-quinone of PCB-3 induced an increase in levels of ROS via a semiquinone radical at concentrations inducing micronucleation, suggesting redox cycling of the 2′,5′-quinone. PCB-3–2′,5′-quinone caused telomere shortening in cultured HaCaT cells exposed for 11 weeks, an effect that may have been caused by oxidative stress.

The mutagenic activity of PCB-3 was also tested in an assay in transgenic rats in vivo. In the liver of male rats exposed to PCB-3, the mutation frequency was significantly increased and the mutation spectrum changed from predominantly transitions in the controls to predominantly G:C → T:A transversions in the rats exposed to PCB-3. 4′-OH-PCB-3 caused a similar, but not statistically significant, increase

in mutation frequency and a minor shift in the mutation spectrum compared with rats in the control group. A sex-specific and organ-specific difference was noted, since the response was less pronounced in livers of female Big Blue® rats and lungs of males, in which the observed increases in mutation frequency were below the level of statistical significance.

The non-dioxin-like PCB-52 was not tested for gene mutation in bacteria and cultured mammalian cells. Data on chromosomal aberration in cultured mammalian cells were ambiguous, but also of limited value since PCB-52 was never tested in the presence of a metabolic activation system. There were, however, indications of DNA damage caused by PCB-52 metabolites in studies in vitro and in vivo in rats coexposed to PCB-52 and dioxin-like PCB-77 for 1 year. Negative outcomes in other studies of chromosomal aberration in vivo may be attributed to the low doses tested.

The dioxin-like PCB-77 increased the level of DNA adducts in cultured mammalian cells. The lack of data on mutagenicity testing of PCB-77 did not allow for an interpretation of these findings with regard to gene mutation. Data on structural/numerical chromosomal aberrations, including micronucleus formation, were inconclusive in vitro, and negative for chromosomal aberration in female rats after long-term dietary exposure.

The limited data available on PCB-126 suggested no genotoxic potential in vitro or in vivo. However, increased levels of DNA adduct (M_1dG) indicative of the formation of ROS and/or lipid peroxidation were seen in female rats exposed to PCB-126 and PCB-126 + PCB-153 for 1 year (Jeong *et al.*, 2008).

Non-dioxin like PCB-153 gave positive results when tested for micronucleus formation in two cultured mammalian cell lines and one fish cell line. Also, reduction in telomerase activity corresponding to shortened telomeres was reported in cultured human cells. [Since no in-vivo data were available, the significance of these in-vitro results could not be assessed by the Working Group.]

For the decachlorinated PCB-209, a series of standard assays for genotoxicity that followed internationally accepted testing guidelines for regulatory purposes were performed under good laboratory practice (GLP) conditions, and showed no mutagenic and/or genotoxic potential.

4.3 Biochemical and cellular effects

4.3.1 AhR binding and activation

(a) AhR activity

AhR is a cytosolic, ligand-activated transcription factor that mediates many toxic and carcinogenic effects in vertebrates. TCDD has extremely high affinity to the AhR and is the reference AhR agonist and toxicant. AhR-mediated toxic responses are consequences of deregulated physiological functions, and sustained (chronic) AhR activation by persistent "dioxin-like" compounds is the key process in dioxin-like toxicity (Bock & Köhle, 2006). Toxicological evaluation of dioxin-like-PCBs (DL-PCBs) is based on various end-points associated with activation of the AhR and AhR-mediated physiological and toxic responses (Haws *et al.*, 2006). The major advantages of this concept are that most (if not all) effects of dioxin-like compounds are mediated via AhR activation, and that various effects of TCDD reported in many in-vivo and in-vitro models associated with carcinogenesis and tumour promotion may be extrapolated for DL-PCBs (IARC, 2012).

Effects of AhR-mediated changes in gene expression include the control of xenobiotic-metabolizing enzymes, modulations in cell cycle progression and cell proliferation, suppression of apoptosis, and perturbation of various developmental signalling pathways involved in carcinogenic processes (Vezina *et al.*, 2004; Sartor *et al.*, 2009; Faust *et al.*, 2013). In addition, AhR interacts with other signalling and transcription pathways,

including estrogen, thyroid and retinoic acid receptors, mitogen-activated protein kinases (MAPKs), NF-κB, retinoblastoma protein, and hypoxia-inducible factor-1 α (Tian *et al.*, 2002; Beischlag *et al.*, 2004; Murphy *et al.*, 2007; Puga *et al.*, 2009). Several molecular mechanisms that are related to AhR and that may contribute to carcinogenesis have been proposed:

- Induction of CYP1 enzymes linked to toxicity and cancer initiation (DNA-adduct formation and oxidative DNA damage);
- Sustained AhR-dependent expression of genes directly or indirectly controlling the cell cycle, proliferation and apoptosis, and cross-talk between genes in the AhR and growth-regulatory pathways;
- AhR-mediated cytoskeletal remodelling, reduced cell–cell contacts, modulation of developmental/differentiation pathways, cell plasticity and invasiveness affecting tumour progression;
- Upregulation of proinflammatory genes (Gasiewicz *et al.*, 2008).

Correlations between the immunosuppressive effects of PCBs and activation of the AhR pathway have been also reported (see Section 4.3.4).

(b) Concepts of TEF and TEQ

The concept of toxic equivalency (TEQ) is based on a common mechanism of action (mediated through AhR activation) of persistent organic pollutants (including polyhalogenated dibenzo-*p*-dioxins, dibenzofurans and biphenyls). It uses relative effective potencies (REP) of individual compounds to activate the AhR, and AhR-dependent toxic or biological effects relative to the reference toxicant TCDD; toxic equivalency factors (TEFs) for individual compounds were established/extrapolated from the database of many in-vivo studies. Since the 1980s, the TEF concept has been developed and refined (Safe *et al.*, 1985; Safe, 1990; Ahlborg *et al.*, 1994; Van den Berg *et al.*, 1998). Current TEF values were reevaluated recently using a refined TEF database (Haws *et al.*, 2006; Van den Berg *et al.*, 2006).

TEQ is defined by the sum of concentrations of dioxin-like compounds multiplied by their TEF values. A limitation of the concept is the additivity model being used, but its major advantage is the transformation of data on chemical concentration of complex mixtures into a single TCDD-like activity of the mixture. Many experimental studies with complex mixtures have confirmed that the TEQ approach is consistent with an additive model, although some deviations from additivity are observed. Another disadvantage is that the potential toxic and carcinogenic effects of non dioxin-like-PCBs (NDL-PCBs) are not included in this concept; high levels of NDL-PCBs may even suppress AhR-mediated toxicity, and thus act as antagonists.

Importantly, studies of carcinogenic and tumour-promoting activity were accounted for in the refined TEF database. Based on the TEF approach, carcinogenic hazard in humans may only be identified for DL-PCBs. The current TEF values for the PCB congeners included in the TEF concept are presented in Section 1, Table 1.4.

(c) Validation in experimental systems

AhR activation by DL-PCBs has been reported in many studies in vitro and in vivo, including comparative toxicogenomic analyses in primary human, monkey, and rodent hepatocytes (Silkworth *et al.*, 2005; Westerink *et al.* 2008). In a comparative in-vitro study in primary cultures of human and rat hepatocytes exposed to TCDD or PCB-126 at various concentrations for 48 hours, dose–responses and relative effective potencies (REP-values) were calculated for induction of CYP1A1 and other AhR-responsive genes (Carlson *et al.*, 2009). Previously, Silkworth *et al.* (2005) found that human cells are about 10–1000 times less sensitive to TCDD, PCB-126, and Aroclor 1254 than are rat and monkey cells.

Importantly, the newly calculated rat–human interspecies relative potency factors for PCB-126 were more than 100 times lower than the current rodent-derived value (Silkworth *et al.*, 2005).

These and other studies showed a relative insensitivity of the human AhR and human cells to PCB-126. In addition to a lesser potency of TCDD in human models (Haws *et al.*, 2006), lower potencies of PCB-126 might be due to species differences in relative intrinsic efficacy and/or species-specific differences in recruitment of transcriptional co-activators (Carlson *et al.*, 2009). In spite of the discrepancies between relative potencies of PCB-126 and TCDD in rodent and human liver cells, REP estimates based on induction of CYP1A1 or other AhR target genes might be relevant to evaluate the carcinogenic and hepatotoxic potential of TCDD and PCB-126 in humans.

The TEF approach and additivity concept were evaluated in 2-year cancer bioassays in groups of 53–55 female Harlan Sprague-Dawley rats receiving TCDD at a dose of 3–100 ng/kg bw per day, PCB-126 at a dose of 30–1000 ng/kg bw per day, 2,3,4,7,8-pentachlorodibenzofuran (PeCDF) at a dose of 6–200 ng/kg bw per day, or a mixture of the three toxicants. Dose–response curves for hepatic, pulmonary, and oral mucosal neoplasms showed that carcinogenic effects could be predicted from the WHO TEF values (Walker *et al.*, 2005).

In a short-term study, female Harlan Sprague-Dawley rats were exposed for 13 weeks to toxicologically equivalent doses of four polychlorinated aromatic hydrocarbons based on their TEF: TCDD (100 ng/kg bw per day), PeCDF (200 ng/kg bw per day), PCB-126 (1000 ng/kg bw per day), or PCB-153 (1000 µg/kg bw per day) (Vezina *et al.*, 2004). The AhR agonists (TCDD, PeCDF, and PCB126) produced very similar global gene-expression profiles, while PCB-153 showed a different, non-AhR-mediated response. All four compounds induced significant liver hypertrophy. TCDD and PCB-126 were more effective in activating AhR-dependent gene expression and inducing hepatic hypertrophy than was PeCDF, although the administered doses of each compound were based on equal TEQ values. These data fitted perfectly with the TEF value for PCB-126 in rats. Nevertheless, the gene-expression data might not bear a direct relevance to carcinogenicity of the studied compounds (Vezina *et al.*, 2004).

Global gene expression was investigated in vitro in the contact-inhibited rat liver progenitor WB-F344 cells exposed to PCB-126 at a concentration of 100 nM, or TCDD at 1 nM, for 6, 24, and 72 hours (Faust *et al.*, 2013). AhR dependency was validated using both chemical inhibition of AhR and knockdown of the AhR or the aryl hydrocarbon receptor nuclear translocator (ARNT) using small interfering RNA (siRNA). Gene ontology analysis revealed that, apart from deregulation of drug and lipid metabolism, genes participating in regulation of the cell cycle and growth control, developmental and cancer pathways, cell–cell communication and adhesion were significantly affected. Importantly, transcriptional regulation mediated by PCB-126 was very similar to that induced by TCDD in rat liver in vivo (Vezina *et al.*, 2004), and in rat liver progenitor WB-F344 cells. [Nevertheless, the relevance of these data to human carcinogenesis remained limited due to the species-specific pattern of AhR-dependent gene expression (Dere *et al.*, 2011).]

4.3.2 Cell death and proliferation

(a) Apoptosis, cell proliferation, and cell cycle control

(i) Apoptosis

DL-PCBs and NDL-PCBs have been shown to suppress DNA damage-induced apoptosis in vitro (Knerr & Schrenk, 2006; Al-Anati *et al.*, 2010).

PCB-28, PCB-101, and PCB-187 inhibited ultraviolet irradiation-induced apoptosis in hepatocytes from male Wistar rats pre-exposed to ultraviolet radiation before being treated with PCBs for 12 hours. A statistically significant suppression of apoptosis was found after the treatment with PCB-28 at 1 nM, PCB-101 at 10 nM, or PCB-187 at 1 μM (Bohnenberger *et al.*, 2001; Schrenk *et al.*, 2004).

PCB-126, and several NDL-PCBs (concentration range, 0.01–10 μM), attenuated the TP53-mediated apoptotic response via phosphorylation of the regulatory protein MDM2 in human hepatoma HepG2 cells (Al-Anati *et al.*, 2009). PCB-28, PCB-101, and PCB-153 reduced benzo[*a*]pyrene-induced phosphorylation of MDM2, and amplified the benzo[*a*]pyrene-induced TP53-dependent apoptotic response; however, benzo[*a*]pyrene-induced apoptosis was inhibited. Reduced levels of phosphorylated forkhead family transcription factor FOXO3a [FOXO3] were also reported after treatment with NDL-PCBs (Al-Anati *et al.*, 2010). FOXO3a probably functions as a trigger for apoptosis through expression of genes necessary for cell death. Thus NDL-PCBs may also inhibit benzo[*a*]pyrene-induced apoptosis by preventing phosphorylation of FOXO3a (Al-Anati *et al.*, 2010).

(ii) Cell proliferation

Cell proliferation can be caused either by cytotoxicity/injury and regenerative proliferation, or by a sustained increase in proliferation. It is mediated via several signal-transduction pathways leading to pro-proliferative changes in gene expression (controlled by specific transcription factors, such as AhR, CAR, NF-κB or AP-1). These events may drive genotoxic and nongenotoxic processes associated with tumour promotion and progression. PCBs have been reported to induce such proliferative events in a series of experimental in-vitro and in-vivo models (Tharappel *et al.*, 2002; Marlowe & Puga, 2005; Puga *et al.*, 2009).

CAR is known to control the hepatic expression of detoxification enzymes and to induce sustained cell proliferation in the liver. *Ortho*-substituted PCBs induce expression of CYP isoenzymes (see Section 4.1.3) via CAR (Muangmoonchai *et al.*, 2001). The activation of CAR-dependent gene expression by NDL-PCBs in vivo has been observed, e.g. in rat liver after 28-day exposure to PCB-180 (Roos *et al.*, 2011), or in the liver of immature, ovariectomized C57BL/6 mice treated with PCB-153 (Kopec *et al.*, 2010). Using a range of genetically engineered human cell models derived from liver, lung, and colon tissues, it has been shown that several NDL-PCBs, such as PCB-99, PCB-138, PCB-153, PCB-180 or PCB-194, may activate CAR-controlled reporter vectors, as well as PXR reporters, in a tissue-specific manner (Al-Salman and Plant, 2012). [The Working Group was aware that the relevance to human risk of CAR-driven hepatocarcinogenic effects seen in rodents has been questioned (Holsapple *et al.*, 2006).]

In the 13-week study by Vezina *et al.* (2004), modulation of global gene expression was analysed in liver of female rats given PCB-153 at a dose of 1000 μg/kg bw per day. In addition to CYP2B1 and CYP2B2, PCB-153 also modulated the expression of anti-apoptotic genes (*Bcl2* and *Wee1* were downregulated), and other genes associated with liver injury. PCB-153 selectively enhanced expression of the cAMP response element modulator (CREM), which is a signature response to liver regeneration after hepatocyte injury.

In an initiation–promotion study in female Sprague-Dawley rats, an increase in the frequency of several preneoplastic foci, and increased NF-κB and AP-1 binding activities were observed in the liver of rats given PCBs (Tharappel *et al.*, 2002). Although cell proliferation was not affected by PCB-153, apoptotic indexes were decreased in focal hepatocytes by PCB-153. The induction of altered hepatic foci appeared to be related to compensatory cell proliferation in rats treated

with PCB-77, while inhibition of apoptosis appeared to be important for rats treated with PCB-153 (Tharappel *et al.*, 2002). In a subsequent study, a single dose of PCB-153 (at 150 or 300 µmol/kg bw), but not PCB-77, induced hepatocyte proliferation and hepatic NF-κB activation in male Sprague Dawley rats (Lu *et al.*, 2003). Comparison of the effects of PCB-153 in wild-type mice and in mice deficient in the NF-κB p50 subunit suggested possible involvement of NF-κB in PCB-153-modulated cell proliferation and apoptotic changes (Lu *et al.*, 2004). Absence of the NF-kB p50 subunit inhibited the promoting activity of PCB-153, as illustrated by the NF-κB knockout study in mice treated with diethylnitrosamine/PCB-153. Taken together these data implicate a possible role for oxidative stress-mediated activation of specific transcription factors, such as NF-κB, as a possible mode of action for NDL-PCBs (Glauert *et al.*, 2008).

Brown *et al.* (2007) have reported a correlation between incidence of tumours of the liver and increased activity of mixed function oxidases and increased expression of proliferating cell nuclear antigen (the indicator of cell proliferation) in Sprague-Dawley rats exposed to repeated doses of Aroclor mixtures for 24 months. [From these data, it was not clear to which class of PCB congeners (DL- or NDL-PCBs) the effects could be attributed.]

In nontumorigenic human mammary epithelial MCF-10A cells, PCB-153 at a concentration of 1–15 µM, Aroclor 1254 and 2-(4-chlorophenyl) benzo-1,4-quinone increased levels of reactive oxygen species, and caused cell-cycle delay and growth inhibition by suppressing levels of cyclin D1 (Venkatesha *et al.*, 2008, 2010; Chaudhuri *et al.*, 2010).

Further studies also examined the role of AhR in PCB-mediated deregulation of cell proliferation. Activation of AhR is known to cause a delay in cell-cycle progression in several cancer cell lines, models of differentiated cells (e.g. rodent hepatoma cells), and in primary rodent hepatocytes (Elferink, 2003; Marlowe & Puga, 2005). However, AhR ligands have been found to elicit opposite effects in liver progenitor cells: induction of cell proliferation in contact-inhibited rat liver progenitor cells in vitro by DL-PCBs was reported to be an AhR-dependent process (Vondráček *et al.*, 2005). Like TCDD, PCB-126 at 100 pM, 4′-OH-PCB-79 (a metabolite of coplanar PCB-77) at 1 µM, or PCB-105 (mono-*ortho*-chlorinated congener) at 10 µM increased the percentage of cells in S-phase and the total number of cells. In contrast, the NDL-PCBs and their metabolites had no effect on cell proliferation at concentrations up to 10 µM. Only PCB-126 (AhR-activating), and not PCB-153 (not AhR-activating), upregulated levels of cyclin A and D2 protein (Vondráček *et al.*, 2005). The proliferative effects of PCB-126 were further potentiated by tumour necrosis factor-α (Umannová *et al.*, 2007).

(iii) DNA synthesis

The rate of DNA synthesis in altered hepatic foci and in tumours in PCB-treated rats and mice was studied by Tharappel *et al.* (2002), who gave rats DEN at a dietary concentration of 150 mg/kg followed by four biweekly intraperitoneal injections of PCB-77 or PCB-153 at a dose of 100 or 300 µmol/kg bw. Rats were given bromodeoxyuridine (BrdU) in Alzet osmotic pumps for the measurement of DNA synthesis in focal and nonfocal hepatocytes. PCB-77 increased the BrdU labelling indexes in GSTP-positive foci and in normal hepatocytes, but PCB-153 did not. Similarly, PCB-153 did not influence the BrdU labelling index in DEN-initiated hepatic tumours in mice (Glauert *et al.*, 2008). Haag-Grönlund *et al.* (2000) found that weekly subcutaneous injections of PCB-118 at doses of 10–10 000 µg/kg bw did not increase BrdU labelling in focal hepatocytes after 20 weeks, but that PCB-118 at a dose of 10 000 µg/kg bw increased the BrdU labelling index after 52 weeks.

(b) Cell–cell communication

Several studies have demonstrated that PCBs can inhibit gap-junctional intercellular communication (GJIC) both in vivo (Krutovskikh *et al.*, 1995; Bager *et al.*, 1997) and in vitro in rat liver epithelial cells, mouse and rat hepatocytes, human keratinocytes, and normal human breast epithelial cells (Ruch & Klaunig, 1986; Swierenga *et al.*, 1990; Hemming *et al.*, 1991; Kang *et al.*, 1996). The *ortho*-substituted PCBs were potent inhibitors of GJIC at low micromolar concentrations, while the coplanar PCBs did not inhibit GJIC after a single dose (Machala *et al.*, 2003). The assay for GJIC inhibition showed good predictability for tumour promotion of *ortho*-substituted PCBs. Recently, inhibition of GJIC has been confirmed using single doses of ultrapure NDL-PCB congeners (Hamers *et al.*, 2011).

Different cell- and connexin-specific mechanisms of action probably account for the inhibitory effects of PCBs on GJIC. Of the NDL-PCBs, PCB-153 decreased the number of gap-junction plaques, and decreased levels of connexin 43 (constitutive protein of gap junctions) in liver epithelial cells. PCB-153 enhanced proteasomal and lysosomal degradation of connexin 43 and inhibited trafficking of connexin 43 to the plasma membrane (Šimečková *et al.*, 2009a). In contrast, inhibition of GJIC by AhR ligands (i.e. DL-PCBs such as PCB-126) seems to proceed mainly through downregulation of mRNA of connexin 32 in hepatocyte-derived models (Herrmann *et al.*, 2002).

(c) Other cellular mechanisms relevant to PCB-induced carcinogenesis

NDL-PCBs have been shown to elicit additional nongenomic effects on membrane-associated proteins, which are closely related to tumour promotion and progression.

PCB-153 was found to increase the incidence of glutamine synthetase-positive tumours of the liver in male B6129sf2/J mice, and almost 90% (34 out of 38) of all tumours from mice treated with PCB-153 contained mutations in the β-catenin gene (*Catnb*), compared with ~45% (17 out of 37) of tumours in the control group. Tumours containing mutations of Ha-*ras* [*Hras*] and B-*raf* [*Braf*] were rare and not significantly different between treatment groups. Exposure to PCB-153 appeared to strongly select for *Catnb*-mutated, glutamine synthetase-positive tumours of the liver in mice (Strathmann *et al.*, 2006).

In the rat liver progenitor WB-F344 cell line, PCB-153 was found to decrease levels of several proteins at adherens junctions involved in cell–cell communication and intracellular signalling, including E-cadherin, β-catenin, and plakoglobin (Šimečková *et al.*, 2009b). Such mechanisms may be involved in the effects of NDL-PCBs, contributing to promotion of tumours.

Oral administration of dioxin-like PCB-126, mono-*ortho*-substituted PCB-118, and non-dioxin-like PCB-153 differentially altered expression of the tight junction proteins claudin 5, occludin, and ZO-1 in brain capillaries in C57/B16 mice. These alterations were associated with increased permeability of the blood–brain barrier. Most importantly, exposure to individual PCB congeners enhanced the rate of formation and progression of brain metastases by luciferase-tagged melanoma cells (Seelbach *et al.*, 2010).

As vascular endothelial cells create a selective barrier to the passage of cancer cells, it is of interest to note that non-dioxin-like PCB-104 induced endothelial hyperpermeability of human microvascular endothelial cells HMEC-1 and trans-endothelial migration of human breast cancer cells MDA-MB-231; these effects were associated with overexpression of vascular endothelial growth factor (Eum *et al.*, 2004).

Structurally different PCBs may induce proinflammatory mediators, which further contribute to metastasis. PCB-77, PCB-104 and PCB-153 induced expression of intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), and monocyte

chemoattractant protein-1 (MCP-1) in the liver, lung, and brain of male C57Bl/6 mice. PCB-77 and PCB-104 also increased levels of matrix metalloproteinase-7 (MMP-7) mRNA in the liver and brain (Sipka *et al.*, 2008).

The mixture of seven NDL-PCBs (PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, PCB-180, and PCB-209) increased cell motility of human non-metastatic MCF-7 cells and human metastatic breast cancer MDA-MB-231 cells in vitro via production of reactive oxygen species, and activation of the Rho-associated kinase (ROCK). In a follow-up study in vivo, the PCB mixture enhanced the capability of metastatic breast cancer cells to metastasize to bone, lung, and liver (Liu *et al.*, 2010).

To explore the possible effects of PCBs on telomeres and telomerase, human skin keratinocytes were exposed to a synthetic mixture of volatile PCBs, or the prominent airborne PCB congeners, PCB-28 or PCB-52, for up to 48 days (see also Section 4.2.2b). The PCB mixture and the two congeners significantly inhibited telomerase activity from day 18, while telomere length was reduced by PCB-52 from day 18, and by PCB-28 and by the mixture from day 30 onwards (Senthilkumar *et al.*, 2011).

New bioanalytical tools (e.g. transcriptomics) applied in human, animal, and in-vitro studies might improve the ability to predict the potential carcinogenicity of chemicals by elucidation of similar mechanisms (Guyton *et al.*, 2009). Several analyses of global gene expression in rodent models included identification of the effects of DL-PCBs, especially PCB-126, on pathways related to carcinogenicity.

4.3.3 Endocrine disruption

Extensive data indicate an association between exposure to PCBs and endocrine disruption. The effects include primarily interference with the function of sex hormones, i.e. estrogens and androgens, and their receptors (reviewed by Bonefeld-Jørgensen, 2010; Bonefeld-Jørgensen *et al.*, 2011; Crinnion, 2011; Fucic *et al.*, 2012). In addition, PCBs are able to bind to thyroxine transport protein (TTR), human thyroxine-binding globulin, and thyroid-hormone receptors (reviewed by Cheek *et al.*, 1999; Kawano *et al.*, 2005; Grimm *et al.*, 2013); disruption of the thyroid-hormone system was observed up to 30 years after exposure (Masuda, 2001). Furthermore, PCBs affect hormone-metabolizing enzymes, e.g. of the CYP1, CYP2, CYP3A subfamilies, and uridine-diphosphate-glucuronyl transferase, iodothyronine deiodinase, and sulfotransferase (Brouwer *et al.*, 1998).

OH-PCB and PCB-catechol and PCB-quinone metabolites formed by CYP and other oxidative enzymes have been implicated as direct or indirect endocrine-disrupting agents. The interactions found depended upon the position of hydroxylation, as well as the proximity of chlorine substitutents and the substitution pattern. Some OH-PCBs are retained in blood because they bind to transthyretin (TTR) (Lans *et al.*, 1993). Several OH-PCBs, PCB-catechols and PCB-quinones interact with estrogen receptors and other cellular receptors as agonists or antagonists (Garner *et al.*, 1999). Other OH-PCBs inhibit human estrogen sulfotransferase, thyroid hormone sulfotransferase and phenol sulfotransferases, with inhibitory potencies (IC_{50}) ranging from less than nM to low μM (Schuur *et al.*, 1998a). Species differences in the protein structures of these sulfotransferases are such that there are differences in potency of inhibition of the corresponding sulfotransferases from other species such as fish (Wang & James, 2007). The human sulfotransferase enzymes are more potently inhibited by OH-PCB than those of other species (see details below).

(a) Humans

(i) Effects on sex hormones and their receptors

Serum samples were collected from male residents of an area in eastern Slovakia with extensive environmental contamination from a former PCB-production site, as well as from a neighbouring non-industrial region. The highest quartile of PCB concentrations was significantly associated with reduced estrogen receptor-mediated activity, and a negative correlation was observed between total estrogenic activity and dioxin-like activity. No correlation was found between E_2 [17beta-estradiol] concentrations and total PCB concentrations (R_s = 0.078). E_2 was largely responsible for the estrogenic activity identified in total serum extracts (Plísková *et al.*, 2005).

PCB-induced endocrine dysfunction related to the hypothalamic–pituitary–gonadal axis was evaluated in a birth-cohort study in Germany, initiated in 2000. Healthy mother–infant pairs were recruited in the industrialized city of Duisburg. Dioxins, DL-PCBs, and six indicator PCBs (PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, PCB-180) were measured in maternal blood during pregnancy and in breast milk. Concentrations of testosterone and estradiol were measured in maternal and cord serum of 104 mother–infant pairs. Linear-regression analysis was used to describe the association of PCBs in maternal blood or milk with the serum concentrations of the sex steroids, after adjustment for confounding. Median concentrations for the sum of indicator PCBs were 149 ng/g in maternal blood fat and 177 ng/g in milk fat. Typically, reduction in testosterone concentrations was more pronounced in the cord serum of female babies. In contrast, male babies showed a stronger reduction in estradiol concentrations. The only statistically significant reduction associated with the six indicator PCBs was for testosterone in girls (means ratio, 0.76; 95% CI, 0.61–0.96) (Cao *et al.*, 2008).

Serum concentrations of testosterone in relation to concentrations of PCBs were investigated in an adult Native American (Mohawk) population. Fasting serum samples were collected from 257 men and 436 women, and analysed for the presence of 101 PCB congeners, and for testosterone, cholesterol, and triglycerides. The associations between testosterone and tertiles of PCB concentrations in serum (both adjusted for wet weight and lipid) were assessed by use of a logistic regression model, controlled for age, body mass index (BMI), and other factors. The lowest tertile was taken as the reference level. Testosterone concentrations in men were inversely correlated with total PCB concentration in serum, and with concentrations of the congeners PCB-74, PCB-99, PCB-153, and PCB-206, but not PCB-52, PCB-105, PCB-118, PCB-138, PCB-170, PCB-180, PCB-201, or PCB-203. Testosterone concentrations in women were much lower than in men, and not significantly correlated with serum concentrations of PCBs (Goncharov *et al.*, 2009).

A possible correlation between exposure to PCBs and testosterone concentrations was studied in 834 men from Eastern Slovakia (age, 21–78 years; median age, 48 years), of whom 432 were from a highly polluted area, and 402 were from an area with background pollution. Serum concentrations of 15 PCB congeners were measured by gas chromatography/mass spectrometry, and total testosterone was determined immunochemically (electrochemiluminescence). Correlation coefficients for each PCB congener and for the total of 15 PCBs (Σ15PCBs) with testosterone were determined. The full cohort of 834 men (median concentration of Σ15PCBs, 885 ng/g lipid) showed a highly statistically significant negative correlation between testosterone concentration and age (r = 0.303; $P < 0.0001$). A significant negative correlation ($P < 0.05$) with testosterone concentration was seen only for two mono-*ortho*-congeners, i.e. PCB-105 and PCB-118. No significant correlations were found in the subcohort of 444 men

in a narrower age range (41–55 years), in which there was no effect of age on testosterone concentrations (Langer *et al.*, 2010).

A follow-up study by the same authors included 429 men (age, 41–55 years) from a highly polluted area in Eastern Slovakia. For all subjects, the serum concentrations of 15 PCB congeners and several other chemicals were measured by gas chromatography/mass spectrometry, and total testosterone in serum was determined by electrochemiluminescence immunoassay. Similarly to the previous analysis, there was no statistically significant correlation between Σ15PCBs and testosterone (Langer *et al.*, 2012).

The association of PCBs with sex-hormone concentrations in serum was assessed in 341 men from an infertility clinic in the USA, whose exposure levels to PCBs were comparable to those observed in the general population. In crude regression models, inverse correlations were found between serum concentrations of PCBs and steroid hormone-binding globulin (SHBG) and total and free testosterone. However, after adjustment for lipids, age, and body-mass index, nearly all the significant associations disappeared: an inverse correlation remained between PCB-118 and SHBG ($P < 0.01$), while those between DL-PCBs and SHBG and total testosterone, and between PCB-118 and total testosterone, were suggestive but not statistically significant (Ferguson *et al.*, 2012).

A few studies explored the relationship between levels of steroid hormones in consumers of contaminated fatty fish from the Great Lakes (Persky *et al.*, 2001; Turyk *et al.*, 2006; see below).

(ii) Effects on the thyroid-hormone system

In a study of more than 600 children in Germany, blood samples collected from 320 children showed a significant positive correlation between serum concentrations of PCBs and increased levels of thyroid-stimulating hormone (TSH), and a significant inverse correlation with free total thyroxine (T4), as was to be expected when TSH increases (Osius *et al.*, 1999).

Hagmar *et al.* (2001a) studied the relationship between the amounts of various organohalogenic compounds in fatty fish from the Baltic Sea and hormone levels in adult men who consumed these fish. Plasma samples from 110 men (43 from south-eastern Sweden, 67 from Latvia; age range, 23–79 years) who consumed up to 32 fish-meals per month were analysed for the presence of 18 PCB congeners, five OH-PCBs, and various other chemicals. In addition, plasma concentrations of follicle-stimulating hormone, luteinizing hormone, prolactin, plasma thyrotropin, free and total triiodothyronine (T3), free and total T4, and free testosterone were measured. After adjustment for age, no significant associations were found between any of these markers and any of the PCBs or OH-PCBs. However, a study among 182 fishermen's wives (age range, 23–46 years) from the east coast of Sweden, who had a median consumption of contaminated fatty fish from the Baltic Sea of two meals per month (range, 0–12 meals), found a significant inverse correlation between PCB-153 concentrations (range, 16–776 ng/g lipid) and total T3 levels in plasma, also after adjustment for age ($P < 0.001$) (Hagmar *et al.*, 2001b). An inverse correlation was also observed with total T4, which was borderline significant ($P = 0.07$).

Parallel to a larger investigation of consumption of contaminated fatty fish from the Great Lakes and effects on reproductive function, the association between PCB intake via consumption of fish and effects on thyroid and steroid hormones was studied in 178 men, and on thyroid hormones in 51 women (Persky *et al.*, 2001). Serum concentrations of PCBs and fish consumption were associated with significantly lower levels of T4 and a significantly lower free T4 index in women. Fish consumption, but not serum PCB concentration, was associated with a higher uptake of T3 in men. Results for TSH were inconsistent. Among men, there were significant

inverse associations for serum PCB concentration and fish consumption with SHBG-bound testosterone, but no association with SHBG itself, or with free testosterone. There were no significant overall associations for serum PCB concentration or fish consumption with estrone sulfate, follicle-stimulating hormone, luteinizing hormone, or dehydroepiandrosterone sulfate.

The relationsip between levels of steroid and thyroid hormones and total NDL-PCBs was investigated in 56 men who were frequent or infrequent consumers of fish from the Great Lakes (Turyk *et al.*, 2006). The men had consumed fish meals for 15–57 years. Significant inverse associations with serum PCB concentrations were found for T3, T4, TSH, and SHBG-bound testosterone, after adjustment for age, body-mass index, and use of medication. Follicle-stimulating hormone, luteinizing hormone, free testosterone, and SHBG were not associated with PCB concentrations in serum.

To assess the relationship between exposure to organochlorine compounds and thyroid function and neurodevelopment, a population-based birth-cohort study was conducted on the Faroe Islands (Denmark), where the regular consumption of PCB-contaminated fish is an important source of exposure (see Section 1.4.1). The study included 182 newborns who were followed up until age 54 months. PCB levels (calculated as the sum of congeners PCB-138, PCB-153, and PCB-180, multiplied by two) were measured in breast milk and maternal serum, and maternal blood and cord blood were collected for measurement of thyroid parameters. After covariate adjustments, consistent inverse and monotonic associations were observed between total PCB exposure and the resin T3 uptake ratio, a proxy measure of the binding capacity of T4-binding globulin sites that are not saturated with T4. The resin T3 uptake ratio is high in hyperthyroidism and low in hypothyroidism. No associations with other thyroid parameters (TSH, free T3, free T4) were observed (Julvez *et al.*, 2011).

In a study in 39 healthy pregnant women in the metropolitan area of Tokyo, Japan, associations were studied between in-utero exposure to PCBs or OH-PCBs and free T4 or TSH status in newborns. The concentration of total OH-PCBs and of OH-metabolites of PCB-187 in umbilical cord tissue was significantly correlated with higher levels of free T4 in heel-prick blood samples obtained from neonates aged 4–6 days. On the other hand, the concentration of total PCBs and of the congeners PCB-118, PCB-138, PCB-153, and PCB-180 showed no relationship with free T4 and TSH levels (Otake *et al.*, 2007).

In a study in 232 healthy mother–infant pairs recruited between 2000 and 2002 in the industrialized city of Duisburg, Germany, TSH, total T4, T3, free T4 and free T3 were measured in serum of the pregnant women and in cord serum (Wilhelm *et al.*, 2008). Blood levels (n = 182) of WHO 2005 TEQ (which includes PCDD/PCDF + PCBs) were in the range of 3.8–58.4 pg/g lipid (median, 19.3 pg/g lipid). The corresponding value for human milk (n = 149) was 2.6–52.4 pg/g lipid (median, 19.7 pg/g lipid). Multiple regression analyses did not detect any effects on thyroid hormones related to WHO 2005 TEQs in blood or milk of mothers and their newborns.

In a study among Inuit women and their infants, a positive correlation was found between concentrations of OH-PCBs and total T3 in plasma of 120 women at delivery ($\beta = 0.57$; $P = 0.02$). In umbilical cord plasma of 95 newborns, PCB-153 concentrations were negatively correlated with T4-binding globulin concentrations ($\beta = -0.26$; $P = 0.01$). No associations were observed between organochlorine contaminants and thyroid hormones in blood plasma collected from infants aged 7 months (Dallaire *et al.*, 2009).

(b) Experimental systems

(i) Effects on sex hormones and their receptors

Experimental animals in vivo

Groups of pregnant Wistar WU rats received a daily oral dose of 4-OH-2,3,3′,4′,5-pentachlorobiphenyl [4-OH-PCB-109] at 0.5 or 5.0 mg/kg bw, or Aroclor 1254 at 25 mg/kg bw, on days 10–16 of gestation. The diestrous stage of the estrous cycle was significantly prolonged in 75% and 82% of female offspring exposed to 4-OH-PCB-109 at the lower and higher dose, respectively, compared with 64% of Aroclor-exposed offspring. This effect resembled a state of pseudopregnancy. Plasma estradiol concentrations in female offspring were significantly increased (50%) in the proestrous stage after exposure to 4-OH-PCB-109 at the higher dose, while no effects on estradiol were seen in rats treated with Aroclor 1254 (Meerts *et al.*, 2004).

In the offspring (age, 17 weeks) of Sprague-Dawley dams treated intragastrically with PCB-77 at a dose of 250 ng/kg bw on days 13–19 post-conception, the concentrations of follicle-stimulating hormone, luteinizing hormone, and testosterone were similar to those in the controls (Wakui *et al.*, 2012).

In-vitro assays

In an in-vitro estrogen-reporter assay with T47 human breast-cancer cells, the less chlorinated congeners (PCB-28, PCB-52, PCB-66, and PCB-74) were estrogenic, while the more highly chlorinated congeners (PCB-138, PCB-153, PCB-170, PCB-180, PCB-187, PCB-194, PCB-199, and PCB-203) acted as anti-estrogens. Co-planar PCBs had no effect on estrogen-receptor activation in this assay (Plísková *et al.*, 2005).

Less chlorinated, *ortho*-substituted, non-co-planar PCBs were weakly estrogenic in some in-vitro assays. Results in MCF-7 human breast-cancer cells were generally consistent with, but not absolute in, the requirement for *ortho*-chlorine substitution and *para*-hydroxylation for estrogenic potency (Gierthy *et al.*, 1997).

In MCF-7 human breast-cancer epithelial cells, three abundant PCBs, i.e. PCB-138, PCB-153 and PCB-180, showed pleiotropic effects on the estrogen and androgen receptors. Slightly increased cell proliferation was observed at low PCB concentrations (1–10 nM) in cells co-treated with E_2 at 0.01 nM, while the PCBs significantly inhibited cell growth at higher concentrations (1 and 10 μM). In a reporter assay (ERE-*tk*-CAT analysis), the three congeners induced a significant decrease of ER-E_2-mediated CAT activity. PCB-138 had a dose-dependent antagonistic effect on androgen-receptor activity in transiently co-transfected Chinese hamster ovary cells, with an IC_{50} of 6.2 μM. Thus the three PCBs compete with the binding of two natural hormone-receptor ligands (Bonefeld-Jørgensen *et al.*, 2001). In reporter-based assay with LNCaP human prostate-cancer cells, the congeners PCB-42, PCB-128, PCB-138 and the Aroclor mixtures 1242, 1248, 1254, and 1260, showed antagonizing effects on androgen-receptor activity (Portigal *et al.*, 2002).

The effects of PCB-77, PCB-118, PCB-126, and PCB-153 (at 0.01–20 μg/mL) on the human prostatic carcinoma cell-line LNCaP were investigated in vitro. PCB-77 and PCB-126 reduced androgen-dependent prostate-specific antigen (PSA) secretion and LNCaP cell proliferation, and inhibited 5-α-reductase activity. PCB-118 and PCB-153 had no effect on 5-α-reductase, but showed a biphasic effect on LNCaP cell proliferation, with low concentrations (0.1–1 μg/mL) causing an increase, and higher concentrations (10–20 μg/mL) a significant reduction. Likewise, PCB-118 and PCB-153 enhanced PSA secretion at low concentrations and reduced it at higher concentrations. Since induction of ethoxyresorufin-O-deethylase (EROD) and inhibition of 5-α-reductase activity were not observed, these PCBs act through an AhR- and androgen-receptor-independent mechanism. The anti-androgenic effects of the *meta*- and *para*-substituted PCB-77 and PCB-126 are more pronounced than

those of *ortho*-substituted PCB-118 and PCB-153 (Endo *et al.*, 2003).

The estrogenicity of binary mixtures of the OH-PCBs 2,4,6-trichloro-4′-biphenylol (4′-OH-PCB-30) and 2,3,4,5-tetrachloro-4′-biphenylol (4′-OH-PCB-61), was examined in the MCF-7 focus assay and a competitive estrogen-receptor binding assay. Although the individual OH-PCBs were estrogenic in both assays, there was no synergy when they were combined at various concentrations as equimolar mixtures (Arcaro *et al.*, 1998). Likewise, the estrogenic activities of these two OH-PCBs were additive when tested as equimolar mixture in several systems (MCF-7 cells, MDA-MB-231 human breast-cancer cells, mouse uterus) at high and low levels of estrogen-receptor expression, confirming the lack of a synergistic effect (Ramamoorthy *et al.*, 1997).

PCB-138, PCB-153, and PCB-180, as well as other non-*ortho*- and di-*ortho*-substituted PCBs, were shown to interfere with the function of the androgen and estrogen receptors in vitro (Schrader & Cooke, 2003; Hjelmborg *et al.*, 2006). Similarly, some OH-PCBs showed estrogenic and/or anti-estrogenic effects (Jansen *et al.*, 1993; Rasmussen *et al.*, 2003).

PCB-54 was chosen as a prototypical *ortho*-substituted PCB to test the hypothesis that *ortho* substitution in the absence of *para*- or *meta*-substituted chlorines may lead to enhanced estrogenic activity. The results indicated that PCB-54 is estrogenic both in vitro in the MCF-7 cell-focus test, and in vivo in the rat uterotropic assay (Arcaro *et al.*, 1999). The estrogenic activity of PCB-54 in MCF-7 cultures was inhibited by the estrogen-receptor antagonist LY156758. Competitive binding assays with recombinant human (rh) estrogen receptor indicated that PCB-54 does not bind to rhERalpha or rhERbeta, but the 4-hydroxylated metabolite of PCB-54 does. This metabolite was also 10-fold more estrogenic than PCB-54 in the MCF-7 focus assay, but was not detected in the medium of MCF-7 cultures exposed to PCB-54. These results suggested that the estrogenicity observed in the human breast-cancer cells and the rat uterus may be due to (i) binding of an undetected metabolite of PCB-54 to the estrogen receptor; (ii) direct binding of PCB-54 to a novel form of the estrogen receptor; or (iii) an unknown mechanism involving the estrogen receptor (Arcaro *et al.*, 1999).

Evidence that PCB-77 can act as an estrogen – with effects mediated by the estrogen receptor– was based on results from a variety of assays, including those assessing binding to the receptor in a competitive binding assay (where PCB-77 at 700-fold molar excess inhibited [^{3}H]-estradiol binding to the estrogen receptor by 50%); regulation of gene expression from a transfected exogenous (ERE-*tk*-CAT) or endogenous (*pS2*) estrogen-regulated gene; regulation of cell growth in the estrogen-dependent human breast-cancer cell lines MCF-7 and ZR-75–1; and activity in the immature mouse uterine-weight bioassay in vivo. These data demonstrated that PCB-77 mimics estrogenic action at concentrations in the nanomolar range (292 ng/L), which is comparable to concentrations of PCBs found in human tissues (Nesaretnam *et al.*, 1996).

The estrogenic effects of PCBs may be mediated in part by their hydroxylated metabolites. Both the parent compound and the OH-metabolite show low affinities for both the α- and β-isoform of the estrogen receptor, which suggests that they have only weak activity as estrogen-receptor agonist or antagonist. However, PCBs and OH-PCBs may be indirectly estrogenic by inhibiting human estrogen sulfotransferase (hEST). When 31 OH-PCBs were tested for their inhibitory effect on hEST, hydroxylation of one of the phenyl rings appeared to increase the inhibitory effect in the order *para*-OH > *meta*-OH > *ortho*-OH. Indeed, various environmentally relevant OH-PCBs (e.g. 4-OH-2,3,3′,4′,5-pentachlorobiphenyl, 4-OH-PCB-109; and 4,4′-dihydroxy-3,3′,5,5′-tetrachlorobiphenyl, 4,4′-$(OH)_2$PCB-80) are very potent inhibitors of hEST. Since sulfation by this enzyme is an

important pathway for E_2 inactivation, inhibition of this metabolic step would lead to increased bioavailability of estradiol. This would explain the indirect estrogenicity of hEST inhibitors (Kester *et al.*, 2000).

A series of twelve PCBs were investigated for their ability to bind to the uterine estrogen-receptor protein, by use of a competitive equilibrium-binding assay with enriched cytosol-receptor preparations (0–40% ammonium sulfate fraction) from uteri of ovariectomized mice. PCBs that showed strong affinities generally possessed either single or multiple *ortho*-chlorine substituents. For OH-metabolites, *ortho*-chlorine substitution on the phenolic ring seemed less effective than on the nonphenolic ring. Thus 4′-OH-2,4,6-trichlorobiphenyl (4′-OH-PCB-30), which has two *ortho* chlorines and a *para* substituent, had the strongest binding affinity. For PCBs without *ortho* chlorines, the binding activity decreased 10–100-fold. PCBs that demonstrated appreciable receptor-binding activity were also active in vivo in stimulating an increase in uterine weight, while weak binders were inactive in this respect. The *ortho*-chlorine substitution appears essential in determining receptor-binding activity, probably because of decreased conformational flexibility due to restricted rotation about the inter-ring bond (Korach *et al.*, 1988).

The effects of structure and substituent position on the estrogenic and anti-estrogenic activities of various OH-PCBs were investigated in a series of assays. The presence of an *ortho* or *meta* substitution in the phenolic ring had minimal effects on estrogenic activity, while the 2,4,6-trichloro- and 2,3,4,6-tetrachloro configuration in the non-phenolic ring were required for this response. Substitution in the phenolic ring had no effect on anti-estrogenic activity (Connor *et al.*, 1997).

In-vitro toxicity profiles were determined for 24 NDL-PCBs with respect to 10 different mechanisms of action. All NDL-PCBs antagonized androgen-receptor activation; none were androgenic. Less chlorinated NDL-PCBs (PCB-19, PCB-28, PCB-47, PCB-51, PCB-53, PCB-100, PCB-104, PCB-136) were weak estrogen-receptor agonists. More highly chlorinated NDL-PCBs (PCB-138, PCB-153, PCB-170, PCB-180, PCB-190) were weak estrogen-receptor antagonists; several inhibited estradiol-sulfotransferase activity by > 50% (PCB-28, PCB-47, PCB-51, PCB-53, PCB-100). On the basis of hierarchical analysis of the toxicity profiles, three separate clusters of NDL-PCBs and a fourth cluster of reference DL-PCBs could be distinguished. The indicators PCB-28, PCB-52, PCB-101, PCB-118, PCB-138, PCB-153, and PCB-180 contributed most to the anti-androgenic, anti-estrogenic, anti-thyroidal, tumour-promoting, and neurotoxic potencies calculated for PCB mixtures reported in human samples, while the most potent AhR-activating DL-PCB, PCB-126, contributed at most 0.2% to any of these calculated potencies. It was suggested that PCB-168 should be added to the list of indicator congeners, given its relatively high abundance and its anti-androgenic and TTR-binding properties (Hamers *et al.*, 2011).

(ii) Effects on the thyroid-hormone system

Experimental animals in vivo

Marmoset monkeys were treated with oral doses of PCB-77 at 0.1, 1, or 3 mg/kg bw, twice per week, for 18–23 weeks. Histological examination of the thyroid gland showed dose-dependent hyperplasia of follicular cells, which was associated with various changes in thyroid function. The average serum concentrations of T4 during the treatment period were reduced by 35% in monkeys at 0.1 mg/kg bw, 81% at 1 mg/kg bw, and > 99% at 3 mg/kg bw. A reduction in serum concentrations of T_4 was observed from 2 weeks and throughout the entire treatment period (18–23 weeks), and was reflected in a decrease in the free T4 index in the groups at 1 and 3 mg/kg bw. Serum T3 concentrations were reduced in the group at 3 mg/kg bw within 2

weeks. Concentrations of TSH were increased in the group at the highest dose as a feedback response to the strongly reduced serum T4 concentrations (van den Berg *et al.*, 1988).

Pregnant Wistar WU rats were given Aroclor 1254 as daily oral dose at 5 or 25 mg/kg bw on days 10–16 of gestation to determine effects on thyroid-hormone concentrations in plasma and brain, on peripheral thyroid-hormone concentrations, and on peripheral thyroid-hormone metabolism in fetal and weanling rats. Maternal exposure to Aroclor 1254 significantly reduced fetal (day 20 of gestation) and neonatal (postnatal day 4) plasma concentrations of total T4 and free T4. These effects were less pronounced in offspring at age 21 days and absent at 90 days. T3 concentrations in brain tissue in the exposed fetuses were significantly decreased relative to controls, but only in the group at the lower dose. On postnatal day 21, T4 concentrations had significantly decreased in the forebrain of female weanling rats from the group at the higher dose, but no reductions were seen in male or female neonates. The deiodination of T4 to T3 was significantly increased in fetal forebrain homogenates at both doses. No alterations in thyroid-hormone metabolism were seen in forebrain homogenates from adult offspring exposed pre- and postnatally to Aroclor 1254. Accumulation of the PCB metabolite 2,3,3′,4′,5-pentachloro-4-biphenylol [4-OH-PCB-109] was observed in fetal plasma and forebrain tissue on day 20 of gestation, and in neonatal and weanling plasma on postnatal days 4, 21, and 90 (Morse *et al.*, 1996).

In groups of Sprague-Dawley rats given two or five weekly intraperitoneal injections of PCB-126 (0.2 mg/kg bw) or PCB-114 (20 mg/kg bw), total T4 concentrations in serum were lower than those in the controls. The expression of TTR was significantly higher in the PCB-treated group than in the control group (Han *et al.*, 2010).

Reduced thyroid-hormone levels were found in serum of Sprague-Dawley rats treated with $MeSO_2$ metabolites of the following PCB congeners: 3-$MeSO_2$-2,2′,3′,4′,5,6-hexachlorobiphenyl [5′-$MeSO_2$-PCB-132]; 3-$MeSO_2$-2,2′,3′,4′,5,5′-hexachlorobiphenyl [3′-$MeSO_2$-PCB-141]; 3-$MeSO_2$-2,2′,4′,5,5′,6-hexachlorobiphenyl [5-$MeSO_2$-PCB-149] and 4-$MeSO_2$-2,2′,4′,5,5′,6-hexachlorobiphenyl [4-$MeSO_2$-PCB-149]. These $MeSO_2$-PCBs are found in human milk, liver, and adipose tissue. All four metabolites (20 µmol/kg bw, intraperitoneal injection, once per day, for 4 days) reduced the serum concentration of total T4 by 22–44%, on days 2, 3, 4 and 7 after the last dose. Concentrations of total T3 were reduced by 37% on day 7 after treatment with 4-$MeSO_2$-PCB-149. A 30% increase in thyroid weight was seen after treatment with 3′-$MeSO_2$-PCB-141. These data suggest that these 3- and 4-$MeSO_2$ metabolites act as endocrine disrupters, but probably through different mechanisms (Kato *et al.*, 1998). A similar study was conducted with the *meta*-$MeSO_2$ metabolites of tetra- and pentachlorinated biphenyls: 3-$MeSO_2$-2,2′,4′,5-tetraCB [3′-$MeSO_2$-PCB-49], 3-$MeSO_2$-2,3′,4′,5-tetraCB [3-$MeSO_2$-PCB-70], 3-$MeSO_2$-2,2′,3′,4′,5-pentaCB [3′-$MeSO_2$-PCB-87], 3-$MeSO_2$-2,2′,4′,5,5′-pentaCB [3′-$MeSO_2$-PCB-101], and the *para*-$MeSO_2$-metabolite 4-$MeSO_2$-2,2′,4′,5,5′-pentaCB [4′-$MeSO_2$-PCB-101]. The data showed that all five $MeSO_2$-PCBs influence thyroid-hormone metabolism (Kato *et al.*, 1999). A further study by this group demonstrated that the *meta*-$MeSO_2$ metabolites of PCB-49, PCB-70, PCB-87, PCB-101, PCB-132, PCB-141, PCB-149 [3′-$MeSO_2$-PCB-49, 3-$MeSO_2$-PCB-70, 3′-$MeSO_2$-PCB-87, 3′-$MeSO_2$-PCB-101, 5′-$MeSO_2$-PCB-132, 3′-$MeSO_2$-PCB-141, 5-$MeSO_2$-PCB-149] and the *para*-$MeSO_2$ metabolite of PCB-101 [4′-$MeSO_2$-PCB-101] induced hepatic microsomal UDP-glucuronosyl transferase (UDP-GT) in male Sprague-Dawley rats. The increase in hepatic glucuronidation of T4 after the administration of the eight test compounds was the probable cause of the reduced serum concentration of T4 (Kato *et al.*, 2000).

Thyroid hormone status and metabolism were studied in groups of pregnant Wistar WU rats given oral doses of 4-OH-2,3,3′,4′,5-pentachlorobiphenyl [4-OH-PCB-109] ([^{14}C]-labelled or unlabelled) at 5 mg/kg bw on days 10–16 of gestation. Fetuses were studied at days 17 and 20 of gestation. The test compound accumulated in the fetal compartment, with fetal/maternal ratios of 11.0, 2.6, and 1.2 in liver, cerebellum, and plasma, respectively, at day 20. Radiolabel was bound to plasma TTR in dams and fetuses. Fetal plasma concentrations of total T4 and free T4 were significantly decreased at days 17 and 20 of gestation (89% and 41%, respectively, at day 20), while fetal concentrations of TSH were increased more than twofold at day 20 of gestation. No effects were seen on T3 concentrations in fetal brain (Meerts *et al.*, 2002).

In a study to investigate the effects of PCBs on thyroid-hormone status, female Sprague-Dawley rats were given Aroclor 1254 at a dose of 4 mg/kg bw per day by gastric intubation for 14 days. To test underlying mechanisms, microsomal enzyme activities (CYP isozymes and UDP-GT, indicating metabolic activation and/or biliary clearance), ex-vivo binding of [^{125}I]-T_4 to plasma proteins (suggesting effects on peripheral thyroid-hormone transport), and light microscope morphology of the thyroid gland were studied. The extent of thyroid-hormone reduction (free T4 to 30% and total T4 to 60% of control) observed after exposure to Aroclor 1254 corresponded with a decrease in the ex-vivo binding of [^{125}I]-T_4 to plasma TTR, and with induction of the microsomal phase-I enzymes (ethoxy- and methoxy-resorufin dealkylase, EROD and MROD). The phase-II enzyme UDP-GT was moderately elevated. The thyroid morphology showed activation of the epithelium, but no degenerative alterations correlated with exposure to Aroclor 1254. The results suggested that the decrease in T4 is mainly due to disturbed serum transport, as a result of binding of Aroclor 1254 metabolites to TTR (Hallgren & Darnerud, 2002).

Miller *et al.* (2012) studied the effects of exposure to PCBs and PBDEs on T4 levels in rat offspring from day 6 of gestation until postnatal day 21. In male rat offspring, exposure to PCBs or PBDEs at a dose of 1.7, 5, 10, 20, 40, or 60 µmol/kg bw per day induced equivalent and dose-dependent reductions in T4 from postnatal days 7 to 21. Exposure to equimolar mixtures of PCBs and PBDEs at a dose of 3.4, 10, 20, 40, or 80 µmol/kg bw per day additively reduced T4 levels during the exposure period. The effects on T4 levels were similar in males and females.

In-vivo and ex-vivo systems

The OH-PCB metabolites 4-OH-PCB-69, 4-OH-PCB-106, and 4-OH-PCB-121 were tested for capacity to disrupt the thyroid-hormone system via proliferation of thyroid hormone-dependent rat-pituitary GH3 cells. Growth of GH3 cells was stimulated by all three 4-OH-PCBs (Ghisari & Bonefeld-Jørgensen, 2005). These OH-PCBs were previously reported to bind to the thyroid receptor and to thyroid-hormone transport proteins (Cheek *et al.*, 1999).

PCBs are the most concentrated class of pollutant found in polar bears (*Ursus maritimus*). In plasma samples collected from polar bears, no binding of [^{125}I]-T4 to TTR was observed. Incubation of these plasma samples with [^{14}C]-2,3,3′,4′,5-pentachloro-4-biphenylol [[^{14}C]-4-OH-PCB-109], a PCB metabolite with a higher binding affinity to TTR than the endogenous ligand T4 itself, resulted in competitive binding. Incubation of plasma with T4 at up to 1 mM (a concentration that is not physiologically relevant) did not result in any detectable competition. These results suggested that the binding sites on TTR for T4 in wild polar bears are completely saturated (Gutleb *et al.*, 2010).

Disruption of thyroid-hormone transport may be an important mechanism by which PCBs can alter thyroid-hormone homeostasis. In a systematic in-vitro study of PCB-binding to TTR, the role of *ortho* substitution was investigated in

more detail. PCBs that have only *ortho* substitution show significant binding activity. The congeners most closely resembling the diiodophenolic ring of T4, i.e. di-*meta*-substitution in one or both rings, showed the highest binding activity to TTR. Multiple *ortho* substituents decreased the binding activity of such congeners. PCBs with a single *meta* substitution in one or both rings resemble more closely the monoiodophenolic ring of T3, and showed significantly lower binding activity to TTR. This was consistent with the relatively low binding activity of T3 and the smaller size of chlorine compared with iodine. The addition of *ortho* substituents gave variable results, depending on their position (Chauhan *et al.*, 2000).

In in-vitro studies that assessed the effect of OH-PCBs on thyroid-hormone sulfation, the inhibition of sulfotransferase activity towards 3,3′-diiodo-thyronine (T2) appeared to be similar to that towards T3. Hydroxylated metabolites of PCBs strongly inhibited T2 sulfotransferase activity, the most potent inhibitor being 3-OH-2,3′,4,4′,5-pentachlorobiphenyl (3-OH-PCB-118). An important structural requirement for inhibition of T2 sulfotransferase by OH-PCBs is the presence of a hydroxyl group in the *para* or *meta* position, with *ortho*-OH-PCBs being much weaker inhibitors (Schuur *et al.*, 1998a, b).

4.3.4 Effects on the immune system

The effects of PCBs on several parameters related to the immune system have been reported for humans, and more extensively for experimental animals (reviewed by Tryphonas & Feeley, 2001).

(a) Adults

Immunomodulatory effects of PCBs have been reported in workers occupationally exposed to these chemicals, in humans following consumption of contaminated fish, and in populations accidentally exposed to PCBs and their heat-degradation products, PCDFs, and polychlorinated quarterphenyls (PCQ) via consumption of contaminated rice oil (the Yusho and Yucheng poisoning incidents). In addition, PCB exposure during prenatal and early life has been associated with incidence of infectious and allergic diseases in children, and alterations in immune-system development.

Lawton *et al.* (1985) tested 194 workers exposed occupationally (152 men, 42 women) to one or more of the Aroclors 1254, 1242, and 1016 in a capacitor plant factory for an average duration of 17 years. The results taken in 1976 were compared with those from the same workers taken in 1979, two years after discontinuation of all PCB use in 1977. Significantly increased levels of leukocytes, with a concomitant increase in levels of lymphocytes, monocytes and eosinophils, were observed when these workers were tested in 1976. Interestingly, the levels of circulating polymorphonuclear cells were reduced in the same workers. Similar, but not statistically significant, shifts in leukocyte levels were noted when testing was repeated in 1979. A positive association was observed between serum PCB concentrations and blood monocytes, and was reported to persist even 2 years after discontinuation of PCB use. [The Working Group noted that the extent to which PCB exposure compromises the immune system could not be estimated on the basis of immune-cell alterations, since measurement of functional immune parameters was not part of the study protocol.]

In contrast, a study by Emmett *et al.* (1988a, b) of 55 transformer repairmen working in a factory and exposed to Aroclors 1260 and 1242 did not report any significant exposure-related effects on the immune system. The percentage of workers with positive skin responses (delayed-type hypersensitivity) to mumps and trichophyton antigens was similar to that of 56 nonexposed workers.

Follow-up studies of the Yusho and Yucheng populations indicated that several immune-related parameters were disrupted in exposed

adults. These included a statistically significant decrease in serum levels of immunoglobulins A and M, reduced T-helper (Th) and increased T-suppressor cells (Ts) resulting in reduced Th:Ts cell ratio, persistent respiratory distress caused by Gram-negative bacilli-infected airways, and increased in-vitro lymphoproliferative responses of peripheral blood leukocytes to phytohaemaglutinin, concanavalin A, and pokeweed mitogens at 1 and 3 years after exposure. Furthermore, a reduced number of patients with positive skin-test reactivity to streptokinase/streptodornase antigens was observed at 1 year after exposure, and to tuberculin antigens at up to 4 years after exposure (Lü & Wu, 1985; Nakanishi *et al.*, 1985), while some other immunological effects persisted up to 30 years after exposure (Masuda, 2001).

Consumption of contaminated fish has been associated with some effects on the immune system. High consumption of fatty fish from the Baltic Sea correlated positively with B-cell numbers, but negatively with the percentage of cytotoxic ($CD8^+$) T-cells in 68 fishermen in Latvia (Hagmar *et al.*, 1995). [The significance of these observations was not clear, since no functional immune parameters were examined.]

Svensson *et al.* (1994) studied levels of leukocytes in a group of 23 men in Sweden who consumed high levels of fatty fish species from the Baltic Sea and compared results with 20 men who ate practically no fish. No effects were reported on leukocyte counts, the number of total lymphocytes or their subsets, or serum immunoglobulin levels. A marginal reduction in natural killer (NK) cell activity was reported for the fish-eating population. This was in agreement with the weakly negative correlation observed between NK cell numbers and blood concentrations of PCB-126 and PCB-118 in some of the same subjects tested 3 years previously.

(b) Children

Weisglas-Kuperus *et al.* (1995) studied children residing in the Netherlands and who were exposed, in utero and via breastfeeding, to ambient concentrations of PCBs. The study group consisted of 207 healthy mother–infant pairs. Prenatal exposure to PCBs was estimated by the sum of PCB-118, PCB-138, PCB-153, and PCB-180 (ΣPCB) in maternal and cord plasma, and in breastfed infants by the TEQ levels (based on 17 dioxins and 8 dioxin-like PCBs) in human milk. A higher prenatal PCB/dioxin exposure was associated with increased numbers of T lymphocytes bearing T-cell receptors of the gamma/delta type, increased cytotoxic T-cells at age 18 months in breastfed infants; higher prenatal and postnatal concentrations of PCB/dioxin was associated with reduced monocytes and granulocytes at age 3 months. In follow-up studies, statistically significant associations were observed between prenatal PCB exposure and increased number of lymphocytes, T-cells, and cytotoxic ($CD3^+CD8^+$) cells, memory ($CD4^+$ $CD45RO^+$) cells, T-cell receptor (TcR) $\alpha\beta^+$, and activated T-cell ($CD3^+$ HLA-DR^+) numbers in the toddlers.

Horváthová *et al.* (2011a, b) collected blood specimens from newborns, and infants aged 6 and 16 months, from two districts in Slovakia, Michalovce and Svidnik/Stropkov, that had respectively high and low environmental PCB contamination, and correlated blood PCB concentrations with lymphocyte-receptor expression. The percentages of lymphoid dendritic cells and naive/resting T lymphocytes were significantly increased at 6 months in the Michalovce area compared with those in cord blood samples ($P < 0.001$). Overall there was a positive correlation of terminally differentiated effector memory T-lymphocyte population with age, and a negative linear correlation for myeloid dendritic cells from birth to 6 months in both regions. The Michalovce samples indicated

significantly higher expression of memory T lymphocytes (at birth, 6, and 16 months), terminally differentiated effector memory T lymphocytes (at birth and at 6 months), and lymphoid dendritic cells (at 6 months) than in samples from Svidnik/Stropkov.

Jusko *et al.* (2012) investigated the effect of several PCB congeners on thymus volume in 1134 mother–infant pairs residing in eastern Slovakia. Samples of maternal and infant (age 6 and 16 months) blood were collected and analysed for 15 PCB congeners. Higher maternal concentrations of PCBs were associated with reduced thymus volume at birth, while maternal PCB concentration was not predictive of thymus volume in the infants aged 6 and 16 months.

In a subgroup of 331 children aged 7–10 years from the Hesse, Germany cohort, mean concentrations of PCBs were 0.50 µg/L, and this value was significantly associated with increased levels of serum immunoglobulin M (IgM) (Karmaus *et al.*, 2005).

Similar immune-related sensitivities in adolescence were reported by Van Den Heuvel *et al.* (2002) for a study in Flanders, Belgium. In this study, serum concentrations of PCB-138, PCB-153 and PCB-180, and combined serum dioxin-like activity as determined by AhR-mediated expression of a reporter gene luciferase, were measured in samples from boys and girls (aged 17–18 years) with certain immune-related respiratory complaints. A significantly negative correlation between the percentage of eosinophils and NK cells in peripheral blood and TEQ in serum ($P = 0.009$ and $P = 0.05$, respectively) was observed. Similarly, significant negative correlations were calculated between serum TEQs and levels of specific IgE antibodies to allergens (cat dander, house dust mite, and grass pollen), and the incidence of reported allergies of the upper airways. A significant positive correlation was observed between increased serum TEQs and increased serum IgA levels ($P = 0.05$).

(c) Non-human primates

Unlike all other experimental animal models in which exposure levels were high, the available studies in non-human primates used PCB doses that were relatively low (< 1 mg). Such studies have shown that non-human primates are more sensitive to the immune-related effects of PCBs than any other experimental animal tested. Alterations in the immune system and immunotoxicity were also reported after PCB exposure during prenatal or early life.

Thomas & Hinsdill (1978) investigated immunological parameters in groups of eight rhesus monkeys fed diets containing Aroclor 1248 at a dose of 0.1 or 0.2 mg/kg bw per day for 11 months. The reported immune-related effects were seen only at 0.2 mg/kg bw and included significantly reduced titres of antibodies to sheep red blood cells (SRBC) at weeks 1 and 12 after primary immunization, and decreased percentage of gamma-globulin after 20 weeks, compared with a control group of five monkeys. The response to tetanus toxoid was not affected by treatment. Reduced titres to SRBC were also reported in the single female cynomolgus monkey *(Macaca fascicularis)* treated with a PCB mixture with constituents similar to those ingested by Yusho patients, and containing predominantly penta- and hexachlorobiphenyls and no PCDFs, prepared from Kanechlor 400 and administered at 5 mg per day for 20 weeks (Hori *et al.*, 1982).

Differences in PCB-induced toxicity were investigated in cynomolgus *(Macaca fascicularis)* and rhesus *(Macaca mulatta)* monkeys (Tryphonas *et al.*, 1986; Arnold *et al.*, 1990). In these studies, groups of four cynomolgus and four rhesus monkeys ingested Aroclor 1254 in apple juice-gelatin-corn oil emulsion at doses of 0.00 (control) or 280 µg/kg bw per day for 12–13 months (cynomolgus monkeys) and 27–28 months (rhesus monkeys) respectively. The total serum IgM levels and titres to anti-SRBC (primary response) antigens were significantly

reduced in both species. Based on clinical and pathological findings, the rhesus monkeys were more sensitive to PCB-induced toxicities than the cynomolgus monkeys, although effects on the immune system were similar in both species.

A long-term study with Aroclor 1254 (Tryphonas *et al.*, 1989, 1991a, b; Arnold *et al.*, 1993, 1995) was of particular significance since it was the only long-term study in which low doses (range, 5–80 µg/kg bw per day) of commercial PCB mixtures were used. Immunological effects were reported after 23–25 months (phase I) (Tryphonas *et al.*, 1989), during which time a blood PCB pharmacokinetic equilibrium was established, and after 55 months (phase II) (Tryphonas *et al.*, 1991a, b). Testing at phase I detected significant shifts in Th and Ts lymphocyte subsets (decreased Th, increased Ts and decreased Th:Ts cell ratio) at 80 µg/kg bw per day, and significantly reduced titres in response to SRBC antigens (Tryphonas *et al.*,1989). The response to SRBC antigens was significantly reduced even at a dose of 5 µg/kg bw per day. These effects in monkeys were comparable to those reported for the Yucheng population at 1 and 3 years after exposure (Lü & Wu, 1985). Several significant immune-related parameters were affected in monkeys exposed continuously to Aroclor 1254 for 55 months (phase II). Effects included: a dose-related decrease in the anamnestic (IgM and IgG) response to SRBC antigens; a dose-related decrease in the lymphoproliferative response of leukocytes to the mitogens concanavalin A and phytohaemaglutinin, but not to pokeweed mitogen (mostly B-cell dependent); reduced monocyte activity (peak chemiluminescence after phorbol myristate acetate activation); significantly higher levels of serum complement (CH_{50}) activity across all treated groups compared with controls; a dose-related significant increase in thymosin α1 ($T\alpha_1$) levels in treated groups compared with controls; a significant but not dose-related increase in levels of interferon at the 20 and 80 µg/kg bw per day, with a significantly reduced interferon level at 40 µg/kg bw per day. Tumour necrosis factor (TNF) levels were not affected significantly by treatment (Tryphonas *et al.*, 1991a, b).

Hand-reared infant rhesus (*Macaca mulatta*) monkeys (age, 66 weeks) were treated with a mixture of PCB congeners at a dose of 7.5 µg/kg bw per day, which represents the approximate daily intake of a nursing infant whose mother's breast milk contained PCBs at a concentration of 50 ppb. The PCB congeners used for treatment were those commonly found in human breast milk in Canada. Treatment continued until the monkeys reached age 20 weeks. Significant treatment-related effects characterized by reduced antibody responses to SRBC antigens, and reduced levels of the HLA-DR cell surface marker were observed (Arnold *et al.*, 1999).

Groups of eight adult female rhesus monkeys were fed diets containing Aroclor 1248 at a concentration of 2.5 or 5.0 ppm for approximately 1.5 years (Allen & Barsotti, 1976). Six of the eight monkeys treated with Aroclor 1248 at 5.0 ppm, and all monkeys at 2.5 ppm were successfully bred after 6 months of exposure. There was one live infant born among monkeys at 5.0 ppm, and five infants born to monkeys at 2.5 ppm. Infants were permitted to nurse with their mothers. Three infants died within 8 months, after 44, 112 and 239 days, respectively. At necropsy, histopathological observations of the infant tissues included a near complete absence of thymocytes in the cortical and medullary areas of the thymus, extremely small lymph nodules of the spleen with inapparent germinal centres, and hypocellularity of the bone marrow.

(d) Rodents and rabbits

(i) Effects on the thymus

Commercial PCB mixtures

Thymic atrophy was detected in female White New Zealand rabbits fed diets containing Aroclor 1260 at a dose of 118 mg/kg bw per

day for 38 days, or Aroclor 1260 at a dose of 120 mg/kg bw per day for 28 days (Vos &Beems, 1971; Vos & Notenboom-Ram, 1972); in male White New Zealand rabbits fed Aroclor 1254 at a dietary concentration of 20, 45.8, or 170 ppm [0.92, 2.10 or 6.54 mg/kg bw per day] for 56 days (Street & Sharma, 1975); in male Fischer 344 rats given Aroclor 1254 at a dose of 10 or 25 mg/kg bw per day by gavage for 15 weeks (Smialowicz *et al.*, 1989); in female guinea-pigs fed Clophen A60 at a dietary concentration of 50 ppm for 49 days (Vos & van Driel-Grootenhuis, 1972) and in male Sprague-Dawley rats fed Aroclor 1262, 1254, or 1248 at 1% of the diet for 6 weeks. The severity of thymic atrophy was Aroclor 1254 = Aroclor 1248 > Aroclor 1262 (Allen & Abrahamson, 1973).

Thymic atrophy was not detected upon exposure to Aroclor 1248 when fed to female outbred albino mice (50, 100, 500 or 1000 ppm) for 3 to 5 weeks (Thomas & Hinsdill, 1978), or to Aroclor 1242 (167 ppm) fed to Balb/c mice (Loose *et al.*, 1979).

PCB congeners

Thymic atrophy characterized by reductions in cortical and medullary volume was also reported in weanling male and female Sprague-Dawley rats treated with feed containing individual PCB congeners for 13 weeks at the following concentrations: PCB-126, 0.1–100 ppb (0.01–7.4 µg/kg bw per day) (Chu *et al.*, 1994); PCB-153, 0.05–50 ppm (3.6–3534 µg/kg bw per day) (Chu *et al.*, 1996b); PCB-28, 0.05–50 ppm (2.8–3783 µg/kg bw per day) (Chu *et al.*, 1996c); and PCB-105, 0.05–50 ppm (3.9–4327 µg/kg bw per day) (Chu *et al.*, 1998). In contrast, PCB-77, PCB-118, and PCB-128 did not have any significant effects on the thymus when fed to weanling male and female Sprague-Dawley rats for 13 weeks at the following concentrations: PCB-77: 0.01–10 ppm (0.73–768 µg/kg bw per day) in males; 0.01–10 ppm (0.92–892 µg/kg bw per day) in females (Chu *et al.*, 1995); PCB-118: 0.01–10 ppm (0.66–683 µg/kg bw per day) in males; 0.002–2 ppm (0.17–170 µg/kg bw per day) in females; PCB-128: 0.05–50 ppm (4.5–4397 µg/kg bw per day) (Lecavalier *et al.*, 1997).

In male C57BL/6 (Ah+) and DBA/2 (Ah-) mice given intraperitoneal doses of PCB-77 (DL-PCB) or PCB-52 (NDL-PCB) at 0, 10, or 100 mg/kg bw per day, thymic atrophy was observed only in C57BL/6 mice treated with PCB-77 (Silkworth & Grabstein, 1982). The results suggested that PCB immunotoxicity in mice is mediated through the AhR, present only in the C57BL/6 mice.

(ii) Effects on humoral immunity

Commercial PCB mixtures

Several studies reported effects of PCBs on humoral immune reactivity. A significant reduction in production of antibodies to tetanus toxoid was noted in guinea-pigs fed Clophen A60 (Vos & van Driel-Grootenhuis, 1972), to keyhole limpet haemocyanin (KLH) in rats fed Aroclor 1254 (Exon *et al.*, 1985), and to SRBC using the plaque-forming cell assay in mice given Aroclor 1254 intraperitoneally (Wierda *et al.*, 1981; Loose *et al.*, 1979). Mice genetically engineered to be either aryl hydrocarbon-responsive (Ah^b/Ah^b) or non-responsive (Ah^d/Ah^d) did not exhibit the same sensitivity to PCB-induced suppression in the plaque-forming cell assay. For example, C57BL/6N (Ah^b/Ah^b) mice injected intraperitoneally with Aroclor 1254 at a dose of 250–750 mg/kg bw exhibited significant reductions in plaque-forming cell numbers after 5 days, compared with controls, while DBA/2N (Ah^d/Ah^d) mice failed to demonstrate any significant PCB-induced effects on plaque-forming cell numbers, compared with controls (Lubet *et al.*, 1986).

PCB congeners

Cotreatment of C57BL/6 B6 mice with PCB-153 and TCDD showed that PCB-153 partially antagonized TCDD-mediated immunotoxicity in various assays (Biegel *et al.*, 1989).

Individual congeners were also assessed for their immunotoxicity in AhR-responsive or AhR-non-responsive mouse models. Bandiera *et al.* (1982) reported that PCB-77 binds AhR with high affinity and causes severe suppression of the humoral antibody response in C57BL/6 B6 (Ah^b/Ah^b) mice. In comparison, PCB-77 exhibited lower binding affinity for AhR in DBA/2N (Ah^d/Ah^d) mice and did not cause any immune-related effects (Silkworth & Grabstein, 1982). In contrast, the di-*ortho*-substituted PCB-52 had weak AhR binding affinity and was not immunosuppressive in either mouse strain (Silkworth & Grabstein, 1982).

(iii) Effects on cellular immunity

The effects of PCBs were less pronounced on cellular immune responses than on humoral immune reactivity. Reduced skin reactivity to tuberculin was detected in female guinea-pigs fed Clophen A60 at 50 or 250 ppm for 49 days (Vos & van Driel-Grootenhuis, 1972; Vos & Van Genderen, 1973). In contrast, no effects were detected when dinitrochlorobenzene was used as the skin sensitizer in female Swiss-Webster mice fed Aroclor 1254 at 10, 100, or 250 ppm [1.17, 116, 292 mg/kg bw per week] for 12 weeks (Talcott & Koller, 1983). Similarly, White New Zealand male rabbits fed Aroclor 1254 at 170 ppm [6.54 mg/kg bw per day] for 56 days did not show any effects on skin reactivity to tuberculin sensitization (Street & Sharma, 1975).

Studies on the mitogen-induced proliferative activity of splenic mononuclear leukocytes and the mixed lymphocyte response, both in-vitro correlates of cellular immune responses, also gave conflicting results and suggested that PCBs may affect a specific subpopulation of T lymphocytes. A few studies reported that phytohaemagglutinin-induced leukocyte blastogenic activity was increased upon exposure to Aroclors, while no effect was noted when concanavalin A, *S. typhimurium*, or pokeweed mitogens were used (Bonnyns & Bastomsky, 1976; Wierda *et al.*, 1981; Smialowicz *et al.*, 1989). The mixed lymphocyte response was not affected by treatment with the less chlorinated Aroclor 1242 (Carter & Clancy, 1980; reviewed by Silkworth & Loose, 1981).

Nakanishi *et al.* (1995) treated female Sprague-Dawley rats (age, 8 weeks) intraperitoneally with 5 mg of Kanechlor 400 in 2 mL of corn oil, and effects on the immune system were examined at termination of the study 4 weeks later. The percentage of T lymphocytes, and T-helper and T-suppressor cells, was significantly decreased in the treated groups compared with the controls. In contrast, the percentage of T lymphocytes in the bronchoalveolar lavage fluid was not significantly increased after treatment with Kanechlor 400. The percentage of T-suppressor cells increased significantly, while the percentage of T-helper cells was not affected by treatment. Release of O_2^- by alveolar macrophages, stimulated with either wheat germ lectin or phorbol myristate acetate, increased significantly compared with the controls (Martin *et al.*, 1981). In addition, there was mild inflammation of the alveoli after administration of PCBs. In support of this observation, Kikuchi *et al.* (1971) reported that lung autopsies for two Yusho patients showed the presence of pulmonary haemorrhage and pulmonary oedema. [It is conceivable that failure to remove O_2^- produced by macrophages might be responsible for the observed pathogenesis of interstitial changes of the lung after treatment with PCBs].

(iv) Effects on innate (non-specific) immunity

The cellular components of innate immunity, including phagocytic cells (neutrophils, macrophages) and NK cells, are targets of PCB-induced immunotoxicity. Functional impairment of these cells is characterized by reduced phagocytic activity and consequently diminished ability to eliminate pathogenic infections in PCB-exposed experimental animals, as well as compromised immunosurveillance mechanisms.

Male ICR mice fed diets containing Kanechlor 500 at 400, 200, or 100 µg per gram feed showed increased susceptibility to herpes simplex virus compared with control mice (Imanishi *et al.*, 1980). Likewise, Koller (1977) demonstrated that Balb/c male mice fed diets containing Aroclor 1242 at 375 ppm for 6 months showed a significant increase in susceptibility to Moloney leukaemia virus; this effect was not seen with Aroclor 1221. In Balb/c male mice given feed containing Aroclor 1242 at 167 ppm for 6 weeks, there was significantly increased susceptibility to *S. typhosa* endotoxin and to malaria parasite *Plasmodium berghei* (Loose *et al.*, 1979). Reduced clearance of *Listeria monocytogenes* was observed in adult and neonate male and female ICR mice given Aroclor 1254 at 75 mg/kg bw per day by gavage for 14 days (Smith *et al.*, 1978). Thomas & Hinsdill (1980) reported increased sensitivity to endotoxin challenge in outbred, female albino mice fed Aroclor 1248 at 100 ppm, but no effect on resistance to *S. typhimurium* in mice fed Aroclor 1248 at 1000 ppm.

NK-cell activity was reported to be decreased in male Fischer 344 rats exposed daily to Aroclor 1254 at 10 or 25 mg/kg bw by gastric intubation for up to 15 weeks (Smialowicz *et al.*, 1989), and in male Sprague-Dawley rats fed Aroclor 1254 at 50 or 500 ppm for 10 weeks (Talcott *et al.*, 1985; Exon *et al.*, 1985).

Paradoxically, despite the evidence that PCB-induced immunosuppression impairs immune surveillance, Aroclor 1254 protected mice and rats against certain kinds of experimentally induced tumours, such as Ehrlich's tumour ascites (Keck, 1981) and primary Walker 256 tumour (Kerkvliet & Kimeldorf, 1977).

(e) Fish and marine mammals

As top predators, marine mammals and large fish bioaccumulate PCBs at high concentrations in fat. Several studies have reported on the immunotoxic effects of PCBs on fish and marine mammals in contaminated environments (Mahy *et al.*, 1988; Osterhaus & Vedder, 1988; Cleland *et al.*, 1989; Dietz *et al.*, 1989; Visser *et al.*, 1993; De Swart *et al.*, 1994; Ross *et al.*, 1995, 1996; Hammond *et al.*, 2005; Iwanowicz *et al.*, 2009; Frouin *et al.*, 2010; Duffy-Whritenour *et al.*, 2010).

4.3.5 Effects on inflammatory response

Several in-vivo and in-vitro studies have investigated the role of PCBs in the development of inflammatory responses, and are reviewed in the following section. Pertinent to this review are the following questions: (i) is the observed inflammation in PCB-treated animals directly related to PCB exposure, or is it a secondary development following PCB-induced toxicity in target organs; and (ii) does inflammation play an active role in the development of cancer after PCB exposure?

(a) Humans

No studies defining an association between exposure to PCBs and the development of inflammation in relation to cancer in humans were available to the Working Group.

(b) Experimental animals in vivo

(i) Commercial PCB mixtures

Tryphonas *et al.* (1984) reported significant changes indicative of an ongoing inflammatory response in the liver of cynomolgus monkeys *(Macaca fascicularis)* treated with Aroclor 1254 or Aroclor 1248. These changes included "ground glass" appearance of the cytoplasm and pyknosis of the nuclei with or without neutrophil infiltration, eosinophilic necrosis of single or clusters of hepatocytes often with neutrophilic infiltration or collapse of the connective tissue framework, and moderate, diffuse sinusoidal fibrosis and hypercellularity, and were associated with PCB-induced necrosis of the liver.

Interstitial inflammation of the liver was also observed in cynomolgus monkeys fed with

P-KC-400 (Kanechlor 400 from which PCDFs had been removed, largely containing tri- and tetrachlorobiphenyls), or PY-PCB (a PCB mixture with constituents similar to those ingested by Yusho patients, and largely containing penta- and hexachlorobiphenyls and no PCDF) at 5 mg per day, for 20 weeks (Hori *et al.*, 1982).

(ii) PCB congeners

Inflammatory responses, presumably secondary to PCB-induced toxic effects, have been reported in long-term studies of carcinogenicity in rats treated with PCB-126 (NTP, 2006a), PCB-153 (NTP, 2006b), PCB-126 + PCB-153 (NTP, 2006c, varying ratios study), PCB-126 + PCB-118 (NTP, 2006d), and PCB-118 (NTP, 2010). The incidence and severity of inflammation in the treated groups varied according to the congener administered. For PCB-118 and PCB-126, the incidence of inflammation and degree of severity were significantly increased in core groups receiving the three higher doses than in the controls, while for PCB-153, the incidence in the core groups was only slightly increased compared with the controls and was not dose-dependent. In addition to the core groups, inflammation, albeit of low incidence and intensity, was also observed in the control groups in the studies with PCB-118 and PCB-126, and in the uterus of rats in the PCB-153 control group, but not in the ovary of rats in the same group.

Sipka *et al.* (2008) investigated the potential for various PCB congeners to induce inflammation in mice. Mice were given a single gavage dose (150 µmol/kg bw) of PCB-77, PCB-104, or PCB-153. The levels of specific inflammatory mediators including intercellular adhesion molecules (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1) mRNA and monocyte chemoattractant protein-1 mRNA (MCP-1) were determined in the liver, lung, and brain. All three PCB congeners activated inflammatory mediators, and the organs affected varied according to the congener used. PCB-77 and PCB-104 caused induction of all three inflammatory mediators in the liver and lungs, but not in the brain. In contrast, the effects of PCB-153 varied across mediators and were predominantly seen in the lung and brain. Concentrations of PCB-153 were higher in the lung and brain than in the liver, and PCB-153 was the only PCB to be detected in the brain (Sipka *et al.*, 2008). These observations suggested that the observed differences in target organ for the effects on inflammatory mediators were due to differences in PCB-congener accumulation in the organs affected.

In another study, a single dose of PCB-77 resulted in increased expression of VCAM-1 only in the wildtype (AhR-positive) mice, and not in mice lacking the AhR gene (Hennig *et al.*, 2002b).

Sipos *et al.* (2012) suggested that exposure to environmental toxicants including PCBs may cause vascular inflammation that facilitates the development of brain metastases. The crucial event in metastasis is adhesion of blood-borne tumour cells to the vascular endothelium, followed by transcapillary migration. In wild-type or ICAM-1-deficient mice injected with Lewis lung carcinoma cells via the carotid artery, oral pretreatment with PCB-118 enhanced development of brain metastases by inducing overexpression of ICAM-1 (also designated as CD54) and VCAM-1 in the brain endothelium (Sipos *et al.*, 2012).

(c) In-vitro studies

In-vitro studies by Narayanan *et al.* (1998) indicated that Aroclor 1242 and PCB-47 (a major constituent of Aroclor 1242) impaired the oxidative burst (respiratory burst) in human neutrophils by inhibiting the antioxidant enzyme superoxide dismutase, which converts O_2^- to H_2O_2. Pre-incubation of neutrophils with Aroclor 1242 or PCB-47 before stimulation with phorbol 12-myristate 13-acetate, elevated the respiratory burst, and resulted in a significant increase in intracellular O_2^- production and a significant

decrease in H_2O_2 compared with that in unexposed but agonist-stimulated neutrophils.

Additional in-vitro studies indicated that non-coplanar PCBs stimulate neutrophil production of superoxide anions (O_2^-) by a mechanism that is structure-specific and dependent on the chlorine substitution pattern of the biphenyl rings. On the contrary, coplanar congeners with high affinity for AhR do not activate neutrophils to produce superoxide anions and may inhibit this response (Brown *et al.*, 1998). In these studies, neutrophils were isolated from male Sprague-Dawley rats and exposed to specific PCB congeners at 0 (vehicle), 10, or 50 µM for 30 minutes at 37 °C, before stimulation with phorbol 12-myristate 13-acetate at 0 or 20 ng/mL. PCB-4, PCB-8, or PCB-11 (50 µM) stimulated neutrophils to produce O_2^-. Incubation of neutrophils with PCB-15, PCB-126, PCB-127, or PCB-128 did not result in generation of O_2^-. Of the various congeners tested, PCB-8 elicited the highest production of superoxide anions.

Exposure to PCB-4, PCB-8, PCB-11, or PCB-128 before addition of phorbol myristate acetate caused a significant increase in the amount of O_2^- produced that was greater than that seen with either compound alone. Phorbol myristate acetate-stimulated production of O_2^- was unaffected by prior exposure to PCB-15, PCB-126, or PCB-127. In separate experiments, PCB-126 inhibited the amount of O_2^- produced in response to activation with either PCB-4 or PCB-11. From these results it appeared that non-coplanar congeners are capable of stimulating neutrophil production of O_2^-. Coplanar congeners with a high affinity for AhR do not activate neutrophils to produce O_2^- and may inhibit this response.

Kwon *et al.* (2002) investigated the effects of PCB-153 on the expression of cyclooxygenase-2 (COX-2) and pro-inflammatory cytokines such as IL-1β, IL-6 and TNF-α in a human leukaemic mast cell line. The expression of TNF-α and IL-1β mRNA was not dependent on PCB-153, while the expression of COX-2 and IL-6 mRNA was highly induced by PCB-153. Pre-treatment with pyrrolidine dithiocarbamate, an NF-κB-pathway inhibitor, suppressed induction of COX-2, TNF-α and IL-1β, and reduced the induction of IL-6 mRNA by PCB-153.

The effects of PCBs on the activation of human granulocytes were investigated by Voie *et al.* (1998). Respiratory burst activity was measured as luminol-amplified chemoluminescence in human granulocytes. *Ortho*-substituted PCB congeners (PCB-47 and PCB-4) stimulated chemoluminescence in a concentration-dependent manner (ED_{50}, approximately 10 µM), while *meta*- and *para*-substituted congeners had no significant effect. Furthermore, using several enzyme-specific inhibitors, it was shown that PCB-activated chemiluminescence was dependent on Ca^{++}-dependent phospholipase D or phospholipase C, phosphatidylinositol 3-kinase, and protein kinase C activation before activation of the NADPH oxidase.

In an early experiment, porcine pulmonary artery-derived endothelial cells were incubated for up to 24 hours with PCB-77, PCB-114, or PCB-153, which were selected for their varying binding avidities to AhR and different capacities to induce CYP (Toborek *et al.*, 1995). PCB-77 and PCB-114 significantly disrupted endothelial barrier function in a dose-dependent manner by allowing an increase in albumin transfer across endothelial monolayers. PCB-77 and PCB-114 also enhanced oxidative stress (increasing levels of 2,7-dichlorofluorescein fluorescence, lipid hydroperoxides, and intracellular calcium) and caused increased activity and level of CYP 1A, and decreased levels of vitamin E in the culture medium. In contrast, incubation of endothelial cells with the non-dioxin-like PCB-153 did not have any effect on cellular oxidation, intracellular calcium levels, or on endothelial barrier function.

Additional in-vitro experiments (Hennig *et al.*, 1999; 2002a, 2002b) further suggested

that PCBs are atherogenic, exerting their effect by disrupting normal cellular functions of the vascular endothelium, and confirmed that oxidative stress and activation of the CYP1A subfamily may play a role in the events that lead to atherogenicity.

Treatment of porcine endothelial cells with the DL-PCBs PCB-77, PCB-126, or PCB-169 resulted in increases in expression of the *CYP1A1* gene, oxidative stress, and the DNA-binding activity of NF-κB in a concentration-dependent manner. PCB-126 elicited a maximal response at the lowest concentration (0.5 μM) tested. In addition, all three coplanar PCBs increased endothelial production of IL-6. The expression of adhesion molecule VCAM-1 by endothelial cells was highest at 3.4 μM PCB-77 or PCB-169 (Hennig *et al.*, 2002b).

When human umbilical vein endothelial cells (HUVEC) were treated with PCB-104, a non-coplanar congener, PCB-104 increased the oxidative stress and markedly upregulated the expression of monocyte chemoattractant protein-1 (MCP-1), and the adhesion molecules E-selectin, and ICAM-1, at both the mRNA and protein levels, in a time and concentration-dependent manner. Furthermore, PCB-104 stimulated the adhesion of THP-1 cells (a human acute monocytic leukaemia cell line) to endothelial cell monolayers (Choi *et al.*, 2003).

4.3.6 Quantitative structure–activity relationships (QSAR)

Based on their structure–activity characteristics, PCB congeners are generally grouped as dioxin-like and non-dioxin-like (see Section 1.1.1):

- DL-PCBs are *meta*-/*para*-chloro-substituted PCBs and include PCB-77, PCB-126, PCB-169 and their mono-*ortho*-chlorinated derivatives. These congeners can adopt a coplanar structure and display avid binding to AhR (avidity to AhR diminishes with *ortho*-chloro-substitution). AhR activation leads to a multitude of biological and toxic manifestations, referred to as "dioxin-like activity".
- NDL-PCBs are *ortho*/*para*-substituted PCBs. *Ortho*/*para*-substitution (at least two chlorines in *ortho* positions) is associated with the capacity to induce CAR/PXR-dependent gene expression (e.g. CYP2B, CYP3A isoenzymes). CAR agonists have substitutions in *ortho*, *para* with or without *meta* substitution, while PXR agonists and ryanodine agonists have multiple *ortho* positions substituted with chlorines.
- Some PCB congeners do not elicit activation of AhR, CAR, or PXR.

PCB congeners can also be grouped as lower- and higher-chlorinated congeners. The number of chlorine substituents is linked to persistency and bioaccumulation in animals and humans; less chlorinated congeners are typically volatile and metabolically active, and may produce ROS and genotoxic insults (see Section 4.2).

Additionally, a specific configuration may show activity in a specific bioassay, e.g. for endocrine effects (especially modulation of steroid and thyroid nuclear receptors), neurotoxic activities (release of a neurotransmitter, calcium homeostasis), and/or events associated with tumour promotion (e.g. inhibition of GJIC) (see Section 4.3.2).

The TEQ concept used for risk assessement of PCBs is based on AhR-mediated toxicity of DL-PCBs (see Section 4.3.1). In contrast, the toxicity profiles of NDL-PCBs are insufficiently characterized.

Defining key structural toxicity determinants of individual congeners modulating CAR-, PXR-, androgen receptor-, estrogen receptor-, and other receptor-dependent gene expression is not easy; with the exception of AhR, androgen receptor, and estrogen receptor, there were no systematic studies comparing a large series of PCB congeners in a receptor-based bioassay.

Only a few specific QSAR studies addressing carcinogenicity of PCBs have been published. Ruiz *et al.* (2008) attempted to predict mutagenicity and carcinogenicity of all 209 PCB congeners and some oxidative metabolites using experimental data on DNA-adduct formation, on GJIC-inhibition potency, and National Toxicology Program (NTP) rodent carcinogenicity bioassays. Interestingly, a positive mutagenicity activity was predicted for the less chlorinated PCBs and their hydroxy- and benzoquinone metabolites. Carcinogenicity of many di- to hexachlorinated PCBs was predicted by the QSAR based on NTP carcinogenicity studies in mice, while no carcinogenicity was predicted for tested congeners in the analysis for rats. [A significant drawback was that carcinogenicity predictions were not applicable for the highly abundant, higher-chlorinated congeners PCB-153, PCB-170 and PCB-180 (predicted values were outside the optimum prediction space). Therefore QSAR analyses of carcinogenicity of PCB congeners were inadequate, especially when regarding possible extrapolation to hazards in humans.]

An alternative and more complex approach was reported recently by Stenberg *et al.* (2011). Multivariate toxicity profiles and QSAR modelling of NDL-PCBs were used, based on a variety of molecular descriptors. The toxicity profiles of 24 selected PCBs were identified by in-vitro screening; the different mechanisms of action, which were mostly related to endocrine disruption and neurotoxicity, also included tumour promotion. NDL-PCBs were highly purified, to exclude any contaminating dioxin-like compounds before testing (Hamers *et al.*, 2011). QSAR analysis included also several parameters relevant to carcinogenicity, such as ROS production and inhibition of GJIC. Principal component analysis was used to derive general toxicity profiles from experimental in-vitro data, and individual QSAR models were calculated for each in-vitro response using a set of 67 chemical descriptors. It was shown that PCBs could be divided into at least three major clusters; the DL-PCBs, and two separate NDL-PCB clusters with similar toxicity profiles. The first NDL-PCB cluster included mainly less-chlorinated, *ortho*-substituted congeners with generally higher biological activities (e.g. PCB-28, PCB-95, PCB-101, PCB-136); this subset of congeners was also the most active in the study of GJIC inhibition. The second cluster of NDL-PCBs included congeners with a narrow effective concentration and lower biological activities, with the exception of three assays related to endocrine activity (e.g. PCB-118, PCB-138, PCB-153, PCB-170, PCB-180) (Stenberg *et al.*, 2011).

QSAR approaches might become a useful tool for evaluation and prediction of toxicity of PCBs related to carcinogenesis; however, currently their use is hampered by the lack of data on specific mechanisms of action for larger congener sets.

4.4 Organ toxicity relevant to carcinogenicity

The reader is referred to Section 3.1.2 and Table 3.1 for study design and additional results of the experiments described below.

4.4.1 Hepatic preneoplastic lesions

(a) Promotion of preneoplastic lesions

(i) Commercial PCB mixtures

PCB mixtures, including Aroclor 1254, Clophen A 30, Clophen A 50, and Phenoclor DP6, have shown promoting activity in liver carcinogenesis (Glauert *et al.*, 2001). Several initiating agents were used, including diethylnitrosamine (DEN), aflatoxin B_1, and benzo[*a*]pyrene. The following markers of altered hepatic foci were used in these studies: gamma-glutamyl transferase (GGT), ATPase, and glycogen. The promoting activity of PCBs was observed in males and females. In one study, the promoting activity

of Clophen A 50 was much higher in female rats than in males (Deml & Oesterle, 1982); a similar observation was made for phenobarbital (Xu *et al.*, 1990). In mice, males are more susceptible than females to hepatocarcinogenesis; higher production of IL-6 by Kupffer cells in males may be responsible for this sex-specific difference (Naugler *et al.*, 2007). In a dose–response study with Clophen A 50, a threshold dose (1 mg/kg bw, three times per week, for 11 weeks) was identified (Deml & Oesterle, 1987).

(ii) Individual congeners

Many studies have examined the ability of individual PCB congeners to promote altered hepatic foci in rat liver (Glauert *et al.*, 2001). Most of the studies used DEN as the initiating agent, whether as a single necrogenic dose, as a low dose in conjunction with partial hepatectomy, as a low dose in newborn animals, or in the drinking-water for 10–12 days. The following markers of altered hepatic foci were used in these studies: GGT, GSTπ, ATPase, and/or glucose-6-phosphatase. PCB congeners that had promoting activity included non-*ortho* PCBs (PCB-77 and PCB-126), which activated AhR; di-*ortho*-substituted PCBs (PCB-47, PCB-49, and PCB-153), which activated CAR; and mono-*ortho*-substituted PCBs (PCB-105, PCB-114, PCB-118, and PCB-156), which activated both receptors. Non-*ortho*-PCBs were the most efficacious (Glauert *et al.*, 2001). PCBs that did not induce (PCB-3 and PCB-15) or that weakly induced (PCB-28 and PCB-101) either receptor had poor promoting activity (Oesterle & Deml, 1981; Deml *et al.*, 1985; Buchmann *et al.*, 1991; Kunz *et al.*, 2006). [These differences could be due to pharmacokinetics as well as pharmacodynamics.]

(iii) Combinations of individual congeners

Several studies have investigated the effects of administering combinations of two or more PCB congeners. Most of these studies found that the co-administration of non-*ortho* and di-*ortho* PCBs produced less than additive effects, while administration of two non-*ortho* PCBs produced additive effects. These studies used DEN as the initiating agent, either as a low dose in combination with partial hepatectomy, or as a hepatotoxic dose.

In the earliest study, Sargent *et al.* (1991) examined the separate and combined effects of dietary administration of (di-*ortho*) PCB-52 at 10 ppm, and (non-*ortho*) PCB-77 at 0.1 ppm for 1 year in rats. When administered separately, PCB-77 did not increase the number or volume of altered hepatic foci, but PCB-52 increased the volume fraction but not the number of altered hepatic foci. Coadministration of PCB-52 and PCB-77, however, increased both the number and volume fraction of altered hepatic foci in a more than additive manner. In a study examining the interactive effects of a non-*ortho*-substituted PCB (PCB-126), a mono-*ortho*-substituted PCB (PCB-105), and a di-*ortho*-substituted PCB (PCB-153), no more than additive effects were observed. An additive effect was observed with PCB-105 + PCB-153, while less than additive effects were observed for PCB-126 + PCB-153, and for PCB-126 + PCB-105 (Haag-Grönlund *et al.*, 1998). In another study, PCB-77 and PCB-153 were administered every 2 weeks separately at 300 µmol/kg bw, or in combination at 150 µmol/kg bw (total PCB dose, 300 µmol/kg per injection) for four injections (Berberian *et al.*, 1995). Numbers and volume of foci induced by PCB-77 were decreased by the coadministration of PCB-153. In a study using a similar experimental design, rats were injected four times with PCB-77 or PCB-153 (100 or 300 µmol/kg bw), or PCB-77 + PCB-153 (100 µmol/kg bw each) biweekly. Both PCB-77 and PCB-153 separately increased the number and volume of GSTP-positive foci, but coadministration of PCB-153 inhibited the number and volume of foci induced by PCB-77 (Tharappel *et al.*, 2002). When PCB-126 (non-*ortho*) and PCB-153 (di-*ortho*) were coadministered using 14 combinations of doses, a less

than additive effect was observed (Dean *et al.*, 2002). Finally, the tumour-promoting activity of a polyhalogenated aromatic hydrocarbon mixture (TCDD; 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin; 2,3,4,7,8-pentachlorodibenzofuran; PCB-126; PCB-118; and PCB-156) with or without PCB-153 (di-*ortho*) was compared with that of TCDD alone, the mixture and TCDD having the same total TEF (van der Plas *et al.*, 1999). The mixture produced a lower mean volume of foci and volume fraction of foci in the liver than did TCDD alone. The addition of PCB-153 slightly increased the mean volume of foci and volume fraction of foci in the liver, but still not above that produced by TCDD alone. TCDD and PCB-126 (non-*ortho*) were found to have an additive effect in another study (Hemming *et al.*, 1995).

(b) Initiation of preneoplastic lesions

Studies examining the effect of PCBs as initiating agents fell into two categories: those that examined the effect of PCB treatment with no subsequent chemical treatment, and those in which PCB treatment was followed with protocols designed to shorten the latency period and increase the number and size of lesions, such as the Solt-Farber selection protocol (Solt *et al.*, 1977; Tsuda *et al.*, 1980; Semple-Roberts *et al.*, 1987). Groups of animals treated with PCBs only were often control groups in initiation–promotion studies, e.g. PCB-only groups being used to compare initiator + PCB groups.

Several studies have observed a small increase in the number of altered hepatic foci after treatment with PCBs only. These PCBs included Clophen A 50, PCB-49, PCB-77, and PCB-114 (reviewed in Glauert *et al.*, 2001). There are two possible explanations for this phenomenon: first, these PCBs have initiating activity; or second, these PCBs are very efficient at promoting cells that have initiated spontaneously (e.g. from errors in DNA replication, exposure to background chemicals or radiation, etc.). Other studies, however, have observed that certain PCBs, including Aroclor 1254, Clophen A 50, PCB-52, PCB-77, and PCB-153, do not produce any increase in the number of altered hepatic foci after treatment with the PCB congener only (reviewed in Glauert *et al.*, 2001). [Possible reasons for obtaining different results for the same PCBs included use of different doses, use of different proliferative stimuli, and different latency periods.]

Three studies have used PCBs as initiating agents in the Solt-Farber protocol to determine whether altered hepatic foci would develop. This protocol involves treatment with an initiating agent (either known or to be tested) in conjunction with a proliferative stimulus. After a recovery period (usually 2 weeks), rats are treated with 2-acetylaminofluorene (2-AAF; to inhibit cell proliferation), given either in the diet or by gavage, for 2 weeks, with a proliferative stimulus (usually an oral dose of carbon tetrachloride or partial hepatectomy) after the first week.

Hayes *et al.* (1985) assessed Aroclor 1254, a reconstituted human breast milk mixture of PCB congeners, PCB-47, PCB-52, and PCB-153, and found that none of them had initiating activity. Espandiari *et al.* (2003) examined less chlorinated PCBs, and observed that some (PCB-3, PCB-15, PCB-52, and PCB-77) increased the number of GGT-positive foci, while others did not (PCB-12 and PCB-38). A subsequent study showed that the PCB-3 metabolites 4-OH-PCB-3 and the *ortho* 3,4-quinone of PCB-3 acted as the proximate and ultimate carcinogens (Espandiari *et al.*, 2004). [Negative results obtained after the administration of PCBs could indicate lack of initiating activity, likely due to low metabolic activation, or could be caused by alteration of other components of the protocol, such as acetylaminofluorene metabolism and effects.]

4.4.2 Liver

Liver toxicity is commonly observed in long-term studies in rats and mice exposed to PCBs, with dose- and duration-dependent increases in the incidence, severity, and breadth of spectrum of lesions observed (Kimbrough & Linder 1974; Mayes *et al.*, 1998; NTP, 2006a, c, d, 2010).

For PCB-126, PCB-118, and binary mixtures of PCB-126 with PCB-153 or PCB-118, hepatic toxicity increased with increasing dose and duration of exposure, and was characterized by increases in the incidence and severity of hepatocyte hypertrophy (most likely due to alterations in PCB-induced CYP expression), diffuse fatty changes, multinucleated hepatocytes, pigmentation (likely due to haemosiderin accumulation), inflammation, altered hepatic foci, necrosis, oval cell hyperplasia, cholangiofibrosis, bile-duct hyperplasia, bile-duct cysts, and nodular hyperplasia (NTP, 2006a, c, d, 2010).

With PCB-153, hepatocyte hypertrophy was seen after 14, 31, and 53 weeks in female rats treated with doses of up to 3 mg/kg bw by gavage; at 2 years, there were also increases in the incidence of fatty change, bile-duct hyperplasia, oval-cell hyperplasia and pigmentation (NTP, 2006b).

While none of these hepatic responses are specifically preneoplastic, cholangiofibrosis and cholangiocarcinoma represent different diagnoses along the same continuum of pathogenesis. Cholangiofibrosis was seen in the above-mentioned NTP studies of female rats treated with specific PCB congeners by gavage, in female rats fed with Aroclor 1260 (Kimbrough *et al.*, 1975), and in female rats treated with other dioxin-like compounds by gavage (NTP, 2006e, f, g). In general, the higher the dose and duration of exposure, the higher the incidence, severity, and breadth of spectrum of responses observed. The observations of biliary and hepatocellular lesions are characteristic of an initial insult and the response of the liver to repair the injury and regenerate, leading to a hepatic stem-cell response and a bifurcating lineage of subsequent pathologies of both bile-duct cells and hepatocytes.

4.4.3 Lung

In the long-term NTP studies in female Harlan Sprague-Dawley rats treated with PCB-126, PCB-118, PCB-118 + 126 and PCB-126 + 153 by gavage, there were clear increases in the incidence of cystic keratinizing epithelium of the lung and of squamous cell carcinoma (NTP, 2006a, c, d, 2010). The two common effects seen in PCB-treated rats were an increased incidence of alveolar epithelial bronchiolar metaplasia and of squamous metaplasia of the lung (reviewed in Sells *et al.*, 2007). Squamous metaplasia was characterized by the transition of alveolar epithelial cells to squamous metaplastic cells with distortion of the normal architecture. Keratin formation was evident and inflammation was sometimes observed. The more expansive lesions formed keratinizing cysts, which consisted of a cystic structure with a thin uniform wall composed of mature squamous cells that contained various amounts of keratin. The term "cystic keratinizing epithelioma" was used for a benign neoplasm in this family of lesions, and "squamous cell carcinoma" was used as a diagnosis for the malignant form of the lesion (Sells *et al.*, 2007). Alveolar epithelial bronchiolar metaplasia was characterized by metaplasia of alveolar epithelium to respiratory type primarily at the junction of the terminal bronchioles and along alveolar ducts. Alveolar epithelial bronchiolar metaplasia did not appear to be associated with progression to neoplasia, but may have been characteristic of increased metabolic activity in the metaplastic area (Brix *et al.*, 2004).

No pulmonary toxicity was observed in a long-term NTP study with PCB-153 in female rats (NTP, 2006b). Pulmonary toxicity was not reported in long-term bioassays with Aroclors 1016, 1242, 1254, and 1260 in CD Sprague-Dawley

rats (Mayes *et al.*, 1998). [Differences between the studies included strain of rat used (Harlan Sprague-Dawley versus Charles River Sprague-Dawley), route of exposure (feed for Aroclors versus gavage for the PCB congeners), and use of complex mixtures (Aroclors) versus individual or binary mixtures of single PCB congeners.]

4.4.4 Thyroid

In long-term NTP studies of female Sprague-Dawley rats treated with PCB-126, PCB-118, PCB-126 + 118, and PCB-126 + 153 by gavage, there were increased incidences of follicular cell hypertrophy of the thyroid in the exposed groups at 14, 31, 53 weeks, and 2 years (NTP, 2006a, c, d, 2010; Yoshizawa *et al.*, 2010). Increased incidence of follicular cell hypertrophy was also seen with PCB-153 only at 53 weeks and 2 years (NTP, 2006b).

The observation of thyroid follicular cell hypertrophy in treated rats was attributed to alterations in the expression of UDP-GT in the liver, leading to a decrease in circulating T4, disruption of thyroid-hormone homeostasis, and compensatory hypertrophy (Hill *et al.*, 1989). [The Working Group noted that other mechanisms may be operational.] A persistent increase in the incidence of follicular cell hypertrophy has often been linked to increased incidences of follicular cell tumours of the thyroid in studies in experimental animals (see Section 3). No neoplasms were observed in treated females. Increased incidence of thyroid follicular cell tumours was seen in male CD SD rats exposed to Aroclors 1242, 1254, or 1260, although without significant increase in the incidence of thyroid follicular cell hypertrophy (Mayes *et al.*, 1998). The morphological appearance of the thyroid tumours was characteristic of those developed as a secondary response to chronic overstimulation of TSH. [This phenomenon is more common in males than females rats due to higher circulating levels of TSH in males.]

4.4.5 Adrenal gland

In the long-term NTP study in female Harlan Sprague-Dawley rats treated with PCB-126 by gavage, increased incidences of adrenal atrophy and cytoplasmic vacuolization were observed in those groups in which elevated incidences of adrenal adenoma were seen (NTP, 2006a). In long-term NTP studies in female rats treated with PCB-118, increases in the incidence of adrenal atrophy and cytoplasmic vacuolization, but not adrenal adenoma, were observed (NTP, 2010). Treatment with PCB-153 or Aroclors had no effect on the adrenal gland in long-term studies in female rats (Mayes *et al.*, 1998; NTP, 2006b).

4.4.6 Pancreas

A common occurrence in long-term studies with PCBs with dioxin-like activity (PCB-126, PCB-118, PCB-126 + PCB-118, and PCB-126 + PCB-153) in female rats (males were not studied) was toxicity in the pancreas (NTP, 2006a, c, d, 2010). In the NTP studies with PCB-126 and PCB-118, pancreatic acinar cytoplasmic vacuolization, atrophy, and chronic active inflammation were observed. No effect on the pancreas was seen in female rats exposed to PCB-153 at doses of up to 3 mg/kg bw per day for 2 years (NTP, 2006b). Increased incidence of acinar adenoma was observed in a long-term NTP study of PCB-126/153 in female rats, and sporadic incidences of acinar adenoma were observed in a long-term NTP study of PCB-118 in female rats, although it was uncertain whether this was a treatment-related effect (NTP, 2006c, 2010).

4.4.7 Female reproductive system

In the long-term NTP study of PCB-118 and PCB-153 in female Harlan Sprague-Dawley rats, there was no increase in the incidence of cystic endometrial hyperplasia of the uterus and squamous metaplasia of the uterus; the incidences of squamous metaplasia and cystic endometrial

hyperplasia in the core study groups were significantly less than the incidence in the vehicle-control group. In the PCB-118 stop-exposure group, in which exposure (to 4600 µg/kg bw) was for only 30 weeks followed by vehicle only (corn oil) for up to 2 years, the incidences of these two lesions were significantly increased compared with those in the core-study group exposed continually at 4600 µg/kg bw per day (NTP, 2006b, 2010). Accordingly, there was a significant increase in the incidence of uterine carcinoma in the stop-exposure group in which exposure was for only 30 weeks followed by vehicle only (corn oil) for up to 2 years, but not in the long-term exposure group (see Section 3.1.1). While the mechanism was not known, it was speculated that exposure to PCB-118 for the first 30 weeks led to the early development of responsive uterine carcinoma, and that the subsequent cessation of exposure reestablished a normal estrogenic milieu that promoted the development of these uterine neoplasms, which would otherwise have been suppressed if exposure had been continued for the full 2 years (Yoshizawa *et al.*, 2009).

In the 2-year NTP study with PCB-153 in female Harlan Sprague-Dawley rats, there was a significant increase in the incidence of chronic active inflammation of the ovary; however, there was no increase in the incidence of ovarian tumours (NTP, 2006b).

4.4.8 Skin

Chloracne and other dermal alterations are well known effects of long-term exposure to PCBs and related compounds (ATSDR, 2000). These effects have been reported in workers exposed occupationally to PCBs, and also in individuals exposed by accidental ingestion of rice oil contaminated with high concentrations of PCBs (Yusho and Yucheng), and in rhesus monkeys fed a diet containing Aroclor 1248. Chloracne is probably caused by interference of PCBs with the metabolism of vitamin A in the skin, resulting in disturbances of the epithelial tissues of the pilo-sebaceous duct (Coenraads *et al.*, 1994).

(a) Human exposure

(i) Occupational exposure

Chloracne is the most easily recognized effect of exposure to PCBs and structurally related chlorinated organic chemicals (Rice & Cohen, 1996). Chloracne first develops on the face, under the eyes and behind the ears, but severe chloracne can cover the entire body. Histologically, the lesions consist of keratinous cysts caused by squamous metaplasia of sebaceous glands. The acute stage is followed by vermiculite skin atrophy. Mild to moderate chloracne was observed in 7 out of 14 workers exposed to Aroclors (formulation not specified) at 0.1 mg/m^3 for an average duration of 14.3 months (Meigs *et al.*, 1954). [Because PCBs were used as a heat-exchange material, it is possible that these workers were exposed to pyrolysis products.] Three cases of chloracne occurred among autoclave operators (number not specified) exposed to Aroclor 1254 at 5.2–6.8 mg/m^3 for 4–7 months (Bertazzi *et al.*, 1987). [The presence of pyrolysis products may have been a confounding factor.] In 1977, four more cases of chloracne were diagnosed among 67 workers from the same plant who were engaged in impregnating capacitors with Pyralene 3010 (0.048–0.275 mg/m^3) and had skin contact confirmed as a major exposure route. An increased incidence of non-adolescent acneiform eruptions was reported in workers exposed to various Aroclors at mean concentrations of 0.007–11 mg/m^3 for > 5 years; 40% of the workers had been exposed for > 20 years (Fischbein *et al.* 1979, 1982). Maroni *et al.* (1981a, b) reported ten cases of acne and/or folliculitis and five cases of dermatitis among 80 capacitor-manufacturing workers in Italy. All the workers with chloracne were employed in jobs with high exposure. Their blood PCB concentrations ranged from 300 to 500 µg/L. No definite association was found

between chloracne and blood PCB concentrations. Other dermal effects reported in workers included skin rashes, pigmentation, disturbances of skin and nails, erythema and thickening of the skin, and burning sensations (Ouw *et al.*, 1976; Fischbein *et al.*, 1979, 1982; Smith *et al.*, 1982). In these studies, the workers were exposed to various Aroclors at concentrations as low as 0.003 mg/m^3 for > 5 years. In those studies that looked at PCB profile of exposure, statistically significant associations between dermatological effects and plasma concentrations of more highly chlorinated PCB congeners were reported (Fischbein *et al.*, 1979, 1982; Smith *et al.*, 1982), while no relationships were found between the incidence of skin rash or dermatitis, and plasma concentrations of less chlorinated PCBs (Smith *et al.*, 1982).

(ii) Accidental exposure

Skin effects were widely reported among victims of the Yusho and Yucheng poisoning episodes (Lü & Wu, 1985; Kuratsune, 1989; Rogan, 1989; Guo *et al.*, 1999). However, these effects could not be attributed solely to exposure to PCBs, since the victims were also exposed to PCDFs and other chlorinated chemicals (ATSDR, 1994). Characteristic skin changes included marked enlargement, elevation and keratotic plugging of follicular orifices, comedo formation, acneform eruptions, hyperpigmentation, hyperkeratosis, and deformed nails. Dark-coloured pigmentation frequently occurred in the gingival and buccal mucosa, lips, and nails, and improved only gradually in most patients (Kuratsune *et al.*, 1971; Fu, 1984; Lü & Wu, 1985; Kuratsune, 1989; Rogan, 1989). At 14 years after the Yucheng incident, exposed men and women had a higher lifetime prevalence of chloracne, abnormal nails, hyperkeratosis, and gum pigmentation (Guo *et al.*, 1999). Skin lesions were commonly observed in children born to mothers exposed during the Yusho or Yucheng incidents (Gladen *et al.*, 1990).

(b) Experimental systems

(i) Animal studies in vivo

Female rhesus monkeys fed diets containing Aroclor 1248 at concentrations of 2.5 and 5.0 ppm developed facial oedema, swollen eyelids, erythema, loss of hair, and acne, within 2 months. After 6 months, the monkeys were bred with control males. In the seven offspring carried to term, and exposed for 4 months to PCBs via the lactating mother, focal areas of hyperpigmentation and acneiform lesions of the face developed within 2 months, and were accompanied by increased skin PCB concentrations (Allen & Norback, 1976).

Developing *Xenopus laevis* tadpoles were exposed to Aroclor 1254 at concentrations of 0 to 100 µg/mL from day 5 to day 9 after fertilization. Exposure at the higher concentrations (10, 50, and 100 µg/mL) caused statistically significant reductions in survival and body size, and resulted in histological abnormalities, including aberrant tail-tips, and aberrant myotomal and melanocyte morphologies; tadpoles treated with Aroclor 1254 were devoid of dendritic arborizations, resulting in decrease in total melanocyte area (Fisher *et al.*, 2003).

(ii) Human cells in vitro

Only two studies were available on the molecular effects of PCBs in human skin cells. Exposure of normal human melanocytes to TCDD resulted in activation of the AhR signalling pathway, AhR-dependent induction of tyrosinase, and consequently, elevated total melanin content. These effects were due to the induction of tyrosinase and tyrosinase-related protein 2-gene expression. Thus AhR is able to modulate melanogenesis by controlling the expression of melanogenic genes (Luecke *et al.*, 2010).

Exposure of human skin keratinocytes to a synthetic mixture of volatile PCBs, or the common airborne congeners PCB-28 or PCB-52

led to significant inhibition of telomerase activity and reduced telomere length. All PCBs decreased cell proliferation, and PCB-52 produced a small increase in the fraction of cells arrested in G0/G1 of the cell cycle. Changes in telomere length and telomerase activity are hallmarks of ageing and carcinogenesis; these effects suggested a potential mechanism by which exposure to PCBs could lead to skin cancer (Senthilkumar *et al.*, 2011).

4.5 Susceptibility

4.5.1 Genetic polymorphisms

Single nucleotide polymorphisms in the genes for metabolizing enzymes or receptors can potentially affect expression or inducibility (if these polymorphisms were in the promoter region of the gene), and stability or function of the protein (if they were in the coding region). The individual response to carcinogens may be influenced by polymorphisms in genes for metabolizing enzymes, including xenobiotic- and steroid-metabolizing CYP, GST, catechol *O*-methyltransferase (COMT), and others (Singh *et al.*, 2008); receptors that control expression of metabolizing enzymes such as AhR (Ng *et al.*, 2010) and the AhR repressor (Hung *et al.*, 2013); and receptors that interact with endogenous molecules such as steroid hormones.

(a) Metabolizing genes

As discussed in Section 4.1.3, CYP plays an important role in PCB metabolism. Knowledge of the particular CYP isoform most likely to bind and/or metabolize a PCB congener is important in evaluating risk from exposure to this congener. Many human CYP isoforms exhibit pharmacogenetic polymorphisms, which can affect expression levels, catalytic activity per unit enzyme with particular substrates, or both parameters (Ingelman-Sundberg *et al.*, 2007). Variations in activity due to polymorphism could lead to inter-individual differences in the capacity to metabolize particular congeners. If metabolism of the congener produced genotoxic metabolites, such as arene oxides, quinones, or reactive oxygen species through the action of CYP, this could mean that greater amounts of these potential carcinogens would be formed in some individuals with increased metabolic activity. Alternatively, people with a lower metabolic activity for some PCBs could accumulate greater amounts of those PCBs, if continually exposed. Both scenarios could lead to increased risk of cancer, through several mechanisms.

(i) Cancer of the breast

Epidemiological studies have provided evidence for increased risk of cancer of the breast in women with a particular genetic polymorphism in the *CYP1A1* gene and high serum PCB concentrations (Moysich *et al.*, 1999; Laden *et al.*, 2002; Charlier *et al.*, 2004; Zhang *et al.*, 2004; Li *et al.*, 2005). In the variant form, *CYP1A1*2C*, also called the m2 variant, has valine substituted for isoleucine at position 462 near the *C* terminus of the protein (Persson *et al.*, 1997). This variant is found in 10–15% of the white population and in a larger proportion of African-Americans (reviewed in Brody *et al.*, 2007). Persson *et al.* (1997) reported that the activity per unit enzyme of this variant, measured in vitro, was similar to that of wildtype *CYP1A1*. Polymorphisms in AhR, or its repressor, that influence the expression of *CYP1A1* may be more important than *CYP1A1* genotype in determining the in-vivo activity of *CYP1A1* (Smart & Daly, 2000; Hung *et al.*, 2013).

Among postmenopausal patients with cancer of the breast in western New York state, USA, the incidence of cancer of the breast was higher in women with total PCB concentrations (73 congeners) of 3.73–19.04 ng/g of serum and the *CYP1A1*2C* polymorphism than in women with lower PCB concentrations or wildtype *CYP1A1* (Moysich *et al.*, 1999). In a study of Caucasian women in Connecticut, USA, in which serum

concentrations of PCB-74, PCB-118, PCB-138, PCB-153, PCB-156, PCB-170, PCB-180, PCB-183, and PCB-187 were measured, cancer of the breast was more prevalent in postmenopausal women with lipid-adjusted serum concentrations of 611–2600 ng/g and the *CYP1A1*2C* polymorphism than in controls (Zhang *et al.*, 2004). If the *CYP1A1* polymorphism was absent (homozygous wildtype alleles), there was no effect of serum PCB concentration on incidence of cancer of the breast. An epidemiological study of African-American and white women in North Carolina, USA, examined lipid-adjusted total plasma PCB concentrations, *CYP1A1* polymorphism, and risk of cancer of the breast (Li *et al.*, 2005). Although results were not conclusive due to small sample size, premenopausal white women with cancer of the breast were more likely to have total PCB concentration > 0.35 ng/mL serum and the *CYP1A1*2C* polymorphism than were controls, while there was no relationship between cancer of the breast in women with total PCB concentration < 0.35 ng/mL serum or lacking this polymorphism. In the African-American women, total PCB concentrations were somewhat higher (≥ 0.430 ng/mL), and the *CYP1A1*3* polymorphism was more prevalent in pre- and postmenopausal patients with cancer of the breast (Li *et al.*, 2005).

Another study found a non-significantly elevated risk of cancer of the breast among women with the CYP1A1-m1 variant and high serum PCB concentrations (McCready *et al.*, 2004).

(ii) Cancer of the testis

Data from 568 cases of testicular cancer and 698 controls enrolled in the United States Servicemen's Testicular Tumor Environmental and Endocrine Determinants Study were used to examine associations between testicular germ cell tumours (TGCT) and exposure to PCBs, as affected by polymorphisms in several hormone-metabolizing genes, i.e. *CYP17A1*, *CYP1A1*, *HSD17B1*, *HSD17B4* and androgen receptor. Among these, the polymorphism rs384346 in *HSD17B4* modified the association of TGCT risk with PCB-118 and PCB-138 concentrations. Among men who were homozygous for the major allele genotype, there was a statistically significant dose-dependent reduction in risk (P for trend, < 0.001) with higher exposure to PCB-118 and PCB-138. Men in the highest quartile of PCB-118 exposure had an almost 50% reduction in risk of TGCT (OR, 0.46, 95% CI, 0.31–0.70) compared with men in the lowest quartile; similar results were seen for PCB-138. For any minor allele of this *HSD17B4* polymorphism, there were no associations between PCB-118 and PCB-138 concentrations and risk of TGCT. No interactions between other PCB congeners of interest (PCB-153, PCB-156, PCB-163, PCB-170, PCB-180, and PCB-187) and enzyme polymorphism were observed (Chia *et al.*, 2010).

(b) Polymorphisms in other genes

Among highly exposed Yucheng patients, combined *CYP1A1-Msp1* mutant genotype and *GSTM1-null* genotype were associated with an increased risk of chloracne (OR, 2.8; 95% CI, 1.1–7.6). Among intermediately exposed individuals, the *GSTM1-null* genotype was associated with skin allergy (Tsai *et al.*, 2006).

Patients with non-Hodgkin lymphoma and PCB-118 concentrations in the highest quartile (> 12.85–202.13 µg/L plasma) were more likely to have a polymorphic variant of AhR (IVS + 4640 null; G/G genotype) than controls, although the effect was not strong and was also related to highest levels of oxychlordane and *trans*-nonachlor (Ng *et al.*, 2010).

Among women with cancer of the breast who carried a variant of the tumour-suppressor gene *TP53*, total PCB exposure in the highest quartile was associated with an increased risk of cancer of the breast, but this was not statistically significant (OR, 3.0; 95% CI, 0.66–13.62) (Høyer *et al.*, 2002).

4.5.2 Exposure in utero, postnatally, and of children

PCBs can pass through the placenta during embryonic development and accumulate in breast milk. In addition, compared with adults, children have a lower barrier to absorption through the skin, gastrointestinal tract and lungs, and lower levels of detoxifying enzymes (Lindström *et al.*, 1995). A combination of all these factors leads to a higher accumulation of PCBs in children.

(a) Toxicokinetics and distribution in tissues

(i) Children

Grandjean *et al.* (2008) studied the elimination kinetics of PCBs in two groups of children with elevated PCB concentrations due to breastfeeding. Children were followed from age 4.5 to 7.3 years (99 subjects) and 7 to 14 years (101 subjects). Subjects with exposures above the median and in the highest quartile showed half-lives of about 3–4 years for PCB-138; 4.5–5.5 years for PCB-105 and PCB-118; 6.5–7.5 years for PCBs 156, 170 and 187; and 7–9 years for PCBs 153 and 180. The longest half-lives correspond to elimination of the parent PCB solely with a daily fat excretion rate of 1–2 g, while shorter half-lives assume metabolic break-down.

Scheele *et al.* (1992) measured the concentrations of PCB-138, PCB-153, and PCB-180 in bone marrow (collected during routine bone-marrow aspiration) of 38 children with leukaemia and 15 control children (nine had idiopathic thrombocytopenia and six were bone-marrow donors). Most of the samples were pooled to ensure sufficient volume for analysis. Total PCB concentrations were determined on the basis of congeners PCB-138 + PCB-153 + PCB-180 and multiplied by 1.7 (Deutsche Forschungsgemeinschaft, 1988). The mean and median concentrations of total PCBs in bone marrow of children were 3.6 mg/kg fat basis and 2.9 mg/kg, respectively. PCB concentrations in bone marrow were two- to threefold those in fat tissue. [The reason for the high affinity of bone marrow for PCBs was not clear. It is possible that genetic factors may play a role.] There were no significant differences in PCB concentrations between the group of children with leukaemia and the control group. [The Working Group noted that the authors did not report whether parental smoking, an important confounding factor, was accounted for in their statistical analysis.]

A study in 360 schoolchildren (a subgroup of the Hesse, Germany cohort) in 1995 (Karmaus *et al.*, 2001a, b) focused on the potential of early childhood factors such as breastfeeding, parity, and parental smoking to contribute to the variety of effects observed with exposure to organochlorine compounds including PCBs, at approximately age 7 years. Concentrations of PCBs (sum of congeners PCB-101, PCB-118, PCB-138, PCB-153, PCB-170, PCB-180, PCB-183, and PCB-187) were determined in whole blood. A significant dose–dependent relationship ($P < 0.0001$) existed between the duration of breastfeeding (none, 1–4 weeks, 5–8 weeks, 9–12 weeks, > 12 weeks) and the sum of PCB concentrations. Of all the potential factors analysed, breastfeeding accounted for most of the variance in PCB concentrations. Exclusive breastfeeding beyond 12 weeks was associated with a doubling of PCB concentrations in whole blood compared with bottle-fed children (sum of PCBs, 0.25 µg/L versus 0.55 µg/L).

(ii) Experimental animals

Sixteen (eight/group) adult female rhesus monkeys were exposed to diets containing Aroclor 1248 at 2.5 or 5.0 ppm for approximately 1.5 years (Allen & Barsotti, 1976). Six out of the eight monkeys treated with Aroclor 1248 at 5.0 ppm, and eight out of the eight monkeys at 2.5 ppm were successfully bred after 6 months of exposure. One live infant was born to dams exposed at 5.0 ppm, and five infants were born to monkeys at 2.5 ppm. Infants were permitted to nurse with their mothers. All six surviving infants had PCBs

in their tissues at birth: PCB concentrations in skin biopsies (epidermis, dermis and the attached underlying subcutaneous tissue) ranged from 1.0 to 4.8 µg per g of tissue. By the third month, skin PCB concentrations ranged from 86.4 to 136.8 µg per g of tissue. The infant that died after 239 days had PCB concentrations of more than 20 µg per g in seven organs (adrenal gland, cerebrum, kidney, muscle, pancreas, testes, thymus). In the two infants that survived for shorter periods, this PCB concentration was exceeded only in three tissues (bone marrow, lung, thymus) in one infant and two tissues (bone marrow, pancreas) in the other.

Female rhesus monkeys were fed a daily dose of Aroclor 1254 (0, 5, 20, 40 or 80 µg/kg bw) for approximately 6 years (Arnold *et al.*, 1993, 1995; Mes *et al.*, 1994, 1995a). Blood and adipose tissue from offsprings exposed in utero/during lactation who had nursed for 22 weeks were analysed for PCB content at 120 weeks after birth. PCB concentrations in the adult monkeys increased with their dosage. Tissues of live infants of dosed dams contained more PCBs than those of infants of control dams, and less PCBs than those of stillborn infants. Also, offspring with higher PCB concentrations showed a marked shift from tetra- and hexachlorobiphenyls to penta- and heptachlorobiphenyls. The PCB distribution pattern in tissues from a dosed mother–infant pair differed considerably. A larger percentage of heptachlorobiphenyls was found in the infant than in its dam (Mes *et al.*, 1995a). Depletion studies revealed that PCB concentrations in the blood of exposed infants declined rapidly after weaning due to growth dilution and approached maternal levels within 40–50 weeks. Approximately 100 weeks after weaning, PCB concentrations in adipose tissue of infants from treated dams reached levels of those in the control group (Mes *et al.*, 1994).

Male Swiss mice aged 8 days were given a single intraperitoneal injection of a mixture of PCB-99, PCB-105, PCB-118, PCB-128, PCB-138, PCB-153, PCB-156, PCB-170, and PCB-180 at 500 mg/kg bw (Anderson *et al.*, 1993). Groups of 25 mice were killed at 1 and 7 days, and at 8, 12, and 16 weeks after treatment. Congeners in group 1 (PCB-99, PCB-105, PCB-118, PCB-128) were eliminated from the body more rapidly than congeners in group 2 (PCB-138, PCB-153, PCB-156, PCB-170, PCB-180). PCB concentration in the carcass (adipose compartment) was the most predictable finding, since the congeners behaved similarly within each group. In contrast, in lung, after a rapid loss during the first week, all congeners except PCB-153 were retained and decreased in amount only as a function of dilution due to growth. Congeners PCB-105 and PCB-138 were present at higher proportions in the lung than in the carcass. In the liver, retention of all congeners was observed during the prepubertal growth phase, with specific enrichment of PCB-105, followed by more rapid depletion of certain congeners (Anderson *et al.*, 1993).

(b) Effect on gene expression

Dutta *et al.* (2012) used microarray-based differential gene expression analysis of a group of children (mean age, 46.1 months) of central European descent (Slovak Republic) to study the impact of PCBs on different cellular pathways and to explain their possible mode of action. The subset of children having high blood PCB concentrations (> 75th percentile) was compared with their low PCB counterparts (< 25th percentile), with mean lipid-adjusted PCB concentrations of 3.02 ± 1.3 and 0.06 ± 0.03 ng/mg of serum lipid, respectively. A set of 162 genes with statistically significant differential expression ($P < 10^{-5}$) between groups with high and low PCB concentration was identified. Analyses using the IPA tool indicated that cell–cell signalling and interactions, cellular movement, cell signalling, molecular transport, and vitamin and mineral metabolism were the major molecular and cellular functions associated with the genes differentially expressed in children with high PCB concentrations. Furthermore, the

differential gene expression appeared to play a pivotal role in the development of probable diseases and disorders, including cardiovascular disease and cancer. The analyses also pointed out possible organ-specific effects, e.g. cardiotoxicity, hepatotoxicity and nephrotoxicity in the children exposed to high concentrations of PCBs. Expression levels of *BCL2*, paraoxonase 1 (*PON1*), interleukin *IL1F7*, *IL23A* and integrin β 1 (*ITGB1*) were significantly altered in these children; more specifically, *BCL2* and *ITGB1* were downregulated, while *IL1F7*, *PON1*, and *IL23A* were upregulated.

(c) Enzymatic effects in feto-placental unit, and fetal and neonatal liver

Alvares & Kappas (1975) investigated the induction of aryl hydrocarbon hydroxylase (Ahh) by PCBs in the feto-placental unit, fetal livers and neonatal livers during lactation. For the in-utero exposure protocol, pregnant Sprague-Dawley rats were injected intraperitoneally with Aroclor 1254 (25 mg/kg bw per day) for 6 days, and killed 24 hours later on day 20 of gestation. For the lactation experiments, untreated mothers were injected intraperitoneally with Aroclor 1254 (25 mg/kg bw per day) for 6 days starting on day 2 postpartum; the offspring of these dams were killed on day 8 postpartum.

PCBs caused a 10-fold induction in Ahh activity in the placenta, but only a threefold induction in the fetal livers. Ahh activity in placentas of untreated rats was markedly lower than that observed in the fetal liver of the same rats. In the liver of neonates whose mothers were treated with Aroclor 1254 postpartum (infants exposed through lactation), there was an 18-fold increase in Ahh activity, a threefold increase in CYP content, and a twofold increase in *N*-demethylase activity. Thus Aroclor 1254 was a more potent inducer of Ahh activity in placenta and liver when exposure occurred through lactation than through in-utero exposure when administered to pregnant rats.

4.6 Mechanistic considerations

The group of PCBs comprises 209 individual congeners with widely different physical and chemical properties. The number of chlorine atoms on the two phenyl rings and their relative positions determine the biological and toxicological attributes of each congener. Some PCBs are susceptible to metabolic conversion, which may give rise to a series of metabolites, each with its own profile of biological and toxicological activities. In this section, various mechanisms of carcinogenesis will be identified and summarized for specific subgroups of PCBs and their metabolites.

4.6.1 Metabolic activation and genotoxicity of PCBs and their metabolites

(a) Metabolism leading to formation of electrophiles

The 209 PCB congeners vary greatly in their susceptibility to metabolic attack, with less chlorinated biphenyls being much more susceptible. The first metabolic step is mono-oxygenation, which leads to the formation of hydroxylated metabolites, a reaction that is mediated by enzymes of the CYP super-family. There are 837 possible mono-hydroxylated products (Rayne & Forest, 2010; Grimm *et al.*, 2015). Depending on the number of chlorines present, the arene oxide may emerge as a highly reactive, electrophilic species: the lower the number of chlorines, the more reactive the arene oxide.

Mono-hydroxylated PCBs may undergo a second hydroxylation, producing a dihydroxylated PCB derivative, either as catechol (hydroxyl groups in the *ortho* configuration) or as hydroquinone (hydroxyl groups in the *para* configuration) (McLean *et al.*, 1996a). The formation of dihydroxylated PCBs is catalysed primarily by CYP enzymes. PCB catechols and hydroquinones may then be oxidized by peroxidases, prostaglandin synthase, and probably other enzymes,

giving rise to the formation of highly reactive electrophilic PCB quinones (Amaro *et al.*, 1996; Oakley *et al.*, 1996a; Wangpradit *et al.*, 2009).

The oxygenated PCB intermediates and metabolites, i.e. the arene oxides and the quinones, are probably the most relevant to PCB-induced carcinogenesis, but many other metabolites may also be formed. For example, OH-PCBs are substrates for glucuronidation (Tampal *et al.*, 2002) and sulfation (Liu *et al.*, 2006, 2009; Ekuase *et al.*, 2011). All electrophilic PCB metabolites with elevated chemical reactivity, however, should be regarded as probable cancer initiators.

(b) Binding to DNA and protein

Covalent binding to cellular macromolecules (adduct formation) has been observed in mice treated with radiolabelled PCB-153 and PCB-136, the binding of the latter decreasing in the order RNA > protein > DNA (Morales & Matthews, 1979). Formation of protein and DNA adducts was observed in vitro with PCB-3 and the tetrachlorinated congeners PCB-47, PCB-49, PCB-52, and PCB-77 (Wyndham *et al.*, 1976; Shimada & Sawabe, 1984). DNA-adduct formation was also observed with a series of 15 mono- and dichlorinated PCBs, with but not without activation by microsomes, horseradish peroxidase and hydrogen peroxide (McLean *et al.*, 1996b). This suggested that quinones were the ultimate genotoxic agents. Indeed, tests with synthetic quinones of less chlorinated PCBs confirmed the extensive DNA-adduct formation, particularly with deoxyguanosine (Oakley *et al.*, 1996a; Zhao *et al.*, 2004).

These experiments indicated that PCBs require CYP-mediated metabolic activation, that a lower degree of chlorination favours bioactivation, that an arene oxide intermediate and/or possibly a semiquinone or quinone is the ultimate DNA-binding species, and that guanine is the major target site in DNA. Apart from binding to DNA, PCB quinones also bind cellular proteins, preferably, but not exclusively, to cysteine (Amaro *et al.*, 1996; Srinivasan *et al.*, 2002; Bender *et al.*, 2006).

(c) Indirect genotoxicity: metabolism-associated generation of ROS

The arene oxides and quinones are probably the metabolites with most relevance to the cancer-initiating activity of PCBs, since they can be regarded as direct-acting genotoxic intermediates. In addition, dihydroxylated PCBs and their corresponding PCB quinones may undergo redox cycling, thereby generating ROS, which are considered to be active in the initiation, promotion, and progression of cancer. For example, ROS formed during auto-oxidation of a PCB hydroquinone may give rise to oxidative DNA damage, e.g. 8-OHdG. Mutations induced by these lesions may lead to activation of oncogenes or inhibition of tumour-suppressor genes, thus contributing to the carcinogenic potential of PCBs (Amaro *et al.*, 1996; Oakley *et al.*, 1996a). Formation of ROS may also induce DNA strand breaks (Srinivasan *et al.*, 2001).

(d) Mutagenic effects

PCB-3, 4-OH-PCB-3, and two hydroquinones of PCB-3 were tested for mutagenicity in Big Blue® rats and in Chinese hamster V79 cells. These results demonstrated that monochlorinated PCBs are mutagenic in vivo in the target organ, the liver, and studies in vitro suggested that metabolic activation to electrophilic and mutagenic species plays a crucial role. Although the ultimate mutagenic metabolite (*ortho*- or *para*-quinone, or epoxide or other metabolite) could not be deduced with certainty, the evidence pointed towards adduct formation by a quinone, or quinone-induced redox cycling as the mode of action.

Apart from gene mutations, other forms of genotoxicity observed after exposure to PCBs included the induction of DNA strand breaks, and anomalous segregation of chromosomes.

Elevated concentrations of mono- and dihydroxylated metabolites of PCB-3 were shown to induce these types of lesions in vitro (Zettner *et al.*, 2007; Flor & Ludewig, 2010).

With regard to the PCB congeners considered to act primarily through *trans*-activation of nuclear receptors, the available data provided little evidence regarding genotoxicity (see Section 4.2).

(e) Cancer initiation and promotion

The ability of commercial PCB mixtures and individual PCB congeners to initiate and/or promote neoplastic lesions has been studied in rodent two-stage models of liver carcinogenesis. Aroclor 1254, which contains mainly tetra- and pentachlorobiphenyls, acted as a weak tumour initiator in the mouse two-stage model of skin carcinogenesis (DiGiovanni *et al.*, 1977). In contrast, when tested using the Solt-Farber protocol, Aroclor 1254 and the PCB-153, PCB-52, and PCB-47 did not produce a positive response in male F344 rats (Hayes *et al.*, 1985). No nodules were apparent in animals receiving PCB-12 (dichloro-) or PCB-138 (trichloro-) as initiator, while PCB-3 (mono-chlorinated) induced clearly visible nodules in 50% of the exposed rats (Espandiari *et al.*, 2003). Thus less chlorinated PCBs seem to be able to initiate hepatocarcinogenesis in the rat, but in view of the small number of congeners tested, a clear structure–activity relationship could not be established.

A series of synthetic oxygenated metabolites of PCB-3 were studied with respect to focus formation in rat liver. Test compounds included the 2-OH-, 3-OH-, 4-OH-, 2,3-dihydroxyl-, 3,4-dihydroxyl-, 2,5-dihydroxyl-, 2,3-quinone, 3,4-quinone, and 2,5-quinone metabolites of PCB-3. The 4-OH- and 3,4-quinone metabolites significantly increased focus number and focus volume, while none of the other metabolites had a significant effect on either parameter (Espandiari *et al.*, 2004, 2005). The 3,4-*ortho*-quinone of PCB-3 was the initiating metabolite, and that PCB-3 is metabolized in rat liver in vivo to yield this ultimate carcinogenic species.

(f) Direct and indirect endocrine disruption

After the liver, the thyroid gland is the second major target of the toxicity of PCBs. In rats, exposure to PCBs produced an increase in the mass and/or volume of the thyroid gland, and in the number of thyroid neoplasms (Mayes *et al.*, 1998). Both these changes may be linked to the PCB-driven reduction in serum T4 concentrations, a commonly measured effect of PCBs (Knerr & Schrenk, 2006; Pearce & Braverman, 2009). Suggested mechanisms include: (a) PCB-induced alterations in the structure and function of the thyroid gland; (b) PCB-induced alterations in thyroid-hormone metabolism, biliary excretion of T4-glucuronide (Martin *et al.*, 2012), and effects on de-iodonase activity; and (c) interference with the transport of T4. OH-PCBs are competitors for the T4-binding site in the transport protein TTR (Brouwer *et al.*, 1998; Gutleb *et al.*, 2010), with binding affinities up to an order of magnitude stronger than that of the natural ligand, T4 (Chauhan *et al.*, 2000). The sulfate conjugates of OH-PCBs also bind to TTR, with affinities similar to that of T4 (Grimm *et al.*, 2012).

Circulating steroid and thyroid hormones are sulfated by sulfotransferases, which is an important feature of their homeostatic control. Since OH-PCBs are both substrates and inhibitors of these enzymes, they may directly influence the circulating levels of steroids and thyroid hormones by affecting the rates of sulfation (Schuur *et al.*, 1998b, c; Kester *et al.*, 2000; Liu *et al.*, 2009; Ekuase *et al.*, 2011).

OH-PCBs have both estrogenic and anti-estrogenic properties (see Section 4.3.3).

4.6.2 Receptor-driven effects of PCBs and their metabolites

PCBs and their metabolites may bind to and activate a wide range of cellular receptors, as illustrated in Table 4.8. Activation of AhR, CAR, and other receptors results in extensive modulation of expression of genes involved in cell-cycle control, cell proliferation, apoptosis, cell–cell communication, cell adhesion and migration, the pro-inflammatory response, and endogenous metabolism. Deregulation of those processes is directly associated with carcinogenesis, i.e. tumour promotion and progression (see Sections 4.3.1 and 4.3.2). The most significant events include modulation of cell proliferation, suppression of apoptosis (i.e. survival of initiated cells), impaired plasma-membrane function and plasma membrane-mediated signal transduction (i.e. modulation of cell plasticity, cell–cell communication, adhesion and migration) and induction of proinflammatory mediators. In part, induction of cell proliferation may be a consequence of cytotoxicity and tissue injury – after biotransformation processes, oxidative stress, etc. – and is considered regenerative cell proliferation (see Section 4.3.2).

In addition, disruption of endocrine function, due to interaction of PCBs or their metabolites with steroid and thyroid hormone receptors and serum proteins, or as a result of changes in biosynthesis and catabolism of steroids, may be linked to cancer development in hormone target tissues (see Section 4.3.3). Receptor-mediated gene expression is also linked to induction of proinflammatory processes and immunotoxic effects (see Sections 4.3.4 and 4.3.5).

(a) Induction of xenobiotic metabolism

Many highly chlorinated PCB congeners are potent inducers of enzymes involved in the metabolism of xenobiotics (Parkinson *et al.*, 1983) via binding to AhR (Bandiera *et al.*, 1982). Efficient induction has been reported of a wide spectrum of enzymes, notably certain CYP-dependent mono-oxygenases of the CYP1A subfamily, as well as CYP2Bs and microsomal epoxide hydrolase (Parkinson *et al.*, 1983), glutathione transferases, and UDP-glucuronosyl transferases (for a review, see Parkinson *et al.*, 1980).

Individual PCB congeners that showed the strongest binding to the AhR were identified as those in which the chlorines are in the *meta* and *para* positions of the phenyl rings in the absence of *ortho* chlorines (see Section 1.1.1). These PCBs are referred to as "coplanar" or "dioxin-like," typical examples being PCB-77, PCB-126, and PCB-169. Other PCBs, characterized by substitution in the *ortho* and *para* positions of the phenyl rings (e.g. PCB-153), activate CAR. PCBs in this group induce CYP2B1/2 and other enzymes, and as such resemble the drug phenobarbital (Parkinson *et al.*, 1983). Many PCBs that activate CAR also activate the pregnane X receptor (Holsapple *et al.*, 2006). PCBs that have one chlorine in the *ortho* position may be mixed-type inducers of CYPs, for example PCB-118, which induces members of the CYP1A and the CYP2B subfamilies.

Exposure to PCBs may alter the metabolic status in the liver, which will change the metabolism of endogenous or other exogenous compounds. For example, PCBs induce CYPs in the liver, which may redirect the metabolism of endogenous estrogen to more harmful estrogen catechols (Ho *et al.*, 2008), or generate ROS that produce estrogen quinones (Brown *et al.*, 2007).

(b) Immunomodulation

The biochemical events leading to the observed PCB-induced immunomodulation have not been completely elucidated. Studies on structure–activity relationships, and structure–toxicity relationships have demonstrated that some of the PCBs share a common mechanism of action with other structurally related halogenated aromatic hydrocarbons such as dioxins and dibenzofurans (Safe, 1990). These studies

Table 4.8 PCBs and metabolites as ligands for cellular and nuclear receptors

Receptor		Ligands	Gene or function affected	References
AhR	Aryl hydrocarbon	Coplanar, ***meta***-, ***para***-PCBs	CYP1A activation	Bandiera *et al.* (1982)
CAR	Constitutive androstane receptor	*Ortho*-, *para*-PCBs	CYP2B activation	Denomme *et al.* (1983), Al-Salman & Plant (2012)
PXR	Pregnane X receptor	Multi-*ortho*-PCBs, PCB-47, PCB-184; PCB-138, PCB-153, PCB-180, PCB-194	CYP3A activation	Schuetz *et al.* (1998), Al-Salman & Plant (2012)
PPAR	Peroxisome proliferator receptor	Coplanar, *meta*-, *para*-PCBs	CYP4A, repression	Hennig *et al.* (2005), Robertson *et al.* (2007)
RyR	Ryanodine receptor	Non-dioxin-like-PCBs (optimal configuration, multi-*ortho*, *para*-PCBs), OH-PCBs, catechols, $MeSO_2$-PCBs	Ca^{2+}-channel	Pessah *et al.* (2006)
ER	Estrogen receptor	Multi-*ortho*-PCBs, OH-PCBs	Agonism and antagonism	Connor *et al.* (1997), Arcaro *et al.* (1999); Bonefeld-Jørgensen *et al.* (2001), Plísková *et al.* (2005), Hamers *et al.* (2011)
AR	Androgen receptor	Multi-*ortho*-PCBs	Antagonism	Portigal *et al.* (2002), Fang *et al.* (2003); Schrader & Cooke (2003), Hamers *et al.* (2011)
PR	Progesterone receptor	OH-PCBs	Antagonism	Connor *et al.* (1997)
TH	Thyroid hormone	PCBs, OH-PCBs	Disruption of thyroid receptor-dependent gene expression	Gauger *et al.* (2004), Miyazaki *et al.* (2004)
DAT or VMAT	Dopamine active transporter or vesicular monoamine transporters	Coplanar and multi-*ortho*-PCBs	Decrease or increase in dopamine levels	Bemis & Seegal (2004), Richardson & Miller (2004), Seegal *et al.* (2005)
GR	Glucocorticoid receptor	$MeSO_2$-PCBs, OH-PCBs, PCB-28, PCB-153, PCB-118	Competitive antagonism	Johansson *et al.* (1998), Bovee *et al.* (2011), Antunes-Fernandes *et al.* (2011)

$MeSO_2$-PCB, methyl sulfonyl PCB; OH-PCB, hydroxylated PCB; PCB, polychlorinated biphenyl
Adapted from Ludewig *et al.* (2007)

indicated that certain immunotoxic effects seen with dioxin-like PCB congeners depend on the presence of AhR, which regulates the synthesis of a variety of proteins (Safe, 1990). AhR is present in several tissues and cells of the immune system as shown in rodents (e.g. Mason & Okey, 1982), in non-human primates (Van Der Burght *et al.*, 1998) and in humans (Hakkola *et al.*, 1997).

AhR is present in several tissues and cells of the immune system in animals and in humans. Binding of PCBs to AhR is a prerequisite for some of the immunotoxic effects of the DL-PCBs (reviewed in Silkworth *et al.*, 1984; Safe, 1990). TEFs were calculated for individual PCB congeners and several commercial PCB products, based on the suppression of the response in a challenge test against sheep erythrocytes (SRBC) – a parameter predictive of effects on humoral immunity (Davis & Safe, 1989, 1990). Highly chlorinated commercial PCB products, including Aroclors 1260, 1254, and 1248 have higher TEFs, while lower TEFs were calculated for the less chlorinated Aroclors 1242, 1016, and 1232.

Clearly some PCBs produce their immunotoxic effects by binding to AhR present in tissues and cells of the immune system, while others may follow different pathways and produce similar effects. Furthermore, individual congeners in commercial PCB products may antagonize each other's effects by mechanisms that have not been fully elucidated (see Section 4.3.4).

Overproduction of IL-6 has been shown to be responsible for the pathogenesis of inflammation-associated colorectal cancer (Waldner *et al.*, 2012). Furthermore, activation of NF-κB, a hallmark of inflammatory responses, plays a fundamental role in the formation and development of malignant tissue changes caused by inflammation, and is thought to function as a tumour promoter in inflammation-associated cancer (Pikarsky *et al.*, 2004; Karin, 2006).

(c) Interference with endogenous transport by PCBs and their metabolites

Endogenous substances such as vitamins, metals, steroids, and hormones are transported throughout the body by virtue of their binding to serum proteins. Substances that interfere with these processes can severely impair their tissue availability. Notable examples are the ability of PCB metabolites to interfere with vitamin A homeostasis and T4 transport (Grimm *et al.*, 2012), and steroid metabolism (see Section 4.3.3).

Overall, PCBs can induce formation of ROS, genotoxic effects, immune suppression, inflammatory responses, and endocrine effects to various extents and through different pathways. DL-PCBs exert their effects mainly through activation of AhR and the downstream cascade of related events; less chlorinated PCBs act more readily through metabolic activation and the ensuing effects involving their metabolites.

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5. SUMMARY OF DATA REPORTED

5.1 Exposure data

Polychlorinated biphenyls (PCBs) are a class of aromatic chemical compounds in which some or all hydrogen atoms attached to the biphenyl nucleus are substituted by one to ten chlorine atoms. There are 209 congeners, which are arranged according to current nomenclature from 1 to 209 by increasing number of chlorines. Although physical and chemical properties vary widely across the class, PCBs generally have low solubility in water, high lipophilicity, and low vapour pressure; they are chemically stable and generally persist in the environment and in the human body.

PCBs are not known to occur naturally and have been produced commercially by a limited number of companies since 1929. Production peaked between the 1950s and the 1970s, and was banned in most countries by the 1980s; however, manufacturing in the Democratic People's Republic of Korea continued at least until 2006.

Commercial PCB products were manufactured to yield a given degree of chlorination to fulfil technical requirements. Products sold under different trade names (e.g. Aroclor, Clophen, Kanechlor) may be of similar composition with regard to the chlorine content. However, individual congeners have generally not been quantified in these products. A subset of PCBs are referred to as "dioxin-like PCBs," and have been assigned toxicity equivalency factors (TEFs) relative to 2,3,7,8-tetrachlorodibenzo-*para*-dioxin (TCDD).

Laboratory analyses of PCBs have improved in selectivity and sensitivity through the development of advanced instrumentation and analytical strategies allowing the identification and quantification of individual congeners within commercial products. State-of-the-art analytical methods enable detection of PCBs in virtually all types of sample; however, comparability with older methods is limited. Dioxin-like PCBs often occur in lower concentrations than other PCBs and are analysed together with polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Apart from instrumental analysis, analyses based on biological response have been applied as screening tools.

Based on their physical and chemical properties, such as non-flammability, chemical stability, high boiling point, and high dielectric constant, products containing PCBs were widely used in several industrial, commercial, and military open and closed applications. The most important closed applications were as dielectric fluids in capacitors and transformers, and as hydraulic fluid and heat-transfer medium. Although these applications are considered as "closed," PCBs can still be released into the environment due to leakage. The most important open applications were as constituents of permanent elastic sealants, in polymers, and as flame-retardant coatings. To a lesser extent, PCBs were also used in inks, adhesives, dyes for carbonless duplicating paper, conveyor belts, and other rubber products, small ballasts for fluorescent lights, cutting

and lubricating oils, and metal coatings. In all open applications, PCBs can be released from the product into the environment via volatilization or erosion.

Once released into the environment, PCBs can be transported via environmental media and migratory species far from the site of production and use. PCBs are ubiquitous in the environment and are found in biota, air, soil, sediment, and water worldwide, including in polar regions and deep oceans. PCB concentrations vary by several orders of magnitude. Furthermore, congener patterns differ to varying degrees in air, water, sediments and soils as a consequence of transport, and transformation processes such as dechlorination. In the environment, PCBs volatilize easily, or are ingested by fish and other animals and transferred to the food chain, where their concentration may increase.

The general population is exposed primarily through ingestion of contaminated food. Food can become contaminated with PCBs by: (i) uptake from the environment by fish, birds, livestock; (ii) contamination of the foodstuffs through usual practice or industrial processing; and (iii) accidental contamination. In contrast to vegetables and crops, fatty foods typically contain high concentrations of PCBs. Most foodstuffs will have a shift in the congener profile in favour of less volatile, more highly chlorinated congeners.

Six congeners (PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, PCB-180) are found at high concentrations in the environment, food, and in human tissue. These congeners are often used to monitor exposure in epidemiological studies and are referred to as "indicator PCBs."

There have been two major episodes of human food contamination; both of which occurred in Asia; these episodes are commonly referred to as "Yusho" and "Yucheng." These populations were exposed through accidental contamination of cooking oil with either Kanechlor 400 or Kanechlor 500. Exposed people had blood PCB concentrations that were 100 to 1000 times higher than in the non-exposed population. Other accidental releases have occurred in the last few decades.

Indoor air can also contribute to human exposure to PCBs, owing to the use of PCBs in construction material. Exposure can occur in the workplace or at home; importantly, children may be exposed in schools and nurseries where PCB-containing materials have been used. Inhalation of PCBs results in a higher relative exposure to the more volatile, less chlorinated congeners.

Workers may be exposed during manufacturing, repair, use, and disposal of products or equipment containing PCBs. Earlier exposures to PCBs were higher and occurred during PCB manufacture, and filling of PCB-containing transformers and capacitors (up to 11 000 μg/m^3) and during the repair of transformers (up to 60 μg/m^3). More recent exposures may occur during abatement in construction (up to 120 μg/m^3), waste incineration, and recycling of electronic equipment and – to a lesser extent – working in PCB-contaminated buildings (up to 10 μg/m^3). It has been reported that workers in small-scale welding facilities in less developed countries may not use personal protective equipment when extracting PCB-contaminated coolant oil from discarded transformers, and are therefore likely to receive a considerable degree of exposure.

Historically, workers were exposed through inhalation and dermal contact, while occupational exposure to PCBs is nowadays primarily through dermal contact. In the past, workers were exposed during PCB manufacture and use to congener patterns that were similar to those of the products they handled, while today's workers are exposed to congener profiles that are different from those of the commercial mixtures. Occupational exposures to PCBs before the banning of PCB manufacture in the 1980s were much higher than those encountered today from

other sources. Since then, levels of occupational exposure to PCBs have been greatly reduced and now approach levels of environmental exposure.

5.2 Human carcinogenicity data

The association between exposure to PCBs and risk of cancer in humans has been evaluated in a large number of epidemiological studies in several occupational groups, in populations with elevated exposure to PCBs as a result of environmental incidents, and in the general population. Studies have been conducted in several countries, primarily in North America, Europe, and Asia, and have used cohort, nested case–control, and case–control designs.

The Working Group considered more than 70 separate studies with informative data regarding several cancer sites. The most important evidence regarding carcinogenicity came from studies of workers in industries where PCBs were used, and from population-based case–control studies. Occupational studies assessed exposure to PCB mixtures through job-exposure matrices and historical measurements, but most did not report data on non-occupational risk factors, which are important for some cancer sites. In case–control studies, analyses included adjustments for a larger range of risk factors and most used measurements of PCB concentrations (typically for specific congeners or groups of congeners) in blood or adipose tissue as indicators of exposure. The Working Group did not consider any exposure-assessment approach to be superior, each providing contrasting but useful information.

5.2.1 Malignant melanoma

Information on the association between risk of melanoma and exposure to PCBs was available primarily from cohort studies of capacitor- and transformer-manufacturing workers (four studies) and electric power and equipment workers (three studies) in North America and Europe. Excess risks of melanoma were reported in all studies except one. The only study reporting null results combined data from two plants in the USA: risk was significantly increased in the plant with predominantly white workers, but not in the second, where a large proportion of workers of African heritage were employed. Exposure–response relationships were evaluated in three studies and a statistically significant linear exposure–response trend was observed with a 20-year lag in the largest study, which included workers at five electric power companies.

Further evidence came from a high-quality case–control study of skin melanoma in Canada, which reported measurement of plasma concentrations of PCBs. This was the only case–control study in which the association between PCBs and melanoma was evaluated in the general population; the study used biological measurements of exposure and accounted for potential confounding factors. Trends were evaluated for dioxin-like PCBs, non-dioxin-like PCBs, and eight highly chlorinated individual congeners: all trends were positive and statistically significant. Additional support came from a multicentre European case–control study of uveal melanoma that assessed occupational exposure to oils containing PCBs and found positive associations.

The association between malignant melanoma and exposure to PCBs was consistently observed across studies of occupational exposure in different industries in several countries, in the general population, and with both cohort and case–control designs. These findings were unlikely to be a result of chance, since statistically significant associations were observed in large studies. Exposure–response relationships were also observed in several studies using different methodologies among exposed workers and in the general population. Confounding or other bias is unlikely to explain these results: there are few known risk factors for malignant melanoma other than sunlight, which was controlled for in

the case–control studies and in the only large study of occupational exposure that included outdoor workers for whom occupational exposure to sunlight could be significant. Exposure to sunlight is unlikely to confound associations in studies of indoor workers, since there is no reason to believe that exposure to sunlight during leisure time is associated with occupational exposure to PCBs.

5.2.2 Non-Hodgkin lymphoma

Data on the association of NHL and exposure to PCBs are available from studies of five independent occupational cohorts of capacitor manufacturing workers (three in the USA, one each in Italy and Sweden) and two cohorts of transformer manufacturing and repair workers (one in the USA, one in Canada). Four of these studies included specific assessments of the level of PCB exposure (three in the USA, one in Sweden). Statistically significant increases in mortality from NHL were observed in a cohort of capacitor manufacturing workers in Italy and among retired workers at a transformer manufacturing plant in the USA. Non-statistically significant increased risk of NHL was observed in the other capacitor and transformer manufacturing cohorts. However, a separate analysis of one of these latter cohorts by different investigators reported no excess of NHL. None of the four studies that assessed the level of PCB exposure found clear evidence of an exposure–response relationship The number of deaths from non-Hodgkin lymphoma was above that expected among men (deaths, $n = 4$) in a mortality follow-up study of a population in Taiwan, China, as a result of a mass poisoning episode with cooking oil contaminated with PCBs (Yucheng). However, no data on non-Hodgkin lymphoma were reported after a similar episode in Japan (Yusho), with a different exposure profile.

Nested case–control studies were conducted among subsamples of large population cohorts, and presented the advantage of having collected blood at recruitment, and having subsequently identified incident cases. Statistically significant trends in risk were associated with the sum of PCB congeners in three of the five studies considered; and were positive with specific congeners in several studies.

Four out of six good-quality case–control studies provided indications of a positive trend in risk of non-Hodgkin lymphoma with increasing plasma concentrations of the sum of PCBs. The results of a European case–control study of non-Hodgkin lymphoma were null overall, although heterogeneity was observed across the participating centres. A positive interaction was reported with markers of infection with Epstein-Barr virus (EBV), or with polymorphisms in genes encoding inflammatory cytokines, or an ancestral haplotype for human leukocyte antigen (HLA). Regarding non-Hodgkin lymphoma subtypes, follicular lymphoma, but not diffuse large B-cell lymphoma, was positively associated with exposure to PCBs in three studies.

In summary, the balance of evidence, taking into account study size and quality, suggested increased risk of non-Hodgkin lymphoma in relation to PCB exposure, and this is biologically plausible. However, since heterogeneous results were observed in high-quality studies, the Working Group could not exclude chance as a potential explanation for the associations observed. It is noteworthy that bias and confounding were excluded.

5.2.3 Cancer of the breast

Many studies investigated the risk of cancer of the breast in relation to exposure to PCBs, with the rationale that such an association is biologically plausible. The evidence that weighed most strongly in this evaluation came from 12 well designed and implemented case–control studies in the USA, Canada, and Japan that assessed risk in relation to concentrations of PCBs measured

in serum and/or adipose tissue. These studies each included between 175 and 750 cases of cancer of the breast, the controls being comparable women without cancer of the breast, and results were adjusted for relevant confounders. In one large study in the USA, no excess risk was seen, while in the other large study in the USA, increased risk of cancer of the breast in relation to PCBs was seen among African-American women, and among parous never-lactating white and African-American women combined. Of the 10 moderately sized studies, increased risks were seen in six studies in relation to PCBs, with some exposure–response relationships. In some of these studies, risk was also evaluated by subgroup, and increased risks were seen for women who were parous and had never lactated, for pre- and postmenopausal women, by various tumour characteristics, and by *CYP1A1* variants. Statistically significant increases in risk ranged from 1.1- to 4.3-fold. Three additional moderately sized studies from the USA reported no excess risk, while an inverse risk was seen in one study from Japan. Two additional case–control studies assessed PCBs through estimates of occupational or dietary exposure, and although the results suggested some increase in risk, these studies were not weighted strongly. In addition, most of the 10 smaller case–control studies reported some increased risks in relation to PCBs, although they were not weighted strongly in this evaluation due to the imprecise risk estimates.

While a few cohort studies of occupational exposure suggested an increased risk of cancer of the breast, PCB exposure was usually not assessed quantitatively in relation to risk, and important potential confounders were not taken into account. Within a case–control study nested among female capacitor workers, increased risk of cancer of the breast was seen for "non-white" (otherwise unspecified) women, taking into account non-occupational confounders. Other nested case–control studies (six from the USA, two from Denmark, and one from Norway) had a small or moderate number of cases and assessed PCBs in serum or adipose tissue with controls for confounders. The findings suggested some increased risks associated with some of the PCBs analysed, but the studies had limited power to assess associations.

On the balance of evidence, when taking into account study size, quality, and magnitude of risk, an increased risk of cancer of the breast was seen in relation to PCBs, with higher risks among some subgroups, and these associations are biologically plausible. Bias and confounding are unlikely to explain these results. However, as the results across high-quality studies were heterogeneous, the Working Group could not exclude chance as a possible explanation for positive associations.

5.2.4 Other cancer sites

Several other cancer sites were considered in one or more cohort or case–control studies. There were positive findings for cancer of the prostate and brain in several studies, but null findings in others. Other cancers with sporadic positive findings were those of the liver and biliary tract, extrahepatic biliary tract, lung and respiratory tract, thyroid, stomach, pancreas, colon and rectum, urothelial organs, uterus and ovary combined, as well as childhood acute lymphatic leukaemia, and multiple myeloma.

5.3 Animal carcinogenicity data

PCBs (individual congeners, binary mixtures, and commercial mixtures) were evaluated in rats and mice in studies of various design, and ranging in duration from several months up to 2 years. These included 2-year studies of carcinogenicity, studies involving transplacental/perinatal and postnatal exposure, initiation–promotion studies examining the promoting activity, and other co-carcinogenicity studies, using tumours as an end-point.

For the 2-year bioassays, the route of administration was oral, by gavage or feeding. In studies of initiation–promotion, co-carcinogenicity, and transplacental/perinatal exposure, PCBs were also administered intraperitoneally, subcutaneously, or by skin application. There were no studies of exposure by inhalation.

5.3.1 PCB congeners

PCB-126 was tested for carcinogenicity in one study in female rats treated by gavage. PCB-126 caused significant increases in the incidences of benign and malignant tumours of the liver (hepatocellular adenoma, hepatocholangioma, and cholangiocarcinoma), lung (cystic keratinizing epithelioma), and oral mucosa (gingival squamous cell carcinoma). In two studies of transplacental/perinatal exposure in female rats treated by gavage, PCB-126 had an inhibitory effect on the development of tumours of the mammary gland induced by 7,12-dimethylbenz[*a*]anthracene (DMBA) in the offspring.

PCB-153 was tested for carcinogenicity in one study in female rats treated by gavage, one 4-month study of perinatal exposure in mice (including an initiation–promotion experiment), and one initiation–promotion study in mice. In the study of carcinogenicity, PCB-153 did not cause significant increases in the incidence of tumours in rats, but two rare cholangiomas were observed. PCB-153 promoted hepatocellular carcinomas induced by *N*-nitrosodiethylamine (NDEA) in mice. PCB-153 did not induce or promote bronchioloalveolar tumours in mice. PCB-153 was also evaluated as part of a binary mixture in a study examining the effect of increasing the dose of PCB-153 on the carcinogenicity of PCB-126 (see below); increasing the dose of PCB-153 increased the incidences of hepatocellular adenoma and cholangiocarcinoma when coadministered with PCB-126.

PCB-118 was tested for carcinogenicity in one study in female rats treated by gavage. PCB-118 caused significant increases in the incidences of benign and malignant tumours of the liver (hepatocellular adenoma, hepatocholangioma, and cholangiocarcinoma), benign tumours of the lung (cystic keratinizing epithelioma), and carcinoma of the uterus.

A binary mixture of PCB-126 and PCB-153 was tested for carcinogenicity in one study in female rats treated by gavage. The mixture of PCB-126 and PCB-153 caused significant increases in the incidences of hepatocellular adenoma, hepatocholangioma and cholangiocarcinoma, cystic keratinizing epithelioma of the lung, and squamous cell carcinoma of the oral mucosa. As stated above, increasing the proportion of PCB-153 to PCB-126 caused significant increases in the incidences of hepatocellular adenoma and cholangiocarcinoma in one study.

A binary mixture of PCB-118 and PCB-126 was tested for carcinogenicity in one study in female rats treated by gavage. The mixture caused significant increases in the incidences of hepatocellular adenoma, cholangiocarcinoma, and cystic keratinizing epithelioma of the lung.

When given to mice for 4 months, from the perinatal period to adulthood, PCB-138 was not carcinogenic, but did show evidence of a promoting effect based on a significant increase in the multiplicity of bronchioloalveolar adenomas induced by *N*-nitrosodimethylamine (NDMA).

A mixture of PCB-138 and PCB-153 was administered to mice for 4 months, from the perinatal period to adulthood. The mixture was not carcinogenic, and did not promote bronchioloalveolar tumours.

A mixture of non-*ortho*, mono-*ortho*, and di-*ortho* substituted PCB congeners, *p,p′*-dichlorodiphenyltrichloroethane (DDT) and *p,p′*-dichlorodiphenyldichloroethene (DDE) was tested for carcinogenicity in one study of perinatal exposure in rats treated by gavage. The mixture was not carcinogenic.

The hydroxylated mono-*ortho*-PCBs 2′,4′,6′-trichloro-4-biphenylol (4′-OH-PCB-30) and 2′,3′,4′,5′-tetrachloro-4-biphenylol

(OH-PCB-61), alone or as a binary mixture, were tested for carcinogenicity in one study of perinatal exposure in female mice treated by subcutaneous injection. Both the individual congeners and the binary mixture caused a significant increase in the total incidence of malignant tumours of the cervicovaginal tract (squamous cell carcinomas and adenosquamous carcinomas).

A mixture of the three non-*ortho* congeners PCB-77, PCB-126, and PCB-169, six polychlorinated dibenzodioxins, and seven polychlorinated dibenzofurans was tested for carcinogenicity in one study of perinatal exposure in female rats treated by gavage. The mixture caused a significant increase in the incidence of benign lesions of the mammary gland (hyperplasia, adenoma, and fibroadenoma).

A mixture of PCB-126, TCDD, and 2,3,4,7,8-pentachlorodibenzofuran was tested for carcinogenicity in one long-term study in female rats treated by gavage. The mixture caused a significant increase in the incidence of benign and malignant tumours of the liver (hepatocellular adenoma and cholangiocarcinoma) and benign tumours of the lung (cystic keratinizing epithelioma).

5.3.2 Aroclor

In a feeding study of carcinogenicity in male and female rats, Aroclor 1016 caused significant increases in the incidence of hepatocellular adenoma, and of hepatocellular adenoma or carcinoma (combined) in female rats.

In a feeding study of carcinogenicity in male and female rats, Aroclor 1242 caused significant increases in the incidence of hepatocellular adenoma in female rats, and of thyroid follicular cell adenoma, and thyroid follicular cell adenoma or carcinoma (combined) in males.

Aroclor 1254 was tested for carcinogenicity in two feeding studies in male and female rats, one feeding study in male mice, three studies of transplacental/perinatal exposure in mice, two studies examining promoting activity in male rats, five studies examining promoting activity in mice, and three co-carcinogenesis studies in mice. In rats, oral administration of Aroclor 1254 caused significant increases in the incidence of hepatocellular adenoma or carcinoma (combined) in males in the first study, and of hepatocellular adenoma and hepatocellular carcinoma in females, and of thyroid follicular cell adenoma, and follicular cell adenoma or carcinoma (combined) in males in the second study. In mice, oral administration of Aroclor 1254 caused significant increases in the incidence of "hepatomas" of the liver. In the studies of transplacental/perinatal exposure, Aroclor 1254 was not carcinogenic in mice, but promoted NDMA-induced bronchioloalveolar adenomas in two studies, and coalescing liver tumours in one study. In rats, Aroclor 1254 promoted NDEA-induced hepatocellular carcinomas in one study. In mice, Aroclor 1254 promoted NDEA-induced hepatocellular adenomas in one study, and NDEA-induced hepatocellular carcinomas, hepatoblastomas, and cholangiocellular tumours in another study. In a third study, Aroclor 1254 promoted lung tumours induced by NDMA and by 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK).

Aroclor 1260 was tested for carcinogenicity in one feeding study in male rats, one feeding study in female rats, and two feeding studies in male and female rats. Aroclor 1260 caused significant increases in the incidences of "liver tumours" in males in one study, and of hepatocellular adenoma and carcinoma in females in a second study. In a third study, Aroclor 1260 increased the incidence of hepatocellular carcinoma in females, and of cholangioma in males and females. In a fourth study, Aroclor 1260 increased the incidence of hepatocellular adenoma in males, of hepatocellular adenoma, hepatocellular carcinoma, and cholangioma in

females, and of thyroid follicular cell adenoma in males.

5.3.3 Clophen

In one feeding study of carcinogenicity in male rats, Clophen A 30 caused a significant increase in the incidence of benign hepatocellular tumours.

In one feeding study of carcinogenicity in male rats, Clophen A 60 caused significant increases in the incidence of benign hepatocellular tumours and hepatocellular carcinoma.

5.3.4 Kanechlor

Kanechlor 300 gave negative results when tested for carcinogenicity in one feeding study in male and female mice, and one feeding study in male mice.

Kanechlor 400 was tested for carcinogenicity in one feeding study in male and female mice, one feeding study in male mice, and one feeding study in male and female rats. Kanechlor 400 was also tested in three initiation–promotion studies examining promoting activity, one in rats and two in mice. Both studies of carcinogenicity in mice gave negative results. The results of the study of carcinogenicity in rats were inconclusive. Kanechlor 400 promoted hepatocellular tumours in one initiation–promotion study in rats, and in one initiation–promotion study in mice.

Kanechlor 500 was tested for carcinogenicity in one feeding study in male mice, one feeding study in male and female mice, and one initiation–promotion study of transplacental/perinatal exposure in male and female rats. It was also tested in three initiation–promotion studies, one in rats and two in mice, examining promoting activity. Kanechlor 500 caused significant increases in the incidence of hepatocellular carcinoma in both studies of carcinogenicity in male and female mice. Transplacental/perinatal administration of Kanechlor 500 decreased the incidence of NDEA-initiated tumours of the liver in rats. Kanechlor 500 promoted hepatocellular tumours in the three initiation–promotion studies.

5.4 Mechanistic and other relevant data

5.4.1 Absorption, distribution, metabolism, and elimination

(a) Absorption

In humans, gastrointestinal absorption of PCBs was estimated to vary from 50% of the ingested amount to close to 100%, the absorption decreasing as the number of chlorine atoms of the congener increased. A similar situation was observed in experimental animals. Although no quantitative data were available regarding absorption of PCBs in humans exposed by inhalation, the levels of residues detected in individuals exposed to high concentrations of PCBs in air suggested that inhaled PCBs are absorbed to a substantial extent. Data from experimental animals indicated that inhalation of PCBs gives a higher uptake of PCBs than ingestion. Studies assessing dermal exposure to commercial PCB mixtures in humans and animals showed that this route of exposure generally results in absorption levels of between 20% and 40%, with dermal penetration varying inversely with the degree of chlorination of the mixture administered. First-pass metabolism at the site of dermal exposure appears to be responsible for differences in metabolism and disposition between routes of administration. The rate of absorption and the disposition of PCBs after dermal administration may be mediated by transdermal metabolism.

(b) Distribution

PCBs are lipophilic compounds that are preferentially retained and may accumulate in adipose tissue and lipid-rich tissues. A few studies mentioned substantial retention of certain congeners in the lung and spleen in mice and rats, respectively. The pattern of congeners observed in tissues of humans or experimental animals does not correspond to the congener profiles of PCB formulations. The major PCB components in the plasma and adipose tissue of occupationally exposed individuals are the hexa- and heptachlorobiphenyls. PCB congeners with chlorine atoms in the *para* positions are generally found at relatively high concentrations, while PCBs with unsubstituted *meta*,*para* positions on at least one ring are present at lower concentrations. The most abundant congeners found in adipose tissue, plasma, and liver are 2,2′,3,4,4′,5′-hexachlorobiphenyl (PCB-138), 2,2′,4,4′,5,5′-hexachlorobiphenyl (PCB-153) and 2,2′,3,4,4′,5,5′-heptachlorobiphenyl (PCB-180). PCBs have been found to cross the blood–brain barrier, and data from humans and experimental animals provided clear evidence for the transplacental passage of these chemicals. Metabolites of PCBs, including hydroxylated PCBs and methylsulfone PCBs, are also known to distribute to various tissues.

(c) Metabolism

Individual PCB congeners differ greatly in the ease with which they are metabolized in humans and animals. Congeners with four or fewer chlorines and those with adjacent unsubstituted *meta*,*para* positions are metabolized more readily than those with more than four chlorines and with substituents at *meta*,*para* ring positions. The initial step in the biotransformation of all PCB congeners is cytochrome P450 (CYP)-dependent mono-oxygenation. Readily metabolized congeners can be converted to potentially electrophilic and genotoxic metabolites of PCBs, arene oxides, and quinones. Quinones arise from dihydroxylated PCB metabolites through the action of peroxidases or prostaglandin endoperoxide synthase. The other major pathway of metabolism of PCBs is conversion of an arene oxide metabolite to a glutathione conjugate. The glutathione conjugate is then converted either to the excreted non-toxic mercapturic acid, or to the generally poorly excreted methyl sulfone metabolite.

(d) Elimination

Highly chlorinated congeners persist in the body, with half-lives averaging about 8–15 years; the half-lives of less chlorinated PCBs are distinctly shorter. In addition, PCB half-lives vary according to species, being longer in humans than in experimental animals, including monkeys. PCBs are mainly excreted via the faeces, while urine usually represents a minor route of excretion. Faecal excretion concerns not only unabsorbed PCBs, but also the excretion of biliary metabolites in the intestine. The proportion as well as the rate of elimination in the excreta depends on the type of mixture or congener and the route of exposure. Excretion profiles, and metabolite profiles in excreta, were different after administration of a dermal dose of PCBs when compared with an equivalent intravenous dose.

In addition to hydroxylated and dihydroxylated PCBs, the corresponding glucuronide and sulfate conjugates, as well as mercapturic acids, have also been characterized in the urine. Lactation is also a major route of excretion of PCBs in animals and humans. Minor routes of excretion such as elimination through the intestinal wall in the gastrointestinal tract or via the skin may also occur.

5.4.2 Genetic and related effects

A very limited number of studies in humans was available on cytogenetic effects in peripheral lymphocytes (chromosomal aberration, sister-chromatid exchange, micronucleus formation) and urinary concentrations of 8-hydroxy-2′-deoxyguanosine (8-OHdG) in populations with possible exposure to PCBs. Although all these studies provided valuable information on genetic and related effects in humans exposed occupationally and environmentally to PCBs, the interpretation and generalization of the results was hindered by lack of information about PCB exposure, analysis, and levels, the lack of a real unexposed control population, the small number of individuals examined, confounding exposure to other chemicals, and lifestyle factors.

Several reports of sperm DNA damage and chromosome aneuploidy indicated that the testis may be a target organ for toxicity associated with PCBs.

Some very recent studies indicated that PCBs affect DNA methylation patterns in exposed humans, with long-term consequences for gene expression and chromosome stability. Since genes encoding for steroid hormone-synthesizing enzymes and oncogenes have been shown to be targeted, this may have significant implications for a possible mode of action of carcinogenesis by PCBs.

There was a lack of data about levels or even occurrence of individual PCB congeners in publications on the genotoxic effects of PCBs in humans. Only a few recent studies had analysed a very small number of congeners and calculated correlations with biological effects. Statistically positive correlations were found between serum concentration of PCB-118 and formation of micronuclei and DNA strand breaks (comet assay) in peripheral lymphocytes, serum concentrations of PCB-153 and DNA fragmentation in sperm, serum concentrations of PCB-138 and PCB-153 and *KRAS* mutation in tumours of the pancreas and brain, and PCB-95 concentrations and autism with a genetic basis (maternal dup15q11–q13 and Prader-Willi syndrome). These were interesting observations, but not sufficient to allow a structure–activity correlation.

Of all the commercial PCB mixtures, Aroclor 1254 has been by far the most extensively investigated for genetic effects in vitro and in vivo. Although numerous studies in vitro and in vivo with a negative outcome have been reported, almost none are suitable for hazard assessment, primarily due to the low doses tested and, in case of studies in vitro, the lack of an exogenous metabolic system. Thus the Working Group concluded, on the basis of a positive test for cell transformation and a weakly positive study of mutagenicity in transgenic mice in vivo, that mutagenicity associated with long-term exposure to Aroclor 1254 cannot be excluded with certainty.

Studies of mutagenicity with individual PCBs were available for 13 congeners. The most frequently investigated congener was monochlorinated PCB-3 and its metabolites, and studies in vitro and in vivo provided clear evidence that PCB-3 causes mutation in vitro and in vivo. However, metabolic activation to electrophilic species, i.e. quinones, is required, as shown by direct testing of PCB-3 metabolites for gene mutagenicity in vitro. The experimental evidence overall suggested that both DNA-adduct formation and generation of reactive oxygen species must be considered equally plausible modes of action.

Since both in-vitro and in-vivo studies provided evidence that PCB congeners with up to four chlorines are metabolically activated to electrophilic species that cause an increase in DNA-adduct levels, it seems likely that PCBs with one to four chlorines have the same mode of action as PCB-3. In contrast, strong evidence suggested that decachlorinated PCB-209 is very unlikely to cause mutations.

For dioxin-like PCB-126, a dose-dependent increase in DNA-adduct formation – resulting from lipid peroxidation or oxidative damage of the DNA backbone – has been reported in rats exposed to PCB-126 in the long-term. Thus, a genotoxic mechanism, probably via generation of reactive oxygen species, seems to contribute to the mode of action of PCB-126.

For non-dioxin-like PCB-153, a complete lack of genotoxic activity cannot be established with certainty since three in-vitro studies gave positive results. However, mechanistic follow-up studies in vitro and/or in vivo were not available to the Working Group. Thus, the relevance of this finding remains elusive.

For all other nine PCB congeners tested, i.e. PCB-15, PCB-47, PCB-52, PCB-77, PCB-101, PCB-118, PCB-138, PCB-155, and PCB-180, the Working Group considered that the results did not allow a clear conclusion to be drawn.

5.4.3 Cellular and biochemical effects

PCB congeners can be categorized according to their degree of chlorination, substitution pattern, and binding affinity to receptors. Individual PCB congeners activate receptors, including the aryl hydrocarbon, constitutive androstane, and pregnane xenobiotic receptors, and modulate gene expression controlled by these receptors/transcription factors.

(a) Cell death and proliferation

Twelve PCB congeners that have a strong affinity for the aryl hydrocarbon receptor are referred to as "dioxin-like PCBs." Activation of the aryl hydrocarbon receptor is one of the key events linked to carcinogenesis mediated by dioxin-like compounds. Besides its role in induction of CYP1 enzymes (linked to toxicity and cancer initiation), sustained activation leads to deregulation of cell-cycle control and cell proliferation, inhibition of apoptosis, suppression of cell–cell communication and adhesion, and increased cell plasticity and invasiveness. In accordance with the concept of toxic equivalency, PCB-126 is the most potent aryl-hydrocarbon receptor agonist of the PCBs, followed by PCB-169; mono-*ortho* chlorinated PCBs (e.g. PCB-118, PCB-156), and PCB-77 also activate the aryl hydrocarbon receptor, although to a lesser extent.

On the other hand, non-dioxin-like PCBs induce many of their effects via multiple aryl hydrocarbon receptor-independent mechanisms, including activation of the constitutive androstane or pregnane X receptors, and perturbations in cell–cell communication and cell adhesion. Non-dioxin-like PCBs induce production of reactive oxygen species, activation of NF-κB transcription factors, and suppression of plasma membrane proteins, constituents of gap, adherens, and tight junctions, all of which may play a significant role in tumour promotion and progression. A series of non-dioxin-like PCBs, including less chlorinated congeners (e.g. PCB-18, PCB-47, PCB-52, and PCB-74), environmentally abundant congeners (e.g. PCB-138 and PCB-153), and hydroxylated metabolites, such as 3′,4′-di(OH)PCB-5, 4-OH-PCB-109 (4-OH-2,3,3′,4′,5-pentaCB), and 4-OH-PCB-187, inhibited gap junction intercellular communication in rat liver epithelial cells. A mixture of seven non-dioxin-like PCBs (PCB-28, PCB-52, PCB-101, PCB-138, PCB-153, PCB-180, and PCB-209) induced production of reactive oxygen species and cell motility in human breast cancer cells. Both the dioxin-like congener PCB-126, and the non-dioxin-like congeners PCB-118 and PCB-153 disrupted the expression of cytosolic scaffold proteins of tight junctions in brain endothelial cells in mice. Expression of anti-apoptotic *Bcl2* gene in a short-term study in female rat liver, to decrease apoptotic index and to suppress the levels of gap junction and adherens junction proteins (connexin 43, β-catenin, E-cadherin) in rat liver epithelial cells. PCB-28, PCB-101, PCB-153, and also PCB-187 (to a lesser

extent) suppressed apoptosis in rat hepatocytes and human hepatoma HepG2 cells.

(b) Endocrine disruption

Population-based studies in men and women have shown an inverse correlation between serum concentrations of PCBs and circulating testosterone, including testosterone bound to sex-hormone-binding globulin. Studies on mother–infant pairs showed an inverse relationship between indicator PCBs and testosterone in female infants, which was statistically significant with the mono-*ortho* congeners PCB-105 and PCB-118, while male infants showed a stronger reduction in estradiol with higher serum concentrations of PCBs.

In studies on extracts of PCBs from human serum, higher serum PCB concentrations correlated with lower activities of the estrogen, androgen, and aryl hydrocarbon receptors.

The observed inverse trend between dioxin-like PCBs and activities of the aryl hydrocarbon and estrogen receptors suggests that these compounds have anti-estrogenic activity. In cultured cells, highly chlorinated congeners generally act as anti-estrogens and their hydroxylated metabolites are more active than the parent compound. In contrast, less chlorinated PCBs and their hydroxylated metabolites are generally estrogenic, and their potency is dependent upon *ortho* chlorination and *para* hydroxylation; estrogenic activities of the hydroxylated metabolites of less chlorinated PCBs were reported to be additive.

Studies with cultured cells demonstrated that some PCBs are androgen-receptor antagonists, the anti-androgenic effects of dioxin-like PCBs being more pronounced than those of *ortho*-substituted PCBs. This antagonism has been associated in humans with several factors related to an increased risk of cancer of the testis.

In population-based studies, an inverse correlation was also reported between total serum PCBs and triiodothyronine, thyroxine, and thyroid-stimulating hormone. For hydroxylated PCBs, a positive correlation was found with free thyroxin in umbilical cord tissue of fetuses after in-utero exposure.

Studies in rats demonstrated that hydroxylated PCBs that bind to the thyroid receptor act as agonists to the thyroid hormone; one metabolite even displayed a higher binding affinity than does thyroxine, the natural ligand. PCBs with chlorines in the *ortho* position only have significant binding affinity for the transport protein transthyretin.

Hydroxylated PCBs may cross the placental barrier, probably through binding to transthyretin, thus causing a reduction of total and free thyroxine concentrations in fetal plasma and brain. Moreover, pre- and postnatal exposure to PCBs and their hydroxylated metabolites can interfere with the thyroid-hormone system, which may lead to a decrease in levels of thyroid hormone.

Disturbance of thyroxine-binding to transthyretin by PCB metabolites and increased glucuronidation causes a reduction in serum thyroxine concentrations in Aroclor 1254-exposed rats. The interference of PCBs with the thyroid system in vitro as well as in animals corroborates the effects observed in human population studies. The effects of PCBs on thyroid-hormone function, metabolism and transport may increase the risk for toxicity and pre-cancerous processes.

In a study that considered 10 different mechanisms to establish in-vitro toxicity profiles for 24 PCB congeners, hierarchical cluster analysis showed that 7 indicator PCBs contributed most to the anti-androgenic, (anti)estrogenic, and anti-thyroidal effects of PCBs reported to be present in human samples.

(c) Effects on the immune system

The limited data available for human exposure suggested that PCBs may cause immunosuppression. PCBs can affect an impressive number of immune parameters that include

changes in bone-marrow cellularity; shifts in T-lymphocyte subsets and function; thymus and spleen atrophy, which correlate strongly with humoral and cell-mediated immunosuppression; reduced resistance to microbial infection; and a compromised immune-surveillance mechanism. Alterations in the immune system and immunotoxicity were also reported after PCB exposure during prenatal or early life.

An estimation of the degree of immunotoxicity induced by various PCB congeners and mixtures is hindered by the fact that several species with significant differences in sensitivity were used across the studies, with different routes of exposure and levels of treatment. In general, doses of > 1 mg/kg bw per day of the highly chlorinated commercial PCB mixtures (Aroclors 1248, 1254, 1262, and 1260) were more immunotoxic than the less chlorinated PCB mixtures. The few individual congeners tested in rats caused only minor changes in the thymus without affecting other parameters of the immune system.

Non-human primates are more sensitive to PCB-induced immunotoxicity. In long-term studies in rhesus monkeys exposed at levels similar to those in humans, a consistent finding was the significantly suppressed response to challenge with sheep red blood cell antigen in adult and infant monkeys. Similar results were observed in many other experimental animals at higher concentrations of PCBs.

The humoral immune response to sheep red blood cell antigen is the most predictive of the tests currently used in immunotoxicology, and has been used in the calculation of TEFs. The TEF calculation is based on the assumptions that the effects of PCBs on the immune system are mediated through the aryl hydrocarbon receptor, and that PCBs in mixtures may have an additive effect. Nonetheless, certain PCBs exert their immunotoxic effects by mechanisms that are not mediated through the aryl hydrocarbon receptor; such effects are thought to be mediated via metabolism to arene-oxide intermediates capable of alkylating critical cellular macromolecules. Additionally, certain non-dioxin-like PCBs may antagonize the immunotoxic effects of other chemicals, including those of dioxin.

The effects on the immune system were shown to persist in children at a later age. The severity of effects correlated with PCB concentrations in the children's blood, or with those in maternal blood during pregnancy and lactation. Similar results were obtained in experimental animals.

(d) Effects on the inflammatory response

Exposure to PCBs has been associated with the development of inflammation in several studies in experimental animals in vivo; chronic active inflammation can be detected specifically in tissues that are affected by PCB exposure.

In in-vivo studies in mice, it has been reported that PCB-77, PCB-104, and PCB-153 are associated with inflammation in target organs since they induced the production of specific inflammatory mediators, including intercellular adhesion molecules (e.g. ICAM, VCAM-1, MCP-1) in the liver, lungs, and brain. The tissue distribution of these inflammatory mediators varied according to the congener administered, probably due to differences in congener accumulation in the various organs.

PCBs have also been shown to cause vascular inflammation in vivo.

In vitro, PCB-153 may induce expression of several pro-inflammatory cytokines through NF-κB pathway inhibitor.

Several PCB congeners and mixtures, including Aroclor 1242 and PCB-47, interfere with O_2^- elimination by suppressing the activity of superoxide dismutase which converts O_2^- to H_2O_2. Non-dioxin-like PCBs are capable of stimulating neutrophil O_2^- production, while dioxin-like congeners with a high affinity for the aryl hydrocarbon receptor do not activate neutrophils to produce O_2^- and may inhibit this response.

Certain congeners (PCB-77, PCB-114, PCB-126, and PCB-169) disrupted the normal functions of the vascular endothelium, thus allowing increased transfer of albumin across endothelial monolayers. The same congeners enhanced oxidative stress, increased production of interleukin-6 by endothelial cells, increased the levels of intracellular calcium, increased the activity of cytochrome P450 1A, enhanced expression of the adhesion molecule VCAM-1, and decreased levels of vitamin E in the culture medium. In contrast, PCB-153 did not have an effect on cellular oxidation or on endothelial barrier function.

5.4.4 Classification of congeners and quantitative structure–activity relationships

Different key structural determinants of the toxicity of individual PCB congeners were identified in various in-vitro assays for specific effects of tumour promotion, endocrine disruption, and neurotoxicity. Multivariate toxicity profiling of a series of PCB congeners indicated that many of the responses are due to different structure–activity relationships and cannot be integrated. The use of quantitative structure–activity relationships is also hampered at present by the lack of data on specific cancer-related modes of action for larger sets of congeners.

5.4.5 Hepatic preneoplastic lesions

Numerous studies have used preneoplastic lesions as end-points to study the effects of PCBs on two-stage hepatocarcinogenesis. PCBs have promoting activity, especially congeners and mixtures that activate the aryl hydrocarbon and/or constitutive androstane receptors. When non-*ortho* and di-*ortho* PCBs are coadministered, less than additive effects are observed in most studies, while administration of two non-*ortho* PCBs is additive. Several less chlorinated PCBs have initiating activity.

5.4.6 Organ toxicity

Organ toxicity relevant to the carcinogenicity of long-term exposure to PCB congeners and commercial mixtures of PCBs in experimental systems is observed in the liver and also in other organs, notably the lung and thyroid.

5.4.7 Effects on skin

Chloracne and other dermal alterations are well-known effects that have been reported in workers exposed occupationally to PCBs, and in individuals exposed by accidental ingestion of rice oil contaminated with high concentrations of PCBs (Yusho and Yucheng victims). Chloracne generally appears in individuals with serum PCB concentrations that are 10–20 times higher than those of the general population, but there is large variability between individuals. At birth, children exposed in utero during food poisoning incidents had increased rates of hyperpigmentation, eyelid swelling and discharge, deformed nails, and acne, compared with controls.

Long-term oral administration of relatively low doses of PCBs to rhesus monkeys resulted in dermal alterations similar to those observed in humans exposed at high concentrations. Offspring from monkeys exposed during gestation and nursed by exposed mothers also developed dermal alterations after a few weeks of suckling. Rodents also develop skin alterations, but only after high exposures to PCBs.

Exposure of normal human melanocytes to TCDD resulted in activation of the aryl hydrocarbon receptor signalling pathway, an aryl hydrocarbon receptor-dependent induction of tyrosinase and – as a consequence – an elevated total melanin content. These effects were due to the induction of expression of tyrosinase and tyrosinase-related protein 2 genes. Thus, the

aryl hydrocarbon receptor is able to modulate melanogenesis by controlling the expression of melanogenic genes. This lends biological plausibility to the epidemiological findings of increased risks of melanoma of the skin after exposure to PCBs.

5.4.8 Susceptible populations

(a) Genetic polymorphisms

Differences in response to individual congeners may arise from polymorphisms in the genes for CYP, the aryl hydrocarbon receptor and repressor, and other enzymes and receptors that interact with endogenous molecules such as steroid hormone receptors. Studies in the most highly exposed populations reported a higher incidence of cancer of the breast in women with the *CYP1A1*2C* genotype; of non-Hodgkin lymphoma and a polymorphism in the gene encoding the aryl hydrocarbon receptor; and of skin lesions in Yucheng victims who had the *CYP1A1*2C* polymorphism and were null for *GSTM1*.

(b) In-utero and postnatal exposure

PCBs can pass through the placenta during embryonic development and is excreted in breast milk. In addition, compared with adults, children have a lower barrier to absorption through the skin, gastrointestinal tract, and lungs, and lower levels of detoxifying enzymes. A combination of all these factors leads to a higher accumulation of PCBs in children. The determination of PCB concentrations in cord blood, breast milk, and in tissues of mother/infant have contributed significantly to the understanding of the movement of these compounds from mother to infant and their distribution patterns throughout the body.

A significant dose-dependent relationship exists between the duration of breastfeeding and the concentration of the sum of congeners PCB-101, PCB-118, PCB-138, PCB-153, PCB-170, PCB-180, PCB-183, and PCB-187. Exclusive breastfeeding beyond 12 weeks was associated with a doubling in the whole blood concentration of PCBs compared with bottle-fed children.

Elimination kinetic studies in children with elevated PCB concentrations as a result of breastfeeding revealed differences in congener half-lives. The longest half-lives corresponded to elimination of the parent PCB only, with a daily fat excretion rate of 1–2 g, while shorter half-lives were attributable to metabolic breakdown.

Long-term studies in non-human primates receiving Aroclor 1254 have shown that in tissues of mother/infants with higher concentrations of PCBs, a dramatic shift from tetra- and hexachlorobiphenyls to penta-and heptachlorobiphenyls was observed. The PCB distribution pattern in tissues from a dosed mother/infant pair differed between mother and infant, with a larger percentage of heptachlorobiphenyls in the infant than in its dam. PCB concentrations in the infant's blood declined rapidly and approached maternal levels within 40–50 weeks; at 100 weeks after weaning, PCB concentrations in the adipose tissue of exposed infants were similar to background levels found in the control group.

Tissue retention/accumulation of PCBs in postnatal and prepubertal studies in mice showed results consistent with the well known effect of chlorine-substitution pattern on the rate of metabolism. In the lung, all congeners except PCB-153 were retained and decreased in amount only as a function of dilution due to growth. The selective retention of congeners with high affinity for the aryl hydrogen receptor is of interest since it is a property that correlates with toxicity and tumour promotion. In the liver, retention of all congeners was observed during the prepubertal growth phase, with specific enrichment of PCB-105, followed subsequently by more rapid depletion of certain congeners.

Prenatal/postnatal (through breastfeeding) exposure to PCBs can affect the dynamics of cell-surface receptor expression on lymphoid cells. These effects result in dysfunctional

immune responses, which may have adverse immune-system related consequences on the health of infants and toddlers. Furthermore, PCB-induced effects on the thymus and natural killer cells have been reported in children, and these effects may play a role in the development of leukaemia in these children.

5.4.9 Mechanistic considerations

PCBs and their metabolites have multiple modes of action. Less chlorinated congeners involved in oxidative metabolism may produce oxidative stress and genotoxicity; highly chlorinated congeners are very persistent and interact with various receptors including the aryl hydrocarbon, constitutive androstane, pregnane-X (controlling xenobiotic and steroid hormone metabolism and other processes), and steroid nuclear receptors such as the androgen and estrogen receptors. Additionally, PCBs modulate plasma membrane-associated proteins affecting cell communication, adhesion and migration, and also act as tumour promoters. Overall, PCBs occur and act in complex mixtures eliciting both genotoxic and nongenotoxic effects associated with carcinogenesis, tumour promotion, and progression.

6. EVALUATION AND RATIONALE

6.1 Cancer in humans

There is *sufficient evidence* in humans for the carcinogenicity of polychlorinated biphenyls (PCBs). PCBs cause malignant melanoma. Positive associations have been observed for non-Hodgkin lymphoma and cancer of the breast.

6.2 Cancer in experimental animals

There is *sufficient evidence* in experimental animals for the carcinogenicity of PCBs.

There is *sufficient evidence* in experimental animals for the carcinogenicity of PCB-126, PCB-118, Aroclor 1260, Aroclor 1254, and Kanechlor 500.

There is *limited evidence* in experimental animals for the carcinogenicity of PCB-153, 4′-OH-PCB-30, 4′OH-PCB-61, Aroclor 1242, Aroclor 1016, Clophen A30, and Clophen A60.

There is *inadequate evidence* in experimental animals for the carcinogenicity of PCB-138, Kanechlor 300, and Kanechlor 400.

Congeners for which there is *sufficient evidence* in experimental animals for carcinogenicity (PCB-126 and PCB-118) are agonists of the aryl hydrocarbon receptor and exhibit dioxin-like properties. Commercial mixtures for which there is *sufficient evidence* in experimental animals for carcinogenicity are highly chlorinated and are known to include aryl-hydrocarbon receptor agonists that exhibit dioxin-like properties, as well as agonists of the constitutive androstane receptor.

The commercial mixtures for which there is *limited evidence* in experimental animals generally have a low degree of chlorination, but are also known to contain congeners that are agonists of the aryl hydrocarbon and/or constitutive androstane receptors. The relative contributions of the different congeners (dioxin-like and non-dioxin-like) to the carcinogenicity of the commercial mixtures is not known.

6.3 Overall evaluation

PCBs are *carcinogenic to humans (Group 1).*

"Dioxin-like" PCBs, with a toxicity equivalency factor (TEF) according to WHO (PCB-77, PCB-81, PCB-105, PCB-114, PCB-118, PCB-123, PCB-126, PCB-169, PCB-156, PCB-157, PCB-167, PCB-189), are *carcinogenic to humans (Group 1).*

6.4 Rationale

In making this overall evaluation, the Working Group considered that:

- There is strong evidence to support a receptor-mediated mechanism for carcinogenesis associated with dioxin-like PCBs in humans, based upon demonstration of carcinogenicity in experimental animals and upon extensive proof of activity identical to 2,3,7,8-tetrachlorodibenzo-*para*-dioxin (TCDD) for every step of the mechanism described for

TCDD-associated carcinogenesis in humans, including receptor binding, gene expression, protein-activity changes, cellular replication, oxidative stress, promotion in initiation–promotion studies and complete carcinogenesis in experimental animals.

- However, the carcinogenicity of PCBs cannot be attributed solely to the carcinogenicity of the dioxin-like PCBs.

POLYBROMINATED BIPHENYLS

POLYBROMINATED BIPHENYLS

1. Exposure Data

1.1 Identification of the agents

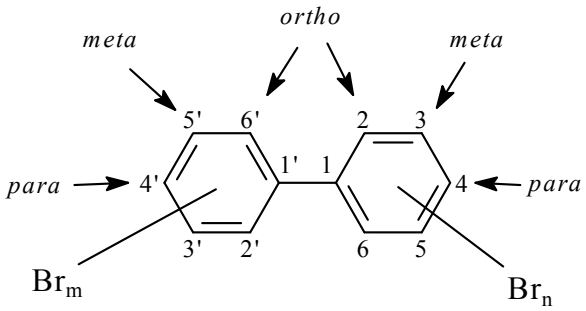
The terms "polybrominated biphenyls" or "polybromobiphenyls" (PBBs) refer to a group of halogenated hydrocarbons that are formed by substituting hydrogen with bromine on a biphenyl ring. PBBs have a molecular formula of $C_{12}H_{(10-n-m)}Br_{(n+m)}$ where n + m = 1 to 10, i.e. from monobromobiphenyl to decabromobiphenyl.

There are 209 possible structural congeners of the brominated biphenyl structure containing one or more bromines; however, only a few of these have been synthesized individually and characterized (Stepniczka, 1976; Sundström *et al.*, 1976a). The number of PBB congeners that actually exist in commercial mixtures is much lower than that of polychlorinated biphenyls (PCB) congeners.

Like for PCBs, positions 2, 2′, 6, and 6′ are called *ortho* positions, positions 3, 3′, 5, and 5′ are called *meta* positions, and positions 4 and 4′ are called *para* positions (Fig. 1.1).

The benzene rings can rotate around the 1,1′ carbon bond. The two theoretical extreme configurations are planar (angle = 0°) and perpendicular (the two benzene rings are in perpendicular planes). The degree of planarity is largely determined by the number of substitutions in the *ortho* positions. Since bromine atoms are more bulky than chlorine atoms, substitution in *ortho* positions for PBBs is much less favoured than for PCBs. The replacement of hydrogen atoms in the *ortho* positions with bromine atoms forces the benzene rings to adopt a configuration with a larger angle. The benzene rings of non-*ortho* as well as mono-*ortho* substituted PBBs adopt a small angle so that the configuration is nearly planar (Fig. 1.2).

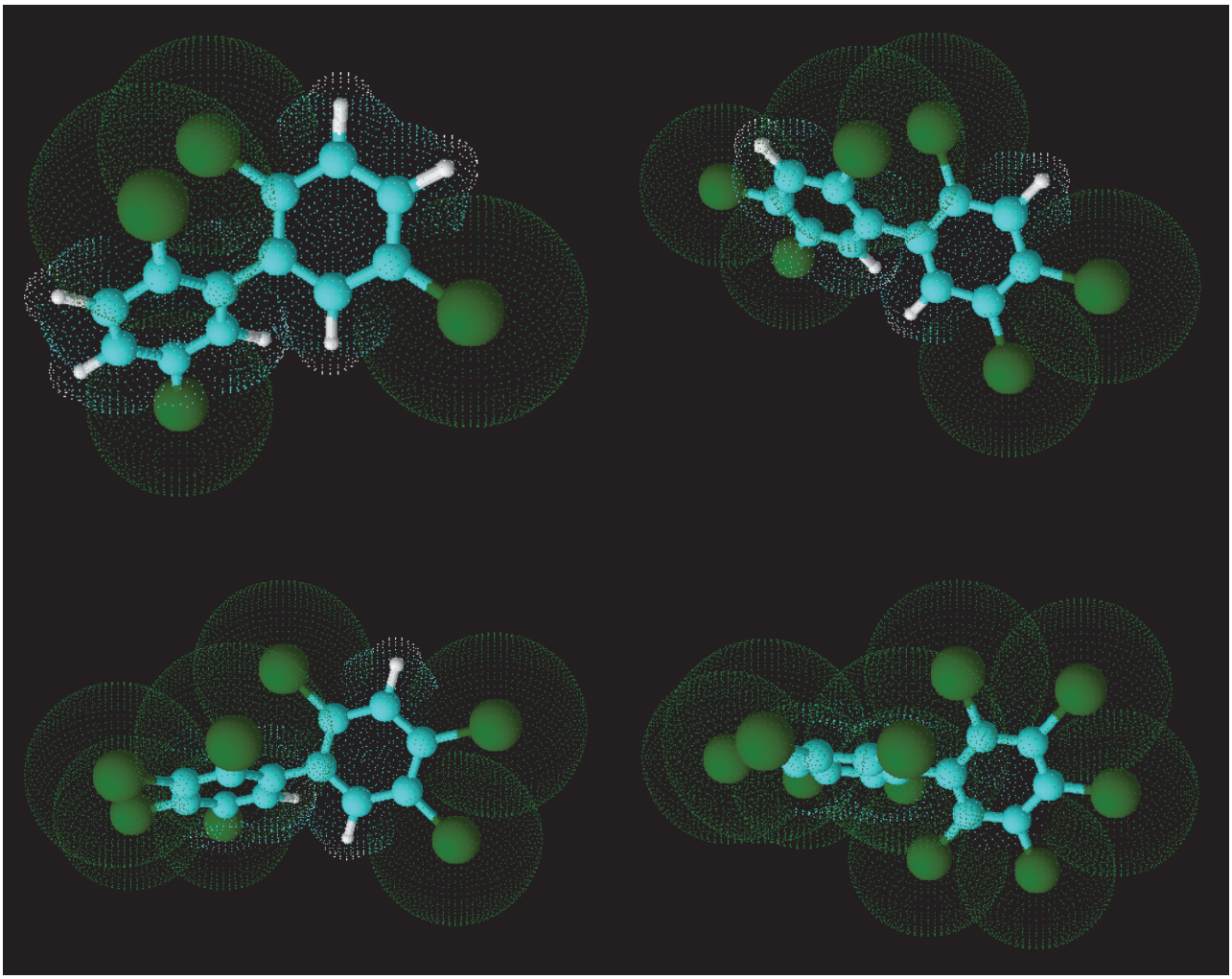
Fig. 1.1 Chemical structure of polybrominated biphenyls and the IUPAC numbering system



Hydrogen atoms in positions 2,2′,6,6′ (*ortho*), 3,3′,5,5′ (*meta*) and/or 4,4′ (*para*) may be substituted by bromine atoms; n+m = 1–10
IUPAC, International Union of Pure and Applied Chemistry
Compiled by the Working Group

The numbering of PBBs from 1 to 209 corresponds to the scheme developed for PCBs by Ballschmiter & Zell (1980) and updated in Ballschmiter *et al.* (1992), i.e. in ascending numerical order, which generally follows the rules of the International Union of Pure and Applied Chemistry (IUPAC) for substituent characterization of biphenyls (rule A-52.3 related to hydrocarbon systems) (Table 1.1). This numbering system, referred to as BZ numbering, is widely used for identifying

Fig. 1.2 Tridimensional chemical structures of PBBs



Spatial configurations of three mono-*ortho* PBBs, e.g. PBB-52 (2,2′,5,5′-tetraBB, up and left), PBB-153 (2,2′,4,4′,5,5′-hexaBB, up and right), and PBB-180 (2,2′,3,4,4′,5,5′-heptaBB, down and left), and non-coplanar configuration of one di-*ortho* PBB, e.g. PBB-209 (2,2′,3,3′,4,4′,5,5′,6,6′-decaBB, down and right)
BB, brominated biphenyl; PBBs, polybrominated biphenyls
Courtesy of Professor B. LeBizec

Table 1.1 BZ number and correspondence between the positions of bromine atoms on each phenyl ring of the PBBs (nomenclature according to Ballschmiter *et al.*, 1992)[a]

Position of bromine atom on each ring	2	3	4	2,3	2,4	2,5	2,6	3,4	3,5	2,3,4	2,3,5	2,3,6	2,4,5	2,4,6	3,4,5	2,3,4,5	2,3,4,6	2,3,5,6	2,3,4,5,6
None	1	2	3	5	7	9	10	12	14	21	23	24	29	30	38	61	62	65	116
2′	4	6	8	16	17	18	19	33	34	41	43	45	48	50	76	86	88	93	142
3′		11	13	20	25	26	27	35	36	55	57	59	67	69	78	106	108	112	160
4′			15	22	28	31	32	37	39	60	63	64	74	75	81	114	115	117	166
2′,3′				40	42	44	46	56	58	82	83	84	97	98	122	129	131	134	173
2′,4′					47	49	51	66	68	85	90	91	99	100	123	137	139	147	181
2′,5′						52	53	70	72	87	92	95	101	103	124	141	144	151	185
2′,6′							54	71	73	89	94	96	102	104	125	143	145	152	186
3′,4′								77	79	105	109	110	118	119	126	156	158	163	190
3′,5′									80	107	111	113	120	121	127	159	161	165	192
2′,3′,4′										128	130	132	138	140	157	170	171	177	195
2′,3′,5′											133	135	146	148	162	172	175	178	198
2′,3′,6′												136	149	150	164	174	176	179	200
2′,4′,5′													153	154	167	180	183	187	203
2′,4′,6′														155	168	182	184	188	204
3′,4′,5′															169	189	191	193	205
2′,3′,4′,5′																194	196	199	206
2′,3′,4′,6′																	197	201	207
2′,3′,5′,6′																		202	208
2′,3′,4′,5′,6′																			209

[a] Nomenclature of the PBBs follows that of polychlorinated biphenyls (PCBs). For several PBB congeners, the indicated structural names do not correspond strictly to the IUPAC rules (primed and unprimed numbers are interchanged). A comprehensive survey of PCB nomenclature, including IUPAC names, is given in Mills *et al.* (2007). This nomenclature includes revised numbering of congeners 107–109.

BZ, Ballschmiter & Zell; IUPAC, International Union of Pure and Applied Chemistry; PBBs, polybrominated biphenyls

individual congeners of PBBs. For example, the PBB congener 3,3′,4,4′,5,5′-hexabromobiphenyl is referred to as PBB-169. The relationship between PBB BZ number and Chemical Abstracts Service (CAS) number is given in Table 1.2.

PBBs can be categorized by degree of bromination, and compounds with the same number of bromines are called homologues. Based on the number of bromine substituents, there are 10 homologous groups of PBBs (monobromobiphenyls to decabromobiphenyls). The mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decabromo congeners can exist in 3, 12, 24, 42, 46, 42, 24, 12, 3, and 1 form(s), respectively (Table 1.3). Homologues with different patterns of substitution are referred to as isomers.

1.1.1 Chemical and physical properties of PBBs

The properties of congeners as reported by earlier investigators may be questionable due to insufficient purification of the congener. More accurate data on physical and chemical properties have been reported recently (Table 1.3; Tittlemier *et al.*, 2002). PBBs are chemically comparable to PCBs, with properties linked to bromine, which is a better leaving group in chemical reactions than chlorine. Pure single PBB compounds are mostly colourless or slightly yellowish, often odourless. The commercial products are typically white, off-white, or beige powdered solids (DiCarlo *et al.*, 1978; Tittlemier *et al.*, 2002). PBBs are characterized by low volatility (Table 1.3), which decreases with increasing bromine number (Farrell, 1980; NTP, 2011). PBBs with three or more bromines are solids (Sundström *et al.*, 1976a; de Kok *et al.*, 1977).

PBBs are nearly insoluble in water, and solubility decreases with increasing bromination. PBBs are soluble in fat (Kay, 1977) and slightly to highly soluble in various organic solvents. Partition ratios between 1-octanol and water (log K_{ow}) increase with the number of bromines (Table 1.3; IARC, 1986). Unlike PCBs, the reactivity of PBBs has not been well studied or documented in the literature. Henry's law constant for the hexabromobiphenyls ranges from 1.4×10^{-8} to 3.9×10^{-8} atm-m^3/mol.

Like PCBs, the chemical stability of PBBs is dependent, in part, on the degree and specific pattern of substitution (bromination). However, PBBs show unusual chemical stability and resistance to breakdown by acids, bases, and reducing and oxidizing agents (Pomerantz *et al.*, 1978). Several of the common isomers photodegrade with reductive debromination upon exposure to ultraviolet light (Sundström *et al.*, 1976a; Kay, 1977; Pomerantz *et al.*, 1978). All highly brominated PBB mixtures are known to debrominate rapidly upon ultraviolet irradiation (DiCarlo *et al.*, 1978). Investigations into the pyrolysis of Firemaster BP-6 in the absence of oxygen (600–900 °C) have shown that bromobenzenes and lower brominated biphenyls are formed, but no polybrominated furans. In contrast, pyrolysis in the presence of oxygen (700–900 °C) yielded some di- to heptabromodibenzofurans (O'Keefe, 1978).

1.1.2 Trade names and composition of commercial mixtures

PBB mixtures have been manufactured mainly as three homologue groups: hexabromobiphenyls, octabromobiphenyls, and decabromobiphenyls (Table 1.4; Neufeld *et al.*, 1977; ATSDR, 2004; NTP, 2011). All commercial PBB mixtures are relatively highly brominated, with bromine contents ranging from about 76% for hexabromobiphenyls to 81–85% for octa- to decabromobiphenyl mixtures (Brinkman & de Kok, 1980). Commercial PBB mixtures were produced primarily by Berk Corporation in the United Kingdom [e.g. Berkflam B-10, Flammex B-10 (decabromobiphenyls)], Chemische Fabrik Kalk [e.g. Bromkal 80–9D (nonabromobiphenyl)] and Ugine Kuhlmann [e.g. Adine 0102

Table 1.2 BZ number, bromine positions, and CAS number for individual PBBs (*n* = 209)

BZ No.	Bromine positions	CAS No.
1	2	2052-07-7
2	3	2113-57-7
3	4	92-66-0
4	2,2′	13029-09-9
5	2,3	115245-06-2
6	2,3′	49602-90-6
7	2,4	53592-10-2
8	2,4′	49602-91-7
9	2,5	57422-77-2
10	2,6	59080-32-9
11	3,3′	16400-51-4
12	3,4	60108-72-7
13	3,4′	57186-90-0
14	3,5	16372-96-6
15	4,4′	92-86-4
16	2,2′,3	
17	2,2′,4	
18	2,2′,5	59080-34-1
19	2,2′,6	
20	2,3,3′	
21	2,3,4	
22	2,3,4′	
23	2,3,5	
24	2,3,6	
25	2,3′,4	
26	2,3′,5	59080-35-2
27	2,3′,6	
28	2,4,4′	6430-90-6
29	2,4,5	115245-07-3
30	2,4,6	59080-33-0
31	2,4′,5	59080-36-3
32	2,4′,6	64258-03-3
33	2′,3,4	
34	2′,3,5	
35	3,3′,4	
36	3,3′,5	
37	3,4,4′	6683-35-8
38	3,4,5	115245-08-4
39	3,4′,5	72416-87-6
40	2,2′,3,3′	
41	2,2′,3,4	
42	2,2′,3,4′	
43	2,2′,3,5	
44	2,2′,3,5′	
45	2,2′,3,6	
46	2,2′,3,6′	
47	2,2′,4,4′	66115-57-9
48	2,2′,4,5	
49	2,2′,4,5′	60044-24-8
50	2,2′,4,6	
51	2,2′,4,6′	97038-95-4
52	2,2′,5,5′	59080-37-4
53	2,2′,5,6′	60044-25-9
54	2,2′,6,6′	97038-96-5
55	2,3,3′,4	97038-99-8
56	2,3,3′,4′	
57	2,3,3′,5	
58	2,3,3′,5′	
59	2,3,3′,6	
60	2,3,4,4′	
61	2,3,4,5	115245-09-5
62	2,3,4,6	115245-10-8
63	2,3,4′,5	
64	2,3,4′,6	
65	2,3,5,6	
66	2,3′,4,4′	84303-45-7
67	2,3′,4,5	
68	2,3′,4,5′	
69	2,3′,4,6	
70	2,3′,4′,5	59080-38-5
71	2,3′,4′,6	
72	2,3′,5,5′	
73	2,3′,5′,6	
74	2,4,4′,5	
75	2,4,4′,6	64258-02-2
76	2′,3,4,5	
77	3,3′,4,4′	77102-82-0
78	3,3′,4,5	
79	3,3′,4,5′	97038-98-7
80	3,3′,5,5′	16400-50-3
81	3,4,4′,5	59589-92-3
82	2,2′,3,3′,4	
83	2,2′,3,3′,5	
84	2,2′,3,3′,6	
85	2,2′,3,4,4′	
86	2,2′,3,4,5	
87	2,2′,3,4,5′	
88	2,2′,3,4,6	77910-04-4
89	2,2′,3,4,6′	
90	2,2′,3,4′,5	
91	2,2′,3,4′,6	
92	2,2′,3,5,5′	

Table 1.2 (continued)

BZ No.	Bromine positions	CAS No.
93	2,2′,3,5,6	
94	2,2′,3,5,6′	
95	2,2′,3,5′,6	88700-05-4
96	2,2′,3,6,6′	
97	2,2′,3′,4,5	
98	2,2′,3′,4,6	
99	2,2′,4,4′,5	81397-99-1
100	2,2′,4,4′,6	97038-97-6
101	2,2′,4,5,5′	67888-96-4
102	2,2′,4,5,6′	80274-92-6
103	2,2′,4,5′,6	59080-39-6
104	2,2′,4,6,6′	97063-75-7
105	2,3,3′,4,4′	
106	2,3,3′,4,5	
107	2,3,3′,4,5′	
108	2,3,3′,4,6	
109	2,3,3′,4′,5	
110	2,3,3′,4′,6	
111	2,3,3′,5,5′	
112	2,3,3′,5,6	
113	2,3,3′,5′,6	
114	2,3,4,4′,5	96551-70-1
115	2,3,4,4′,6	
116	2,3,4,5,6	38421-62-4
117	2,3,4′,5,6	
118	2,3′,4,4′,5	67888-97-5
119	2,3′,4,4′,6	86029-64-3
120	2,3′,4,5,5′	80407-70-1
121	2,3′,4,5′,6	
122	2′,3,3′,4,5	
123	2′,3,4,4′,5	74114-77-5
124	2′,3,4,5,5′	
125	2′,3,4,5,6′	
126	3,3′,4,4′,5	84303-46-8
127	3,3′,4,5,5′	81902-33-2
128	2,2′,3,3′,4,4′	82865-89-2
129	2,2′,3,3′,4,5	
130	2,2′,3,3′,4,5′	82865-90-5
131	2,2′,3,3′,4,6	
132	2,2′3,3′,4,6′	119264-50-5
133	2,2′,3,3′,5,5′	55066-76-7
134	2,2′,3,3′,5,6	
135	2,2′3,3′,5,6′	119264-51-6
136	2,2′,3,3′,6,6′	
137	2,2′,3,4,4′,5	81381-52-4
138	2,2′,3,4,4′,5′	67888-98-6
139	2,2′,3,4,4′,6	
140	2,2′,3,4,4′,6	
141	2,2′,3,4,5,5′	120991-47-1
142	2,2′,3,4,5,6	
143	2,2′,3,4,5,6′	
144	2,2′,3,4,5′,6	119264-52-7
145	2,2′,3,4,6,6′	
146	2,2′,3,4′,5,5′	
147	2,2′,3,4′,5,6	
148	2,2′,3,4′,5,6′	
149	2,2′,3,4′,5′,6	69278-59-7
150	2,2′,3,4′,6,6′	93261-83-7
151	2,2′,3,5,5′,6	119264-53-8
152	2,2′,3,5,6,6′	
153	2,2′,4,4′,5,5′	59080-40-9
154	2,2′,4,4′,5,6′	36402-15-0
155	2,2′,4,4′,6,6′	59261-08-4
156	2,3,3′,4,4′,5	77607-09-1
157	2,3,3′,4,4′,5′	84303-47-9
158	2,3,3′,4,4′,6	
159	2,3,3′,4,5,5′	120991-48-2
160	2,3,3′,4,5,6	
161	2,3,3′,4,5′,6	
162	2,3,3′,4′,5,5′	
163	2,3,3′,4′,5,6	
164	2,3,3′,4′,5′,6	82865-91-6
165	2,3,3′,5,5′,6	
166	2,3,4,4′,5,6	
167	2,3′,4,4′,5,5′	67888-99-7
168	2,3′,4,4′,5′,6	84303-48-0
169	3,3′,4,4′,5,5′	60044-26-0
170	2,2′,3,3′,4,4′,5	69278-60-0
171	2,2′,3,3′,4,4′,6	
172	2,2′,3,3′,4,5,5′	82865-92-7
173	2,2′,3,3′,4,5,6	
174	2,2′,3,3′,4,5,6′	88700-04-3
175	2,2′,3,3′,4,5′,6	
176	2,2′,3,3′,4,6,6′	
177	2,2′,3,3′,4,5′,6′	
178	2,2′,3,3′,5,5′,6	119264-54-9
179	2,2′,3,3′,5,6,6′	
180	2,2′,3,4,4′,5′,6	67733-52-2
181	2,2′,3,4,4′,5,6	
182	2,2′,3,4,4′,5,6′	119264-54-9
183	2,2′,3,4,4′,5′,6	
184	2,2′,3,4,4′,6,6′	119264-56-1

Table 1.2 (continued)

BZ No.	Bromine positions	CAS No.
185	2,2′,3,4,5,5′,6	
186	2,2′,3,4,5,6,6′	119264-57-2
187	2,2′,3,4′,5,5′,6	84303-49-1
188	2,2′,3,4′,5,6,6′	119264-58-3
189	2,3,3′,4,4′,5,5′	88700-06-5
190	2,3,3′,4,4′,5,6	79682-25-0
191	2,3,3′,4,4′,5′,6	
192	2,3,3′,4,5,5′,6	
193	2,3,3′,4′,5,5′,6	
194	2,2′,3,3′,4,4′,5,5′	67889-00-3
195	2,2′,3,3′,4,4′,5,6	
196	2,2′,3,3′,4,4′,5′,6	
197	2,2′,3,3′,4,4′,6,6′	119264-59-4
198	2,2′,3,3′,4,5,5′,6	
199	2,2′,3,3′,4,5,5′,6′	
200	2,2′,3,3′,4,5,6,6′	119264-60-7
201	2,2′,3,3′,4′,5′,6,6′	69887-11-2
202	2,2′,3,3′,5,5′,6,6′	59080-41-0
203	2,2′,3,4,4′,5,5′,6	
204	2,2′,3,4,4′,5,6,6′	119264-61-8
205	2,3,3′,4,4′,5,5′,6	
206	2,2′,3,3′,4,4′,5,5′,6	69278-62-2
207	2,2′,3,3′,4,4′,5,6,6′	119264-62-9
208	2,2′,3,3′,4,5,5′,6,6′	119264-63-0
209	2,2′,3,3′,4,4′,5,5′,6,6′	13654-09-6

BZ, Ballschmiter & Zell; CAS, Chemical Abstracts Service Registry; PBBs, polybrominated biphenyls
From Ballschmiter & Zell (1980), Ballschmiter *et al.* (1992)

(decabromobiphenyl)] in Germany, and Michigan Chemical Corporation, White Chemical Corporation, and Hexcel Corporation in the USA [e.g. Firemaster BP-6 (CAS No. 59536-65-1) and FF-1 (CAS No. 67774-32-7)].

The exact composition of the mixtures varies between batches (see Section 1.3; Table 1.5) and also within each batch, according to sampling (see Section 1.2). The main constituents of Firemaster are hexabromobiphenyl (63–84%) and heptabromobiphenyl (12–25%), together with lesser brominated [pentabromobiphenyl (1–11%) and tetrabromobiphenyl (0–5%)] congeners (Sundström *et al.*, 1976b; DiCarlo *et al.*, 1978; Hass *et al.*, 1978; Robertson *et al.*, 1984a) due to incomplete bromination reactions (IPCS, 1994).

At least four ^{13}C-labelled PBB congeners are available commercially.

1.1.3 Contaminants and impurities of commercial mixtures of PBBs

Mixed polybromochlorobiphenyls (PXBs), e.g. monochloropentabromobiphenyl (CAS No. 88703-30-4), have been observed as minor contaminants in Firemaster (Tondeur *et al.*, 1984). Such compounds probably result from contamination of commercial bromine by chlorine (Domino & Domino, 1980). Contaminants of the initial biphenyl feedstock may ultimately appear in commercial mixtures of PBBs. Described impurities include toluene, naphthalene, methylene biphenyl (fluorene) and various methyl biphenyls (Neufeld *et al.*, 1977). It is assumed that naphthalene for instance, present as an impurity in industrial-grade biphenyl, is brominated during production, and that the presence of numerous isomers and congeners of polybrominated naphthalenes in the final product is possible (Robertson *et al.*, 1984a). Polybrominated benzenes and a possible methylbrominated furan have also been reported to occur in Firemaster(R) (Brinkman & de Kok, 1980). Polybromodibenzo-*p*-dioxins and polybromodibenzofurans were not detected above 0.5 mg/kg in the polar fraction of Firemaster FF-1 (Hass *et al.*, 1978). In another study, O'Keefe (1978) showed that polybrominated dibenzofurans were formed during pyrolysis of Firemaster FF-1. A sample of Adine 0102 (decabromobiphenyl) contained monobromobenzodifurans at 1 mg/kg and polybromodibenzodioxins and polybromodibenzofurans at below 0.01 mg/kg (IPCS, 1994). Although PBBs are relatively stable, highly brominated congeners are susceptible to photolytic debromination when they are exposed to ultraviolet light (see Section 1.1.1).

Table 1.3 Physical and chemical properties of homologue groups of PBBs

Homologue group	CAS No.	Formula	No. of isomers	BZ No.	Relative molecular mass	Melting point (°C)	Solubility (μg/L)	Volatility (Pa at 25 °C)[a] (calculated)[b]	Log K_{ow}[a]
Monobromobiphenyls	26264-10-8	$C_{12}H_9Br_1$	3	1–3	232.9				4.6 (PBB-1)
Dibromobiphenyls	27479-65-8	$C_{12}H_8Br_2$	12	4–15	311.8				
Tribromobiphenyls	51202-79-0	$C_{12}H_7Br_3$	24	16–39	390.7				
Tetrabromobiphenyls	40088-45-7	$C_{12}H_6Br_4$	42	40–81	469.6			5.0×10^{-4}	6.5 (PBB-52)
Pentabromobiphenyls	56307-79-0	$C_{12}H_5Br_5$	46	82–127	548.5			2.7×10^{-5}	7.2 (PBB-101)
Hexabromobiphenyls	36355-01-8	$C_{12}H_4Br_6$	42	128–169	627.4	72	11–3[b]	1.4×10^{-6}	8.0 (PBB-153)
Heptabromobiphenyls	35194-78-6	$C_{12}H_3Br_7$	24	170–193	706.3			1.1×10^{-7}	8.3 (PBB-180)
Octabromobiphenyls	27858-07-7	$C_{12}H_2Br_8$	12	194–205	785.2	200–255	20–30	8.7×10^{-9}	8.7 (PBB-194)
Nonabromobiphenyls	27753-52-2	$C_{12}H_1Br_9$	3	206–208	864.1			1.7×10^{-9}	9.1 (PBB-206)
Decabromobiphenyls	13654-09-6	$C_{12}Br_{10}$	1	209	943.0	380–386	Insoluble	3.2×10^{-10}	9.4 (PBB-209)

[a] Values are examples of one congener in the homologue group.

[b] Calculated using Advanced Chemistry Developement (ACD/Labs) Software VII.02 (©1994–2010 ACD/Labs).

[c] Tittlemier *et al.* (2002)

BZ, Ballschmiter & Zell; PBBs, polybrominated biphenyls

From IARC (1978), EFSA (2010)

Table 1.4 Trade names of commercial PBB mixtures

Main PBB congeners	Trade name
Hexabromobiphenyls	Firemaster FF-1
	Firemaster BP-6
	"Hexabromobiphenyl"
Octabromobiphenyls	BB-8
	Bromkal 80
	Bromkal 80–9D
	Octabromobiphenyl FR 250 13A
	Technical octabromobiphenyl
Decabromobiphenyls	Adine 0102
	Berkflam B-10
	Flammex B-10
	HFO 101
	Technical decabromobiphenyl

PBB, polybrominated biphenyl
From IPCS (1994)

1.2 Analysis

Given that the physical and chemical characteristics of PBBs are similar to those of PCBs, sampling techniques for PBBs are essentially identical to those described for PCBs. However, a considerably smaller body of scientific literature is available on PBBs than on PCBs, and not all environmental matrices have been studied.

As with all brominated flame retardants, samples should not be exposed to sunlight, since PBBs are unstable when exposed to ultraviolet radiation (Brinkman & de Kok, 1980).

1.2.1 Environmental and food samples

PBBs were analysed together with polychlorinated dibenzodioxins/polychlorinated dibenzofurans (PCDD/PCDFs), PCBs and polybrominated diphenyl ethers (PBDEs) in the same air samples (Wang *et al.*, 2010a). Particles and gaseous phase were collected on glass-fibre filters and polyurethane foam, respectively, as described for PCBs. After Soxhlet extraction with toluene, extracts were treated with acid and purified on acid silica. In a second clean-up step on alumina, several fractions were obtained, including fractions with non-polar PBBs/PCBs and polar PBBs/PCBs.

Most studies combine the analysis of PBBs with that of PBDEs, for example, for soil (Wang *et al.*, 2009) or sediment samples (de Boer *et al.*, 2003). After freeze-drying and sieving, the samples were mixed with copper for sulfur removal, as described for analysis of PCBs in soils and sediments, Soxhlet extracted, and cleaned up on silica-gel columns. Besides Soxhlet extraction, pressurized liquid extraction has also been used for sediment analysis (Zhao *et al.*, 2010). Clean-up processes included multilayer columns of acid, neutral and basic silica gel, as well as gel-permeation chromatography and subsequent treatment with sulfuric acid (de Boer *et al.*, 2003; Zhao *et al.*, 2010).

Water samples were analysed in terms of influent and effluent samples of waste-water treatment plants (de Boer *et al.*, 2003). The samples were filtered or centrifuged to separate suspended particulate matter from the water phase, and then treated as for sediment samples, i.e. Soxhlet extraction of the particulate phase, gel-permeation chromatography, acid treatment and clean-up with silica gel.

Several studies have analysed fish samples, with a focus on the most biaccumulative congener PBB-153. The samples were dried, either by freeze-drying (Gieroń *et al.*, 2010) or with sodium sulfate (de Boer *et al.*, 2003), and extracted using a Soxhlet apparatus (de Boer *et al.*, 2003; Zhu & Hites, 2004) or direct extraction with dichloromethane in an extraction column (Luross *et al.*, 2002). Lipids were removed by acid treatment, using acid silica gel (Gieroń *et al.*, 2010) or direct treatment with sulfuric acid (de Boer *et al.*, 2003). Further clean-up and fractionation techniques included gel-permeation chromatography, column clean-up with alumina or with neutral/basic silica and the dialysis through semi-permeable membrane devices (Gieroń *et al.*, 2010).

Table 1.5 Composition of commercial PBB mixtures

Composition	Commercial mixture (range of % bromination)			
	Hexabromobiphenyls	Octabromobiphenyls[a]		Decabromobiphenyls
Tetrabromobiphenyls	2–5			
Pentabromobiphenyls	1–11			
Hexabromobiphenyls	63–84	0	1–2	0–4
Heptabromobiphenyls	12–25	1–7	23–27	0–4
Octabromobiphenyls	0–2	25–57	46–72	0–7
Nonabromobiphenyls		0–28	34–65	2–11
Decabromobiphenyls		2–9	0–1	71–97

[a] The Working Group noted that the "octabromobiphenyls" include two classes of mixtures with different ranges of composition. In IPCS (1994), they are called "octanonabromobiphenyls."
PBB, polybrominated biphenyl
From de Kok *et al.* (1977), IPCS (1994)

Gieroń *et al.* (2010) also analysed other food products using this method; pure lipid samples (butter, pork adipose tissue) were melted before further processing. Analytical methods for PBBs in food were summarized by EFSA (2010), describing solvent extraction, lipid removal and additional clean-up by column chromatography.

1.2.2 Biological samples

Several studies have been conducted on the Michigan cohort, which was established in 1976 following the accidental contamination of cattle feed with PBBs and subsequent exposure of local residents (see Section 1.4). Human serum samples were analysed at enrolment in the cohort (1976–1978) and in the follow-up studies until 1993 (Givens *et al.*, 2007). After protein denaturation with methanol, PBBs were extracted with hexane:diethyl ether (1:1), and extracts were cleaned on Florisil (Burse *et al.*, 1980; Needham *et al.*, 1981). More recent blood analyses did not deviate much from these procedures, but applied a higher degree of automatization (Frederiksen *et al.*, 2010). The first step was generally protein denaturation, often using formic acid. Solid-phase extraction was a common extraction technique, followed by lipid removal using H_2SO_4 (Wang *et al.*, 2010b) or clean-up on acid and neutral silica (Sjödin *et al.*, 2004a) and/or Florisil (Sandanger *et al.*, 2007; Wang *et al.*, 2010b). Blood analyses have generally focused on PBB-153 as the congener with the most pronounced bioaccumulation.

For the analysis of human milk, EFSA (2010) described solvent extraction and solid-phase extraction, followed by the same clean-up method as for food samples. Adipose tissue has also been analysed for PBBs, in combination with PBDEs and PCBs (Fernandez *et al.*, 2007; Miceli *et al.*, 1985). The samples were Soxhlet-extracted using toluene or hexane:acetone. Lipids were removed on acid silica gel. Further clean-up included neutral and basic silica gel and a fractionation into different compound groups. Target PBBs and PBDEs were separated from PCDD/PCDFs on an activated carbon column and further cleaned up on alumina (Fernandez *et al.*, 2007), while separation of PCBs from the brominated compounds was achieved by different solvents eluting the compounds from the silica-gel column (Zhao *et al.*, 2009).

1.2.3 Instrumental analysis

As described for PCBs, the instrumental analysis of PBBs is basically independent of the original matrix, although selectivity and

sensitivity should be considered. It also is worth noting that PBBs often are analysed in conjunction with other substances, making multicompound methods desirable. For example, studies including the determination of dioxins and furans or other coplanar molecules besides PBBs have used high-resolution gas chromatography (HRGC) in combination with high-resolution mass spectrometry (HRMS) (Wang *et al.*, 2010a), which is a highly selective and very sensitive technique. Owing to these advantages, HRGC–HRMS has also been applied in analyses of environmental and biological samples that have combined determination of PBDEs and PBBs (Luross *et al.*, 2002).

Gas chromatography–mass spectrometry (GC–MS) with electron-capture negative ionization (ECNI) is a common method in the analysis of PBBs, providing high sensitivity (de Boer, 1999). However, PBB-153, the predominant congener in biological matrices, co-elutes with the PBDE BDE-154 on several GC columns. As both congeners are detected by the mass fragments m/z = 79 and m/z = 81, chromatographic separation must be achieved to avoid miscalculations, for example on a 60 m capillary column (Zhu & Hites, 2004). On the other hand, a shorter column is advisable for the determination of PBB-209, which is not stable at elevated temperatures (de Boer, 1999; Zhao *et al.*, 2009). As the GC–MS (ECNI) method relies on the detection of the bromide ion, the use of ^{13}C-labelled standards is excluded.

GC–MS with electron impact (EI) ionization has also been used for environmental and biological samples (Zhao *et al.*, 2009; Gieroń *et al.*, 2010), allowing detection of molecular ions and specific fragments. However, this technique is described as being 10 times less sensitive than GC–MS (ECNI) and GC with electron capture detection (ECD) (de Boer, 1999). GC–ECD was generally used in the early studies (Burse *et al.*, 1980), and although still applied in environmental and biological analyses (Wang *et al.*, 2009; Wang *et al.*, 2010b), is increasingly replaced by GC–MS techniques.

1.2.4 PXBs

Only few studies have analysed mixed chlorinated/brominated biphenyls, as recently reviewed by Falandysz *et al.* (2012). Eight native congeners and their ^{13}C-labelled analogues were commercially available for this analysis. Other studies, which rely on custom-made analytical standards, analysed fewer congeners (Ohta *et al.*, 2008a).

PXBs were generally analysed together with other compounds, primarily PCDD/PCDFs, by extending existing methods. Additional fractionation steps on carbon columns were included to isolate the PXB congeners. They were analysed by HRGC–HRMS using isotope dilution quantification, although not all studies used matching native and labelled congeners (Falandysz *et al.*, 2012).

PXBs have been analysed in food (Fernandes *et al.*, 2011), fish (Ohta *et al.*, 2008a) and human milk (Gómara *et al.*, 2011), focusing on five to eight congeners. Fernandes *et al.* (2011) described an extensive sample clean-up involving acid and basic silica gel and several carbon columns, to isolate non-*ortho* and mono-*ortho* PXBs, respectively.

1.3 Production and uses

Production of PBBs generally involves the reaction of biphenyl with bromine and chlorine in a solvent with aluminum chloride as a catalyst (Neufeld *et al.*, 1977). PBBs are also formed as impurities during the production of other brominated compounds. For example, PBBs are formed during the production of decabromodiphenyl oxide because of the presence of diphenyl as an impurity in the starting material, diphenyl oxide (Neufeld *et al.*, 1977). PBBs are also present as impurities in PBDEs (Hanari *et al.*, 2006).

Commercial PBB mixtures were manufactured primarily as flame retardants. In the USA and Europe, PBB mixtures were produced and sold commercially as products with a specific bromine content. Although these commercial products are generally referred to as "hexabromobiphenyl," "octabromobiphenyl," and "decabromobiphenyl," these are misnomers, since each commercial product contained numerous congeners with different numbers of bromine substitutions (see Section 1.1.2). The composition of commercial products varied substantially across lots and producers (Table 1.5), particularly for octabromobiphenyls, many of which may actually have consisted primarily of nonabromobiphenyls.

PBBs were produced by three companies in the USA during the 1970s only. One company in Michigan produced hexabromobiphenyl, and two companies in New Jersey produced octabromobiphenyl and decabromobiphenyl. Total production in the USA was estimated at 13 million pounds [5896 tonnes], 88% of which was hexabromobiphenyl (Table 1.6). Production of hexabromobiphenyl in Michigan was halted in 1974 subsequent to the contamination of animal feed (see Section 1.4), and production of octabromobiphenyl and decabromobiphenyl was discontinued a few years later (Neufeld *et al.*, 1977). In the United Kingdom, PBBs were produced until 1977; in Germany, until the mid 1980s; and in France, until 2000, with only decabromobiphenyl being produced in the later years (EFSA, 2010). No information was available on production volumes in Europe or elsewhere.

In addition to these commercial producers, a few speciality chemical companies produced PBBs with lower bromine content, mostly monobromobiphenyls and dibromobiphenyls, in small batches of 0.1–1 kg, to be used in functional fluids (Neufeld *et al.*, 1977).

The major uses of PBBs were in acrylonitrile-butadiene-styrene (ABS) plastics (used, for example, for housing television sets and other electronic machines), in coatings and lacquers, and in polyurethane foam. Based on a PBB content of 10%, an estimated 118 million pounds [53.5 tonnes] of PBB-containing ABS plastic could have been made during 1971–1975, which would be about 5% of the total production of ABS plastics during those years (Neufeld *et al.*, 1977). In these uses, PBB flakes were physically blended into the product, not chemically incorporated into a polymer (Neufeld *et al.*, 1977). This raises the concern that they could volatilize or leach out of the product (ATSDR, 2004).

Recently, PBBs were detected in electronic waste in cable coatings, stuffing powder for electronic components, and circuit boards, suggesting uses in such equipment. PBBs in these items consisted mostly of mono-, di-, or tribromobiphenyls (Zhao *et al.*, 2008). [The Working Group noted that this is not consistent with hexa-, octa-, and decabromobiphenyl being the only commercial mixtures with large-scale production and use. This suggests that PBBs of predominantly low bromine content may have been used in electronic equipment in China, which was previously unknown. The Working Group noted the small sample size.]

PXBs can be formed when chlorine and bromine are present during the combustion of PCBs or PBBs. PXBs are also contaminants of commercial PCB mixtures, resulting from the presence of bromine gas as a trace contaminant of the chlorine gas used in the production of PCBs. Dioxin-like PXBs can be formed during pyrolysis or photolysis of PBDEs. PXBs are not known to be produced intentionally (Falandysz *et al.*, 2012).

1.4 Environmental occurrence and human exposure

PBBs can enter the general environment from several sources: loss during production of PBBs, loss during manufacture of products containing PBBs, disposal and reprocessing

Table 1.6 Production volumes of commercial PBB mixtures in the USA

Year	Hexabromobiphenyls	Octa- and decabromobiphenyls	Total (tonnes)
1970	0.95	14.0	24
1971	84	14.0	98
1972	1007	14.5	1022
1973	1764	162	1927
1974	2214	48.0	2263
1975	0	77	77
1976	0	365	365
1977	0	> 0	> 0
Total (%)	5079 (88%)	> 7046 (12%)	> 5775 (100%)

PBB, polybrominated biphenyl
Adapted from DiCarlo *et al.* (1978)

of products containing PBBs, and accidental releases. Products from the 1970s that contained PBBs have generally reached the end of their useful life and would have been recycled, disposed of in landfills, or incinerated.

It was estimated that the production of 805 000 pounds [365 tonnes] of decabromobiphenyl in the USA in 1976 resulted in 5% loss to the environment: 900 pounds [408 kg] to air, 0.0037 pounds [1.7 g] to wastewater, and 40 250 pounds [18.3 tonnes] to landfills as solid waste (Neufeld *et al.*, 1977). [The wastewater calculation for decabromobiphenyl considered only the solubility of PBBs in water, which is low, and not the likelihood that solid PBB particles could also be discharged in wastewater.] Similar figures were not located on losses from production of hexabromobiphenyl, but discharges in 1974 from the plant in Saint Louis, Michigan, USA, were estimated at 0.11 kg per day (Archer *et al.*, 1979). [The hexabromobiphenyl mixture has a higher vapour pressure and a higher fraction of congeners with low bromine content, which generally would be more volatile.]

Contamination with PBBs has been high in Michigan, owing to accidental widespread contamination of farms, foods, and residents. In early 1973, several bags of the hexabromobiphenyl mixture "Firemaster" were mistaken for "NutriMaster," an animal feed supplement containing magnesium oxide. Both products were manufactured at the same plant. A shortage of preprinted paper bags at the plant led to 10–20 50-pound [22.7 kg] bags of Firemaster being packed in NutriMaster paper bags and sent to animal-feeding operations (Michigan Department of Community Health, 2011).

PBB concentrations in the contaminated feed were estimated to be between 4000 and 13 500 ppm [mg/kg]. In addition, there were four routes of indirect contamination with PBBs (Kay, 1977):

- Processing or mixing of clean feed in contaminated grain elevators (chicken feed became contaminated in this way).
- Incorporation of material from contaminated animals that died (and were sent to a rendering plant) into animal feed.
- Processing of contaminated milk into milk powder for feeding young animals.
- Swapping of feed by farms and feed mills.

The error was not discovered until April 1974, by which time the PBBs had entered the food chain through contaminated milk, eggs and other dairy products, contaminated beef products, and contaminated swine, sheep, and chickens. More than 500 Michigan farms were quarantined and 30 000 cattle, 1500 sheep, and 1.5 million chickens were destroyed. Inventories of 800 tons [725 tonnes] of animal feed, 18 000

pounds [8.1 tonnes] of cheese, 2500 pounds [1.1 tonnes] of butter, 5 million eggs, and 34 000 pounds [15.4 tonnes] of dried milk products were also destroyed (Michigan Department of Community Health, 2011).

PBBs have generally been replaced by PBDEs. PBBs, however, are present as impurities in PBDEs. On the basis of PBDE production and use in 2001, it was estimated that potential global annual emissions of PBBs would be 40 kg (Hanari *et al.*, 2006). [The Working Group noted that the Michigan incident involved 500–1000 pounds [225–450 kg] of PBBs.] There were few reports of recent concentrations of PBBs in environmental media; most investigations of brominated compounds have focused on PBDEs and newer brominated alternatives for use as flame retardants.

1.4.1 Environmental fate

In the environment, PBBs occur as mixtures of congeners whose compositions differ from that of the commercial products. This is because after release into the environment, composition changes over time because of partitioning, chemical transformation, and bioaccumulation. PBB congeners are highly persistent in the environment and in biological tissues. Air and water are the transport media.

Primarily hydrophobic, PBBs adsorb strongly to soils and sediments. Hydrophobic adsorption generally increases with the bromine content of the PBB congener and the organic content of the soil or sediment. In water, PBBs with high bromine content are less soluble and more likely to attach strongly to sediment. PBB congeners with low bromine content are more likely to be soluble in water. In air, PBB congeners are generally not very volatile, and are less volatile than the corresponding PCB congeners (Pijnenburg *et al.*, 1995).

PBBs are lipophilic and can be dissolved in solvents. Liquid solvents that may be present in landfills or contaminated sites are capable of solubilizing PBBs and carrying them to distant locations (ATSDR, 2011). PBBs are 200 times more soluble in landfill leachate than in distilled water, and more soluble in creek water than in purified water. These results are correlated with the levels of dissolved organic compounds (Lewis, 1981).

PBBs degrade slowly in the environment. In 1988, sediments from Pine River, Michigan, contained 10–12% PBB congeners that are not found in Firemaster, consistent with bromines being selectively removed from *meta* and *para* positions. Microorganisms are capable of debrominating PBB congeners, although this process can be inhibited by organic co-contaminants, petroleum products, and heavy metals. Ultraviolet light can degrade PBB congeners, especially at *ortho* positions (Pijnenburg *et al.*, 1995).

Bioconcentration and bioaccumulation are important processes for PBBs in water. Bioconcentration from water is more pronounced for PBBs with low bromine content. The pattern for bioaccumulation from food is more complex (Pijnenburg *et al.*, 1995).

1.4.2 Natural occurrence

PBBs and PXBs are not known to occur in nature.

1.4.3 Air and dust

In the past, PBBs were released into the air during manufacture. Air emissions through vents were reported to be $2–3 \times 10^{-6}$ mg/L (Neufeld *et al.*, 1977; Vorkamp *et al.*, 2005; Wang *et al.*, 2010a). PBBs were detected at a concentration of 6×10^{-11} mg/L in air samples near a PBB-manufacturing plant, although the same concentration was measured downwind and crosswind from the plant (DiCarlo *et al.*, 1978).

Another potential source of PBBs in air is from incineration of products containing PBBs. Pyrolysis of commercial hexabromobiphenyl

produces small amounts of lesser brominated biphenyls (Thoma & Hutzinger, 1987).

Near a municipal solid waste incinerator in Taiwan, China, PBB concentrations in air were reported as 149–556 fg.N/m^3 (Wang *et al.*, 2010a).

PBB-153 was not detected in house dust in Bavaria, Germany, with a limit of detection of 10 ng/g (Kopp *et al.*, 2012).

No other information was available on recent concentrations of PBBs in outdoor or indoor air.

PXBs have been found in exhaust gas from waste incinerators and in marine sediments in Japan (Ohta *et al.*, 2009).

1.4.4 Water, sediment, and sewage sludge

No data were available on recent concentrations of PBBs in surface water, groundwater, or sediment.

In the past, PBBs were released into water during manufacture. PBBs have been found in a variety of surface waters, groundwater, and sediments. This is most probably due to solid PBB particles being carried along with the water, as PBBs are rather insoluble in water. As might be expected, concentrations in river water and sediments tend to decrease with distance downstream from the source (Table 1.7). Although most sampling has occurred in and around PBB-production plants, PBBs have also been detected in wastewater from the production of decabromodiphenyl oxide (in which PBBs are a byproduct) and in effluents and sludge from a municipal wastewater-treatment plant (Neufeld *et al.*, 1977; Daso *et al.*, 2012).

Wastewater discharges from the Michigan Chemical Corporation plant provide an instructive example. In 1972, PBB particles were measured in wastewater from the plant at concentrations up to 98–503 µg/L. In 1974, after actions to reduce the discharge of PBB particles, concentrations of up to 100 µg/L persisted. In 1975, after PBB production was halted, concentrations as high as 150 µg/L were measured irregularly. In 1977, after removal of contaminated soil from bagging and loading areas of the plant, concentrations fell to below 1 µg/L (Hesse & Powers, 1978).

In 1999, PBBs were not found in suspended particulate matter, sediments, sewage treatment plant influents and effluents, fish, and mussels in the Netherlands (limit of detection, 0.1–1 µg/kg dry weight; 1–10 µg/kg for PBB-209) (de Boer *et al.*, 2003).

1.4.5 Soil

PBBs are found at nine sites on the United States Environmental Protection Agency's National Priorities List ("Superfund" sites), four of them in Michigan (ATSDR, 2011), including the site of the Michigan Chemical Corporation plant. In 1975, soil from bagging and loading areas of the Michigan Chemical Corporation plant contained PBBs at 3500 and 2500 mg/kg, respectively (Hesse & Powers, 1978) and soil near the two PBB-production plants in New Jersey, USA, contained PBBs at 40–3100 and 750–2800 µg/kg, respectively (DiCarlo *et al.*, 1978). Soil samples from 28 fields that received manure from Michigan's most highly contaminated dairy herds had the following distribution of PBB concentrations: below detection limit of 0.1 µg/kg, two fields; 0.1–0.9 µg/kg, six fields; 1–9 µg/kg, nine fields; 10–99 µg/kg, five fields; 100–371 (maximum) µg/kg, six fields. PBBs were not detected in two control farm fields. PBBs also were below the detection limit of 0.3 µg/kg in corn, alfalfa, and sudangrass that was being grown in the contaminated fields (Jacobs *et al.*, 1978).

Soil samples near facilities that processed PBBs in California and West Virginia, USA, contained PBBs at up to 36 000 and 12 µg/kg, respectively (Zweidinger & Pellizzari, 1980).

In 2007, PBB concentrations were measured in soil collected from four villages in China where electronic-waste disassembly sites were located. The median PBB concentration was

Table 1.7 Concentrations of PBBs in various environmental media

Medium	Site	PBB concentration	Reference
Wastewater	Original discharge from Michigan PBB plant	98–503 µg/L	Hesse & Powers (1978)
	After some action to reduce discharges	≤ 100 µg/L	Hesse & Powers (1978)
	After PBB production stopped	Erratic, ≤ 150 µg/L	Hesse & Powers (1978)
	After soil cleanup at plant	< 1 µg/L	Hesse & Powers (1978)
Wastewater	Decabromodiphenyl oxide production	< 0.1–10 µg/L	Neufeld *et al.* (1977)
Storm sewer water	Near New Jersey PBB plant	92 µg/L	DiCarlo et al. (1978)
Swamp water	Runoff from New Jersey PBB plant	135 µg/L	DiCarlo et al. (1978)
River water	Near effluent discharge of Michigan PBB plant	13 µg/L	Archer *et al.* (1979)
	13 km downstream	0.01 µg/L	Archer et al. (1979)
	12 miles downstream of Michigan PBB plant	0.01–0.07 µg/L	Hesse & Powers (1978)
	25–29 miles downstream	ND (< 0.1 µg/L)	Hesse & Powers (1978)
Sediment	Near New Jersey PBB plant	100 mg/kg	Archer *et al.* (1979)
	At Michigan PBB plant	77 mg/kg	Hesse & Powers (1978)
	Just downstream of plant	6.2 mg/kg	Hesse & Powers (1978)
	29 miles downstream	0.1 mg/kg	Hesse & Powers (1978)
Groundwater	Near landfill from Michigan PBB plant	0.1–0.2 µg/L	DiCarlo *et al.* (1978)
Drainage ditch, catch basin	Near landfill from Michigan PBB plant	1.2 mg/kg	DiCarlo et al. (1978)
Effluent	Wastewater treatment plant in South Africa	< 18.4 ng/L	Daso *et al.* (2012)
Sewage sludge	Wastewater treatment plant in South Africa	< 9.97 ng/g	Daso et al. (2012)

ND, not detected; PBBs, polybrominated biphenyls

22 µg/kg (range, 18–58 µg/kg; n = 6) compared with 11 µg/kg (range, 8–19 µg/kg; n = 3) in a remote village at a distance of 30 km where there were no electronic-waste operations. Mono-, di-, and tribromobiphenyls predominated, with PBB-2 being the single most abundant congener (Zhao *et al.*, 2008).

Soils from urban and rural sites in the United Kingdom contained dioxin-like PXBs, with concentrations an order of magnitude greater in urban soil than in rural soil. Concentrations of four mono-*ortho* PXBs were 0.90, 0.49, and 0.17 ng/kg in urban soil, and 0.050, 0.025, and 0.024 ng/kg in rural soil (Fernandes *et al.*, 2011).

1.4.6 Bioaccumulation in wildlife and plants

Field studies in several species show that PBBs are taken up by wildlife. Near the Michigan Chemical Corporation plant, PBBs have contaminated fish in the Pine River downstream from the plant. PBBs were detected in 25 out of 27 composite samples, where each sample represented one out of seven fish species taken at one out of four sampling stations. The highest concentration was 1.33 mg/kg in skinless carp fillets. PBBs were not detected in fish samples collected upstream of the plant above a dam that prevents upstream fish movement, and PBBs were not detected in fish samples from a nearby river. PBBs were detected in the majority of wild ducks collected within 2 miles of the plant. Near a PBB-production plant in New Jersey, a turtle was found to contain hexabromobiphenyl at 20 µg/kg. More recently, PBB congeners, predominantly PBB-153, were detected in lake trout from the Great Lakes (DiCarlo *et al.*, 1978; Hesse & Powers, 1978; Luross *et al.*, 2002).

The strong bioaccumulation potential of PBBs was demonstrated in caged fish in the Pine River near the Michigan Chemical Corporation plant. After 2 weeks of exposure, concentrations in caged fathead minnows were up to 10 000 times those in the surrounding river water. No

PBBs were detected in fish sampled at a control station 3 miles upstream of the plant (Hesse & Powers, 1978).

PBBs have been measured in a variety of marine species. These positive measurements, made at sites far from industrial sources of PBBs, indicate that PBBs can be transported great distances. The detection of PBBs in sperm whales indicate that these compounds have reached deep ocean waters, as sperm whales are not usually found in shallow seas (Jansson *et al.*, 1987, 1993; de Boer *et al.*, 1998). [It is noteworthy that whenever a species has been sampled in the same area in different years, the levels have increased.]

PBB-153 was detected in the eggs of six species of wild aquatic birds, one species of wild terrestrial bird, and two species of captive birds in China. Levels ranged from non-detectable to 0.7 ng/g lipid weight (Vorkamp *et al.*, 2005; Gao *et al.*, 2009).

PBBs were taken up by root vegetables grown in soil artificially contaminated with PBBs. Most of the residue was on the vegetable surface and could be removed by dipping in acetone. Uptake was higher by plants grown in a sandy soil than in a clay soil with higher organic content. This is consistent with the tendency of PBBs to adsorb to soils with high organic content. No PBBs were detected in orchard grass or in carrot tops (Jacobs *et al.*, 1976; Chou *et al.*, 1978; DiCarlo *et al.*, 1978).

1.4.7 Food and estimated dietary intake

Soon after the Michigan incident was discovered, sampling on contaminated farms revealed BBP concentrations as high as 595 mg/L in milk, 4600 mg/kg in poultry tissue, 60 mg/kg in eggs, and 2700 mg/kg in cattle tissue (Kay, 1977).

In milk from contaminated dairy herds, the concentration of PBBs was estimated to have reached 6000 mg/L after 15 days exposure, declining to 1800 mg/L 15 days after exposure ceased, and to 160 mg/L after another 230 days (Fries *et al.*, 1978).

Based on monitoring of PBBs in food and a call for data, the European Food Safety Authority (EFSA) evaluated results on 794 food samples collected during 2003–2009 from Belgium, Estonia, France, Ireland, Spain, and the United Kingdom (5643 analytical results covering 16 PBB congeners). Due to the large number of non-detects for individual congeners in individual samples, EFSA focused the analysis on seven congeners: those with less than 80% non-detects, plus the three coplanar PBBs PBB-77, PBB-126, and PBB-169. The EFSA analysis provided the ranges of these PBB congeners in four food categories (Table 1.8; EFSA, 2010).

Based on recent estimates of mean and high dietary exposure to the different food categories, EFSA calculated average and high-end intakes of five PBB congeners from food. For children aged 1–3 years, the principal source was milk, with an intake of 32 or 64 pg/kg bw per day for average or high-end consumers, respectively. For children aged 3–6 years, the principal source was fish and seafood, with an intake of 15 or 66 pg/kg bw per day for average or high-end consumers, respectively. For adults, the principal source was fish and seafood, with an intake of 8 or 40 pg/kg bw per day, respectively. Food supplements would add another 39 pg/kg bw per day (Table 1.9; EFSA, 2010). [Including only five congeners in the analysis could lead to a substantial underestimate of intake, especially if a potent congener were omitted because it is not widely distributed across a broad food category.]

PXBs were detected in nine species of domestic or imported fish and one species of marine mammal from food markets in Japan. Toxic equivalency (TEQ), calculated as a weighted sum of the concentrations of five non-*ortho* congeners, ranged from 0.09 to 1.3 pg/g wet weight. When compared with TEQs calculated as a weighted sum of the concentrations of 12 PCB congeners, the predicted toxicity levels attributable to PXBs or PCBs were generally within one order of magnitude. The authors concluded that

Table 1.8 Mean concentration (pg/g wet weight) of seven PBB congeners in foods in Europe

PBB congener	Meat, meat products		Milk, dairy products		Fish, seafood		Food for infants, small children	
	LB	UB	LB	UB	LB	UB	LB	UB
PBB-49	–	–	–	–	1.32	1.52	–	–
PBB-52	0.06	0.39	0.01	0.58	4.19	4.31	–	–
PBB-101	0.06	0.39	0.01	0.58	1.55	1.86	–	–
PBB-153	–	–	–	–	0.81	18.9	0	7.64
PBB-77[a]	0.0002	0.0055	0	0.0051	0.0168	0.0226	–	–
PBB-126[a]	0.0045	0.0107	0	0.004	0.0005	0.0087	–	–
PBB-169[a]	0	0.0077	0	0.0071	0	0.0088	–	–

[a] Original data on non-*ortho* PBBs were reported with considerably lower LOQs. Therefore the number of digits after the decimal point has been extended to four in this table, for descriptive reasons.
LB, lower bound; PBB, polybrominated biphenyl; UB, upper bound
From EFSA (2010)

dioxin-like PXBs cannot be considered a negligible contributor to human health risks. The authors also remarked that the lack of availability of analytical standards made it impossible to identify and quantify most PXB congeners (Ohta *et al.*, 2008b). PXBs have been detected in seal blubber in ng/g lipid concentrations (Falandysz *et al.*, 2012).

Non-*ortho* and mono-*ortho* PXBs have been detected in several foods in the United Kingdom, including soft cheese, cow milk, duck eggs, lamb, liver, vegetables, river fish, and marine fish (Fernandes *et al.*, 2011).

1.4.8 Exposure of the general population

In 1976–1977, venous blood samples were drawn from several groups of Michigan residents and analysed for PBBs with a limit of detection of 1 µg/L. They showed a wide range of concentrations within each group of residents, and distinctly higher mean concentrations for three groups: chemical workers engaged in PBB production and their families, residents of quarantined farms, and direct recipients of products from such farms (Table 1.10; Landrigan *et al.*, 1979). [The Working Group noted that the inclusion of family members of chemical workers was likely to have reduced the reported levels for the chemical workers: while Table 1.10 (Landrigan *et al.*, 1979) shows a range of non-detect to 1240 µg/L for 216 chemical workers and their family members, Table 1.11 (Anderson *et al.*, 1978a) shows that none of the 55 chemical workers had a level less than 1.1 µg/L.]

In another report, the distribution of serum PBB concentrations in Michigan chemical workers was shown to be distinctly higher than that of farm residents (Table 1.11; Anderson *et al.*, 1978a).

In the same study, maternal serum, cord serum, and milk were sampled for 65 Michigan mothers potentially exposed to PBBs. They showed a wide range of PBB concentrations and a strong bioaccumulation of PBBs in human milk (Table 1.12; Landrigan *et al.*, 1979).

In 1993, PBBs and other persistent compounds were measured in the serum of people who reported eating at least one meal per week of sport fish caught in the Great Lakes. The overall mean serum PBB concentration in 30 subjects was 0.4 µg/L. When stratified by lake, serum concentrations were highest for people who ate fish from Lake Huron (mean, 0.6 µg/L; range, 0.1–1.7 µg/L), followed by Lake Michigan (mean, 0.4 µg/L; range, 0.04–1.0 µg/L) and Lake Erie (mean,

Table 1.9 Estimates of daily exposure to PBBs from food in Europe

Population	Food category	PBB congener	Average consumers (pg/kg bw per day)		High consumers (pg/kg bw per day)	
			LB	UB	LB	UB
Infants	Human milk	PBB-153	[620, 920]	[920, 1400][a]	–	–
	Ready-to-eat meal	PBB-153	–	0.17, 0.64[b]	–	–
Children aged 1–3 yr	Milk and dairy products	PBB-52	0.34	16.1	0.69	32.1
		PBB-101	0.41	16.2	0.82	32.3
Children aged 3–6 yr	Fish and other seafood	PBB-49	0.76	0.88	3.28	3.79
		PBB-52	2.44	2.50	10.4	10.7
		PBB-77	0.01	0.01	0.04	0.06
		PBB-101	0.90	1.08	3.86	4.63
		PBB-153	0.47	11	2.01	47
	Meat and meat products	PBB-52	0.23	1.66	0.42	2.97
		PBB-101	0.23	1.66	0.42	2.97
Adults	Fish and other seafood	PBB-49	0.39	0.45	1.97	2.28
		PBB-52	1.23	1.26	6.27	6.45
		PBB-77	0.01	0.01	0.03	0.03
		PBB-101	0.46	0.54	2.32	2.78
		PBB-153	0.24	5.53	1.21	28.2
	Meat and meat products	PBB-52	0.1	0.74	0.25	1.76
		PBB-101	0.1	0.74	0.25	1.76
	Milk and dairy products	PBB-52	0.05	1.91	0.1	4.84
		PBB-101	0.04	1.91	0.12	4.86
Adults; specific groups of the population	Fish with > 8% fat; daily intake of 179 g fishmeat	PBB-49	–	–	9.61	11.22
		PBB-52	–	–	34.4	35
		PBB-77	–	–	0.06	0.09
		PBB-101	–	–	12.2	14.1
		PBB-153	–	–	4.33	89
	Supplements with fatty acids daily intake of 15 mL	PBB-49	–	–	2	10.4
		PBB-52	–	–	3	4.5
		PBB-77	–	–	0.01	0.02
		PBB-101		–	3	4.8
		PBB-153	–	–	3.8	18.9

[a] Results reported from a study in Finish and Danish human milk samples, respectively (Shen *et al.*, 2008); the values refer to the mean intake for average and high consumers.

[b] Those estimates refer to two upper bound exposures estimated from the only two available consumption surveys.

bw, body weight; LB, lower bound; PBBs, polybrominated biphenyls; UB, upper bound; yr, year

From EFSA (2010)

0.2 µg/L; range, 0.06–0.7 µg/L). When stratified by state, serum concentrations were highest for residents of Michigan (mean, 0.7 µg/L; range, 0.11–1.7 µg/L), followed by Ohio (mean, 0.2 µg/L; range, 0.06–0.7 µg/L) and Wisconsin (mean, 0.05 µg/L; range, 0.04–0.06 µg/L). The stronger contrasts by state are consistent with Michigan dairy products being the source of PBB contamination and with Wisconsin producing most of its own dairy products (Anderson *et al.*, 1998). [Michigan's Lower Peninsula has long shorelines on Lake Michigan and Lake Huron, and a very short shoreline on Lake Erie. Water from Lake Michigan drains into Lake Huron, which drains

Table 1.10 Serum concentrations of PBBs in residents exposed as a result of the Michigan incident

Population group	Participation rate (%)	*n*	Serum concentration (μg/L)		
			Range	Mean	Median
PBB chemical workers and their families	78.0	216	0–1240	43.0	4.5
Residents on quarantined farms	95.6	1750	0–1900	26.9	4.0
Direct recipients of food products from quarantined farms	95.1	1114	0–659	17.1	3.0
Residents on farms with PBB contamination below quarantine limits	95.0	44	1–13	3.5	2.0
Self-referred residents on farms with PBB contamination below quarantine limits or persons who had eaten food produced on such farms	–	242	0–24	3.5	2.0
Self-referred volunteers who had no direct connection with contaminated farms	–	273	0–111	3.2	1.0
Total		3639	0–1900	21.2	3.0

n, total number; PBBs, polybrominated biphenyls
From Landrigan *et al.* (1979). Copyright (c) 1979, John Wiley and Sons.

into Lake Erie. The Pine River, which borders the Michigan Chemical Corporation plant, flows into Lake Huron (Fig. 1.3).]

Serum PBB concentrations were measured in two cohorts of Michigan children: a "farm exposure" cohort consisting of 87 children enrolled in long-term studies of PBB- or PCB-contaminated farm products and a "fish exposure" cohort consisting of 236 children born to women who had consumed PBB-contaminated fish from Lake Michigan. Serum PBB concentrations were measured in the early 1980s when the children were aged 4 years. The percentages of children in the farm and fish exposure cohorts who had detectable serum PBBs (> 1 μg/L) were 21% and 13%, respectively. Significant predictors of serum PBB concentrations in children aged 4 years were weeks of nursing, PBB concentrations in maternal milk, and PBB concentrations in cord serum (Table 1.13; Jacobson *et al.*, 1989).

In the Michigan Long-Term PBB Study, 27% of children born to mothers exposed to PBBs through contaminated food had detectable serum PBB concentrations (> 1 μg/L). Risk factors for detectable serum PBB concentrations were maternal serum concentrations of 8 μg/L or more, nursing for 5.5 months or more, maternal age at childbirth of 28 years or more, and being born during the period of PBB exposure. Infants who nursed for 5.5 months or more were six times more likely to have detectable concentrations of

Table 1.11 Distribution of serum concentrations of PBBs in people exposed as a result of the Michigan incident

Group	*n*	Percentage with each group of serum concentrations (%)				
		0–1.1 μg/L	1.1–9.9 μg/L	10–99.9 μg/L	100–999 μg/L	> 1000 μg/L
PBB chemical workers	55	0	51	31	13	5
Farm residents	524	23	59	14	4	0.4
Random male farmers and consumers	109	12	64	17	6	1

PBBs, polybrominated biphenyls
Adapted from Anderson *et al.* (1978a)

Table 1.12 Concentrations of PBBs in maternal serum, cord serum, and breast milk in residents exposed as a result of the Michigan incident

Group	*n*	PBB concentration (µg/L)			Ratio to maternal serum (range)
		Range	Mean	Median	
Maternal serum	52	0–1150	26.2	2.5	–
Cord serum	58	0–104	3.2	1.0	7.04[a] (1.5–10.3)
Milk (lipid basis)	32	32–93 000	3614	225	122.0 (62.2–256.7)

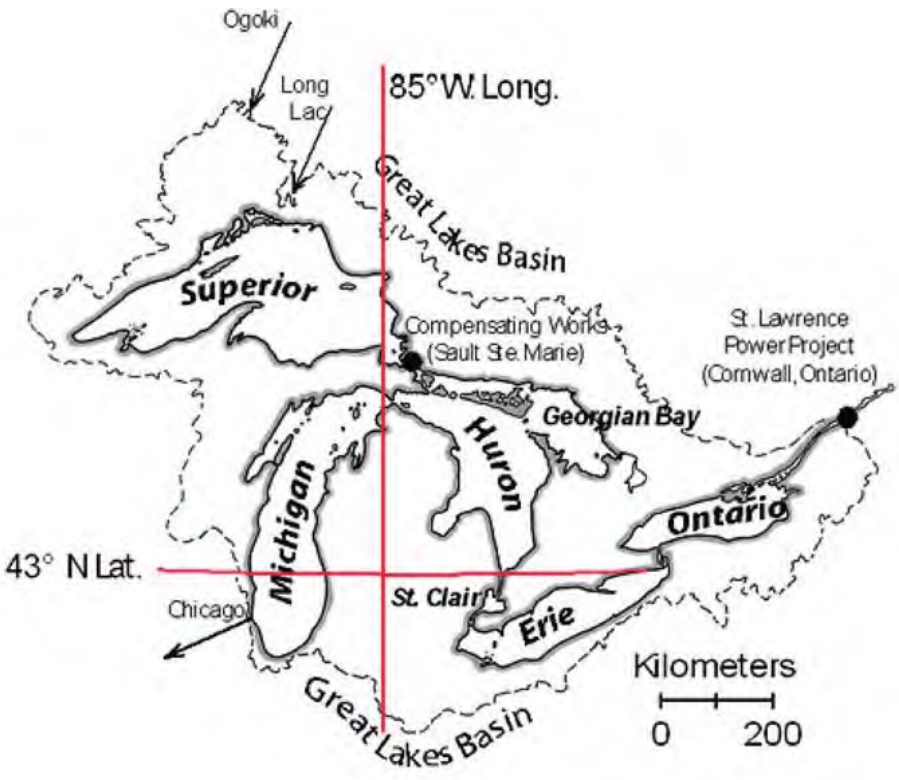
[a] [7.04 was reported by the authors, but this may refer to an inverse ratio]
PBBs, polybrominated biphenyls
Adapted from Landrigan *et al.* (1979). Copyright © 1979, John Wiley and Sons.

serum PBBs (95% CI, 2.0–19.6) (Joseph *et al.*, 2009).

Three out of nine samples of human hair [inferred to be from near the Michigan or the New Jersey PBB plant] contained PBBs at concentrations of 0.03, 1, and 2 ppm (Archer *et al.*, 1979).

Apart from the Michigan incident, several surveys have reported concentrations of the congener PBB-153 in the general population (Table 1.14). Analysis of data from *National Health and Nutrition Examination Survey* (NHANES) 2003–2004 indicated that PBB-153 was detected in 83% of samples (Sjödin *et al.*, 2008). Nonetheless, analysis of archived serum pools indicated that PBB-153 concentrations had been decreasing in the USA, the only country

Fig. 1.3 The Great Lakes basin area



From NOAA, Great Lakes Environmental Research Laboratory (GLERL, 2014)

Table 1.13 Serum concentrations of PBBs in children (age 4 years) born to Michigan mothers who ate contaminated farm products or fish

Cohort	*n*	Percentage of mothers with serum concentration > 1 ng/mL (%)	Serum concentration (ng/mL)	
			Mean	Range
Exposure from farm products	80	21.3	2.95	1.0–9.5
Exposure from fish	205	12.7	2.44	1.0–6.4

PBBs, polybrominated biphenyls
Adapted from Jacobson *et al.* (1989)

with multiple reports; in contrast, concentrations of the PBDEs that replaced PBBs have been increasing (Sjödin *et al.*, 2004b). One survey from Sweden reported much lower levels of PBBs than in the USA (Sjödin *et al.*, 2001). [A difficulty in interpreting PBB-153 concentrations is that this congener co-elutes with PBDE-154.]

In 2007, PBBs were detected in 90% and 50%, respectively, of maternal plasma and umbilical-cord plasma samples from women in Denmark with median PBB-153 concentrations of 181 and 68.6 pg/g lipid weight (Frederiksen *et al.*, 2010).

In 2003, PBB concentrations in human adipose tissue were collected from 20 women undergoing surgery for malignant and benign diseases in Spain. The mean sum of concentrations of seven PBB congeners was 0.358 ng/g lipid, with PBB-153 contributing 79% (Fernandez *et al.*, 2007). The ratio between human serum and adipose concentrations was estimated to be between 1:140 and 1:260 (Hakk & Letcher, 2003).

In 2007, PBB concentrations were measured in human hair collected from four villages in China where electronic-waste disassembly sites were located. Operations included recovering metals by burning cables, stripping metals in open-pit acid baths, and removing electronic components from circuit boards by heating over a grill, resulting in leakage, evaporation, runoff, and leaching of chemicals. PBB concentrations were elevated in two of these villages, compared with a control village 30 km away where there were no electronic-waste operations. Mono-, di-, and tribromobiphenyls predominated, with PBB-2 being by far the single most abundant congener (Table 1.15; Zhao *et al.*, 2008). In tissue samples of cancer patients in the same area, PBBs were detected in all samples of kidney, liver, and lung (n = 19, 55, and 7 samples, respectively). Median concentrations of PBBs in these tissues were 194, 193, and 145 ng/g lipid, respectively (Zhao *et al.*, 2009).

PXBs were detected in milk from seven mothers in Japan, 5 and 30 days after delivery. TEQs, calculated as a weighted sum of the concentrations of five non-*ortho* congeners, ranged from 0.42 to 1.41 pg/g (Ohta *et al.*, 2007). In a second study, five dioxin-like PXBs were measured in 20 mothers in Japan, 1 week after delivery. The sum of concentrations of five non-*ortho* congeners ranged from 12 to 350 pg/g lipid weight, with an average of 57 pg/g. The authors suggested that seafood was an important source of these congeners, as 3′,4′,5′-tribromo-3,4-dichlorobiphenyl was a major congener seen in fish and in human milk (Ohta *et al.*, 2008a).

PXBs were detected in milk from nine mothers in Madrid, Spain. The sum of concentrations of five non-*ortho* and three mono-*ortho* congeners was 0.45 pg/g lipid weight (Gómara *et al.*, 2011).

1.5 Occupational exposure

Historically, workers involved in the production of PBBs, PBB-containing plastics, and PBB-containing plastic products could have

Table 1.14 Concentrations of PBB-153 in human serum from surveys not directly related to the Michigan incident

Group, country	Year	*n*	Concentration (ng/g lipid)		Reference
			Median	Range	
Blood donors, USA	1988	12	19 pmol/g	4.2–84	Sjödin *et al.* (2001)
Female cleaners, Sweden	1997	20	0.59 pmol/g	0.25–1.4	Sjödin *et al.* (2001)
Archived serum pools, USA	1985–9	9	8.0	2.6–9.4	Sjödin *et al.* (2004b)
	1990–4	14	5.3	1.0–8.6	
	1995–9	10	4.7	1.9–10	
	2000–2	7	3.3	1.4–5.5	
NHANES, persons aged ≥ 12 years, USA	2003–4	2062	2.3	–	Sjödin *et al.* (2008)

n, total number; NHANES, National Health and Nutrition Examination Survey; PBB, polybrominated biphenyl

been exposed to PBBs via inhalation of dust and vapour, and/or via dermal contact.

After the Michigan incident in 1973, several studies showed that workers in PBB industries were exposed to high concentrations of PBBs. Several of these studies also showed high exposure among workers on dairy farm from the surrounding areas (Bekesi *et al.*, 1979a; Landrigan *et al.*, 1979; Stross *et al.*, 1979, 1981; Wolff *et al.*, 1979b). The Michigan population was more highly exposed than populations in other states.

In one PBB-manufacturing plant in the USA, 8 hour time-weighted average (TWA) air concentrations of PBB of 0.18 and 0.23 mg/m^3 were reported in 1977 (Bialik, 1982). These samples were collected in the manufacturing area and comprised mostly decabromobiphenyls. Surface-wipe measurements showed concentrations of up to 8 mg/100 cm^2. One surface-wipe sample collected on top of a table in the eating area had 0.1 mg/100 cm^2, which showed that in addition to inhalation and dermal routes of exposure, hand-to-mouth exposure was also possible. At the time of the survey, 95% of plant production consisted of decabromobiphenyl (18%) and decabromobiphenyl oxide (77%).

Several studies reported PBB concentrations in serum and adipose tissues (Table 1.16). Analysis of blood from employees at a hexabromobiphenyl-manufacturing company showed concentrations from 0.015 mg/L (after 3 months of exposure) to 0.085 mg/L (after 26 months of exposure) ($n = 6$), and of 0.006 mg/L in a supervisor employed for 38 months (Kay, 1977).

A study of exposure among PBB-manufacturing workers at another plant in the USA presented a detailed comparison of serum PBB concentrations by type of work activity (Bahn *et al.*, 1980a). A significantly higher number of PBB workers had detectable PBB concentrations compared with other workers (steelworkers and wiremen; 35.9% compared with 12.2%); also, the PBB workers had significantly higher serum PBB concentrations.

A clinical study including 55 exposed Michigan farm residents, 11 Michigan chemical workers and 46 non-exposed Wisconsin farmers showed that 7 out of 10 non-production workers (who did not participate in the production and handling of PBBs) had plasma concentrations of < 1 ng/mg (0.13–0.23 ng/mg protein) (Bekesi *et al.*, 1979a), while four production workers (who worked in the production and bagging section of the plant for several years, having been directly exposed to PBBs) had a PBB plasma concentration of around 10 ng/mg protein.

Clinical findings were reported for workers ($n = 55$) who manufactured Firemaster BP-6 from 1970 to 1974 in the USA (Anderson *et al.*, 1978a).

Table 1.15 PBB concentrations in human hair collected in villages around electronic waste-disassembly sites in China

Village	*n*	Concentrations (ng/g)	
		Median	Range
Tongshan	8	26	18–41
Panlang	11	29	14–55
Xiazheng	9	44	20–66
Xinqiu	8	58	24–103
Yandang (control)	4	26	22–32

PBBs, polybrominated biphenyls
Adapted from Zhao *et al.* (2008)

Other halogenated fire retardant chemicals were also produced at this plant. All 250 employees were invited to participate in the study, in particular those who had worked directly in the PBB-production area. Serum PBB concentrations were reported in ranges: 28 workers had serum PBB concentrations of 1.1–9.9 mg/L, 17 workers had 10–99.9 mg/L, 7 workers had 100–999.9 mg/L, and three workers were above 1000 mg/L.

In a study of liver function among farmers (n = 364) in Michigan after the accident, Anderson *et al.* (1978b) found the distribution of serum PBB concentrations to be: non-detectable–0.2 μg/L, 16 farmers; 0.21–1.0 μg/L, 69 farmers; 1.1–5.0 μg/L, 169 farmers; 5.1–10.0 μg/L, 52 farmers; and > 10.0 μg/L, 58 farmers.

One study assessed the distribution of PBB homologues (penta-, hexa-, and heptabromobiphenyls) in sera from dairy farmers and chemical-manufacturing workers. The relative concentration of two pentabromobiphenyls, both found in the Firemaster FF-1, differed widely between the two groups (Wolff & Aubrey, 1978). This would suggest different levels of exposure to the same mixture, but also that the mixture had been transformed between PBB manufacture and reaching the dairy farm, different routes of exposure, with farmers ingesting PBB partially metabolized in the animal food source. Compared with the original chemical product, one pentabromobiphenyl congener was not found in serum, possibly due to its relative ease of metabolism and excretion.

In the early 1990s, China started to process imported electronic waste ("e-waste") such as scrap metals, obsolete electric capacitors, household appliances, electric generators, and cable wires. Currently, 90% of all e-waste is imported from Japan, the USA, western European countries, and the Russian Federation. The recycling operations involve open-air burning, acid leaching, and physical dismantling by hammer, chisel, screwdriver, and bare hands. In 2008–2009, Chinese workers in the e-waste recycling industry were surveyed for serum levels of thyroid hormone, thyrotropin (thyroid-stimulating hormone), and brominated flame retardant (Wang *et al.*, 2010b). Workers exposed occupationally to brominated flame retardant during dismantling and recycling activities, non-occupationally exposed people, and controls were included in the study. The concentration of PBBs in sera of these occupationally exposed workers was 3.02 ng/mL plasma (n = 239). This value was lower than for farmers in the area surrounding the e-waste site [ΣPBB, 4.34 ng/mL plasma (n = 39)], but higher than for the controls [ΣPBB, 1.43 ng/mL plasma (n = 116)].

Table 1.16 Concentrations of PBBs in serum and adipose tissue of occupationally exposed workers

Year	Group	*n*	Concentration in µg/L				Reference
			Geometric mean	Median	Range	Limit of detection	
Serum – farm workers							
1976	Exposed farm workers	46	14	NR	1–180	NR	Stross *et al.* (1979)
Serum – chemical manufacturing[a]							
1975	Workers	7	NR	NR	6–85	NR	Kay (1977)
1976	Workers	28	48	NR	NR	NR	Stross *et al.* (1981)
1976	Workers, all	55	NR	NR	1.1–1729	NR	Wolff & Aubrey (1978), Wolff *et al.* (1979a)
	Production workers	10	603.9	108.4	NR	1	
	Non-production workers	45	16.5	6.1	NR	1	
1976	Workers	14	230	12	1–1530	< 0.2	Wolff *et al.* (1979b)
1978	Workers	14	227	22	1–1363	< 0.2	Wolff et al. (1979b)
1976–7	Workers and families	216	43.0	4.5	ND–1240	1	Landrigan *et al.* (1979)
1975–80	Workers (men)	29	25.4	20	1–1200	1	Eyster *et al.* (1983)
1978	Workers	35	NR	NR	ND–1340	NR	Bahn *et al.* (1980a, b)
1979	Production workers	4	NR	NR	10–10.2	1	Bekesi *et al.* (1979a)
1979	Non-production workers	7	NR	NR	0.13–0.23	1	Bekesi et al. (1979a)
Serum – e-waste recycling							
2008–9	Workers	236	*ΣPBB*				Wang *et al.* (2010b)
			3.02	NR	NR	NR	
			PBB-209				
			0.34	NR	ND–2.54	NR	
			PBB-103				
			0.67	NR	ND–4.96	NR	
			PBB-77				
			2.01	NR	ND–189.17	NR	
Adipose tissue – chemical manufacturing[a]					*(µg/kg)*		
1976	Production workers	7	196 490	46 940	5 000–580 000	500	Wolff *et al.* (1979a)
	Non-production workers	20	3880	2490	500–10 000	500	
1975–80	Workers (men)	29	5290	6000	400–350 000	1	Eyster *et al.* (1983)
NR	Workers	25	9330	NR	[300–80 000]	NR	Brown *et al.* (1981)
NR	Workers	28	NR	NR	12 820	NR	Stross *et al.* (1981)

[a] All plants studied were in Michigan and manufactured primarily "hexabromobiphenyl", except for the study by Bahn *et al.* (1980b), which studied a plant in New Jersey manufacturing mono- and deca-bromobiphenyl.
ND, not detected; NR, not reported; PBBs, polybrominated biphenyls

1.6 Regulations and guidelines

In 1974, the United States Food and Drug Administration established tolerance limits of 1.0 mg/kg (fat-weight basis) for PBBs in milk and meat fat, and 0.1 ppm in eggs, which were soon afterwards lowered to 0.3 mg/kg and 0.05 ppm, respectively (ATSDR, 2004).

In 1983, the European Union directed that PBBs may not be used in textile articles intended to come in contact with the skin (EFSA, 2010).

In 2002, the European Parliament directed that electrical and electronic equipment in the European Union may not contain PBBs at concentrations greater than 0.1%. Only six substances are restricted to such a degree, the other five being lead, mercury, cadmium, hexavalent chromium, and PBDEs. Plastic containing brominated flame retardants must be removed and treated separately from waste electrical and electronic equipment (EC, 2011).

The commercial product "hexabromobiphenyl" has been included in the Convention on Long-range Transboundary Air Pollution since 1998, and in the Stockholm Convention on Persistent Organic Pollutants since 2009. Parties to these conventions have agreed to take measures to eliminate the production and use of these pollutants (EFSA, 2010).

PBBs are not regulated as contaminants in food or as undesirable substances in animal feed. No other national or international regulations or guidelines were available.

2. Cancer in Humans

Data on the carcinogenicity of PBBs in humans are available from follow-up of a cohort of individuals in Michigan, USA, who were exposed as a result of an industrial incident in 1973 in which PBBs were accidentally mixed with cattle feed, and from one occupational study of chemical workers potentially exposed to several brominated compounds.

The Michigan cohort includes residents of contaminated farms, PBB-manufacturing workers, and people who consumed food from contaminated farms. The 3899 participants were followed by the Michigan Department of Public Health (Landrigan *et al.*, 1979). Two nested case–control studies were designed in this cohort. Hoque *et al.* (1998) evaluated the association between site-specific risks of cancer and serum PBB concentrations. In the follow-up of the cohort until 1993, 195 primary cancers were identified in 187 people. Controls were 696 randomly selected cancer-free individuals who were frequency matched to cases by sex and age. Baseline serum PBB concentrations were measured using standard methods. This study found a dose–response relationship for cancer of the digestive system (liver, stomach, oesophagus, pancreas). Odds ratios (ORs) for digestive cancers were 8.23 (95% CI, 1.27–53.3), 12.3 (95% CI, 0.80–191), and 22.9 (95% CI, 1.34–392), respectively, for serum PBB categories of 4–20 ppb, 21–50 ppb, and > 50 ppb after adjustment for age, sex, family cancer history, cigarette smoking, alcohol drinking, and baseline serum PCB concentration. Odds ratios for cancer of the breast based on the same categorization of exposure were 2.41 (95% CI, 0.92–6.30), not estimable due to zero exposed cases, and 1.39 (95% CI, 0.16–12.5). The analysis for serum PBB concentration and risk of lymphoma adjusted for all covariates except family history and baseline serum PCB concentration also showed a dose–response relationship, with corresponding odds ratios of 3.85 (95% CI, 0.32–46.2), 19.6 (95% CI, 1.52–253), and 48.9 (95% CI, 4.09–585). [This was a unique cohort that provided important information about the effects of PBBs. Positive associations were observed, but quantitative interpretation of the findings was hampered by small numbers, particularly in the analysis of lymphoma, where the referent group contained

only one case, leading to very wide confidence intervals. The excess risk for cancers of the digestive system was based on small numbers of cases at a wide variety of sites.]

Henderson *et al.* (1995) further examined the association between cancer of the breast and serum PBB concentration in a case–control study with 1925 women enrolled in the Michigan cohort. Twenty women who developed cancer of the breast were matched on race and age to 290 control women. The risk of cancer of the breast was elevated among women with serum PBB concentrations of 2.0–3.0 ppb (OR, 3.3; 95% CI, 0.8–13), and 4.0 ppb or greater (OR, 3.2; 95% CI, 0.8–13) compared with women with < 2.0 ppb after adjustment for body-mass index (BMI), history of cancer in a female relative, and other risk factors for cancer of the breast. [This study was informative despite its small size, given the paucity of information available on populations exposed to PBBs.]

Wong *et al.* (1984) conducted a mortality study in a historical cohort of white male chemical workers employed between 1935 and 1976. The workers' potential exposure to several chemicals, including PBBs, was categorized as more highly exposed (routine exposure) and less exposed (non-routine exposure). No detailed analysis of PBB exposure was presented. A total of 91 workers were classified as potentially exposed on a routine basis, and none died during the study period; among the 237 non-routinely exposed workers, 2 deaths were observed versus 6.4 expected, one of which was due to cancer of the large intestine (standardized mortality ratio, SMR, 10, [95% CI, 0.3–55]). This case of cancer of the large intestine was observed among 87 people who worked in the research laboratories and were classified as non-routinely exposed to PBBs (SMR, 80.4 [95% CI, 2.53–557]). [The study was uninformative because of the crude exposure classification and the small number of deaths in the PBB-exposed workers.]

3. Cancer in Experimental Animals

PBBs were previously evaluated for carcinogenicity in experimental animals (IARC, 1978, 1986). In the 1978 evaluation, the Working Group determined that there was inadequate evidence to classify PBBs. However, the 1986 evaluation determined that there was *sufficient evidence* in experimental animals for the carcinogenicity of commercial mixtures of PBBs. Since that time, new data had become available, and were taken into account in the present evaluation. Only data from original research have been summarized in the tables.

3.1 Mouse

See Table 3.1

3.1.1 Oral administration

The United States National Toxicology Program (NTP) studied the carcinogenic potential of Firemaster FF-1 (see Section 1 for composition) in mice when administered orally (NTP, 1983). Groups of 50 male and 50 female B6C3F$_1$ mice were given Firemaster FF-1 at a dose of 0 (corn oil), 0.1, 0.3, 1.0, 3.0, or 10.0 mg/kg body weight (bw) per day by gavage on five consecutive days per week for 6 months. The mice were then observed for an additional 18 months after treatment, i.e. 24 months in total (lifetime observation). There was a statistically significant increase ($P < 0.01$) in the incidence of hepatocellular carcinoma in males and females at 10 mg/kg bw per day: 21 out of 22 (95%) versus 12 out of 25 (48%; controls) in males; 7 out of 8 (88%) versus 0 out of 13 (controls) in females. The incidence of hepatic neoplasms appeared to be dose-dependent. Liver tumours were observed primarily in those groups of mice to which FF-1 was given in doses sufficient to induce hepatic toxicity. There was a trend towards an increase in the incidence of thyroid follicular cell adenoma in females treated

Table 3.1 Studies of carcinogenicity with PBBs in mice

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%) and/or multiplicity of tumours	Significance	Comments
Oral administration				
Mouse, B6C3F$_1$ (M, F) 30 mo Gupta *et al.* (1983a), NTP (1983), Silberhorn *et al.* (1990), EFSA (2010)	Firemaster FF-1 at 0 (corn oil), 0.1, 0.3, 1.0, 3.0, or 10.0 mg/kg bw per day in corn oil, 5 d/wk, for 25 wk; observed for total of 24 mo 50 mice/group; age 7–8 wk	*Males* Neoplastic nodules: 2/25 (8%), 1/27 (4%), 4/24 (17%), 2/25 (8%), 2/23 (9%), 1/22 (5%) Hepatocellular carcinoma: 12/25 (48%), 8/27 (30%), 8/24 (33%), 12/25 (48%), 15/23 (65%), 21/22 (95%)* *Females* Hepatocellular adenoma: 0/13, 2/19 (11%), 0/15, 1/11 (9%), 1/17 (6%), 1/8 (12%) Hepatocellular carcinoma: 0/13, 0/19, 2/15 (13%), 2/11 (18%), 3/17 (18%), 7/8 (88%)*	 *$P < 0.01$ *$P < 0.01$	Firemaster FF-1 blended with 2% calcium trisilicate Hyperplasia/adenoma of the follicular cells of the thyroid was not considered a major finding because of the small sample size and low incidence. Shortened survival time in mice at 10.0 mg/kg bw per day.
Mouse, C57BL/10ScSn (M) 12 mo Smith *et al.* (1990, 1995)	Inferon (iron-dextran): 600 mg/kg, s.c., followed by Firemaster BP-6 in diet [dose, NR] for 2 mo, followed by control diet for 10 mo Group 1: Fe (–) Group 2: Fe (+) Group 3: Fe (–) + Firemaster BP-6 Group 4: Fe (+) + Firemaster BP-6 7–19 mice/group; age 7–10 wks	Hepatocellular carcinoma: 0/15, 0/16, 0/7, 0/7 Hepatocellular nodules: 0/15, 0/16, 1/7 (14%), 4/7 (57%)	 NS	Purity, NR

Table 3.1 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%) and/or multiplicity of tumours	Significance	Comments
Transplacental and perinatal exposure				
Mouse, B6C3F_1 (M, F) 2 yr Chhabra *et al.* (1993), NTP (1993)	Perinatal exposure (F_0): Firemaster FF-1 at 0, 3, 10, or 30 ppm in feed from 60 d before breeding until weaning of the F_1 generation. Adult exposure (F_1): F_1 given same diet as F_0 through gestation, lactation, and 4 wk after weaning (age 8 wk), then diets containing Firemaster FF-1 at 0, 3, 10, or 30 ppm, 7 d/wk for 105 wk F_0:F_1 – 0:0, 0:10, 0:30 ppm (adult exposure only) F_0:F_1 – 0:0, 30:0 ppm (perinatal exposure only) F_0:F_1 – 0:10, 10:10, 30:10 ppm (perinatal plus adult exposure) F_0:F_1 – 0:30, 30:30 ppm (perinatal plus adult exposure) 60 mice/group	**F_0:F_1 – 0:0, 0:10, 0:30 ppm (adult exposure only)** *Males* Hepatocellular adenoma: 9/50 (18%), 48/49 (98%)*, 42/50 (84%)* Hepatocellular carcinoma: 8/50 (16%), 30/49 (61%)*, 36/50 (72%)* Hepatocellular adenoma or carcinoma (combined): 16/50 (32%), 48/49 (98%)*, 48/50 (96%)* *Females* Hepatocellular adenoma: 4/50 (8%), 39/50 (78%)*, 46/48 (96%)* Hepatocellular carcinoma: 1/50 (2%), 22/50 (44%)*, 35/48 (73%)* Hepatocellular adenoma or carcinoma (combined): 5/50 (10%), 42/50 (84%)*, 47/48 (98%)* **F_0:F_1 – 0:0, 30:0 ppm (perinatal exposure only)** *Males* Hepatocellular adenoma: 9/50 (18%), 31/50 (62%) Hepatocellular carcinoma: 8/50 (16%), 17/50 (34%) Hepatocellular adenoma or carcinoma (combined): 16/50 (32%), 40/50 (80%)	*$P < 0.001$ $P < 0.001$ (trend) *$P < 0.001$ $P < 0.001$ (trend) *$P < 0.001$ $P < 0.0001$ (trend) *$P < 0.001$ $P < 0.001$ (trend) *$P < 0.001$ $P < 0.001$ (trend) *$P < 0.001$ $P < 0.001$ (trend) $P < 0.001$ $P = 0.03$ $P < 0.001$	Purity, NR All mice at 0:30 or 30:30 ppm died before the end of the study. The survival of females at 30:10 ppm was lower than that of controls. Survival of males at 3:3 ppm was greater than that of controls. Other microscopic changes included hepatocyte cytomegaly, fatty change, clear cell focus, eosinophilic focus, hepatocyte necrosis, bile duct hyperplasia and hepatocyte cytological alteration.

Table 3.1 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%) and/or multiplicity of tumours	Significance	Comments
Mouse, B6C3F$_1$ (M, F) 2 yr Chhabra et al. (1993), NTP (1993) (cont.)		*Females*		
		Hepatocellular adenoma: 4/50 (8%), 19/50 (38%)	$P < 0.001$	
		Hepatocellular carcinoma: 1/50 (2%), 4/50 (8%)	NS	
		Hepatocellular adenoma or carcinoma (combined): 5/50 (10%), 21/50 (42%)	$P < 0.001$	
		F$_0$:F$_1$ – 0:10, 10:10, 30:10 ppm (perinatal + adult exposure)	**Effect of perinatal exposure on the effect of adult exposure at 10 ppm (compared with 0:10)**	
		Males		
		Hepatocellular adenoma: 48/49 (98%), 46/49 (94%), 48/50 (96%)	NS	
		Hepatocellular carcinoma: 30/49 (61%), 31/49 (63%), 40/50 (80%)*	*$P = 0.01$	
		Hepatocellular adenoma or carcinoma (combined): 48/50 (98%), 46/49 (94%), 48/50 (96%)	NS	
		Thyroid follicular cell adenoma: 0/49, 0/48, 5/48 (10%)*	*$P = 0.02$ $P = 0.003$ (trend)	
		Females		
		Hepatocellular adenoma: 39/50 (78%), 38/50 (76%), 47/50 (94%)*	*$P = 0.005$ $P < 0.001$ (trend)	
		Hepatocellular carcinoma: 22/50 (44%), 26/50 (52%), 44/50 (88%)*	*$P < 0.001$ $P < 0.001$ (trend)	
		Hepatocellular adenoma or carcinoma (combined): 42/50 (84%), 44/50 (88%), 50/50 (100%)*	*$P < 0.001$ $P < 0.001$ (trend)	
		Thyroid follicular cell adenoma: 1/50 (2%), 1/50 (2%), 2/50 (4%)	NS	

Table 3.1 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%) and/or multiplicity of tumours	Significance	Comments
Mouse, B6C3F$_1$ (M, F) 2 yr Chhabra et al. (1993), NTP (1993) (cont.)		**F_0:F_1 – 0:30, 30:30 ppm (perinatal + adult exposure)**	**Effect of perinatal exposure on adult exposure at 30 ppm (compared with 0:30)**	
		Males		
		Hepatocellular adenoma: 42/50 (84%), 48/50 (96%)	$P = 0.007$	
		Hepatocellular carcinoma: 36/50 (72%), 35/50 (70%)	NS	
		Hepatocellular adenoma or carcinoma (combined): 48/50 (96%), 50/50 (100%)	NS	
		Females		
		Hepatocellular adenoma: 46/48 (96%), 41/47 (87%)	NS	
		Hepatocellular carcinoma: 35/48 (73%), 29/47 (62%)	NS	
		Hepatocellular adenoma or carcinoma (combined): 47/48 (98%), 47/47 (100%)	NS	
Initiation–promotion				
Mouse, CD1 (F) 30 wk Berry *et al.* (1978, 1979)	Initiated with topical application of 200 nmol DMBA in acetone. After 1 wk, mice received 2 µg TPA, or 100 µg Firemaster BP-6, 2×/wk for 30 wk Groups: DMBA only TPA only DMBA + TPA DMBA + Firemaster BP-6 Firemaster BP-6 only 30 mice/group; age 6–8 wks	Skin papilloma: 0/30, 1/30 (3%), 28/30 (93%), 0/30, 0/30		Purity, NR

Table 3.1 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence (%) and/or multiplicity of tumours	Significance	Comments
Mouse, HRS/1 hairless (F) 20 wk Poland *et al.* (1982)	Mice (age 8 weeks) initiated with a single dose of MNNG (5 µmol in 50 µL of acetone) or vehicle applied to the dorsal skin, then 2 mg of Firemaster FF-1 (in 50 µL of acetone), 2×/wk for 5 wk, then 1 mg for 15 wk; PBB-153 or PBB-169, 20 µg in 50 µL of acetone 2×/wk topically for 20 wk. MNNG + FF-1, PBB-153-only, and MNNG-only groups: 26 mice/group MNNG + PBB-153, MNNG + PBB-169, PBB-169-only, and FF-1-only groups: 20 mice/group	Skin papilloma: MNNG only: 0/23 (0) MNNG + FF-1: 9/15 (60%)* (2.0)* MNNG + PBB-153: 0/20 MNNG + PBB-169: 12/20 (60%)* × (1.5)* FF-1 only: 1/16 (6%) (0.13) PBB-153 only: 0/22 PBB-169 only: 0/20	* Significant increase in incidence or multiplicity	Purity, NR Statistical analysis, NR

bw, body weight; DMBA, 7,12-dimethylbenz[*a*]anthracene; F, female; M, male; MNNG, *N*-methyl-*N*′-nitro-*N*-nitroguanine; mo, month; NR, not reported; NS, not significant; PBBs, polybrominated biphenyls; s.c., subcutaneous; TPA, 12-*O*-tetradecanoylphorbol-13-acetate; wk, week

with FF-1, but the incidences were low and the numbers of animals were small.

Groups of 7–19 male Ah-responsive C57BL/10ScSn mice received a single dose of iron-dextran (600 mg/kg) and were then fed a diet containing Firemaster BP-6 [dose not reported] for 2 months, followed by the control diet for 10 months. Hepatocellular nodules were observed in one mouse given Firemaster BP-6 only. Pre-treatment with iron-dextran did not have a significant synergistic effect on the induction of hepatocellular tumours (Smith *et al.*, 1990). [The limitations of the study precluded its use in the evaluation process.]

3.1.2 Transplacental and perinatal exposure

The NTP conducted studies of carcinogenicity in male and female B6C3F_1 mice given diets containing PBBs (Firemaster FF-1) to determine: (i) the effects of PBBs in mice receiving adult (F_1) exposure only from age 8 weeks for 2 years [conventional study of carcinogenicity]; (ii) perinatal (F_0) exposure only (dietary exposure of dams before breeding, and throughout gestation and lactation) followed by control diet for 2 years; and (iii) the combined effect of perinatal and adult exposure (Chhabra *et al.*, 1993; NTP, 1993).

Groups of 60 female mice were exposed to Firemaster FF-1 at a dietary concentration of 0, 3, 10, or 30 ppm for 60 days before breeding. After breeding to previously unexposed males, exposure continued throughout pregnancy and lactation. Weaning occurred on postnatal day 28, and dietary exposure at these same concentrations continued until the pups were approximately age 8 weeks. Subsequently, groups of 60 male and 60 female pups (F_1) were given Firemaster FF-1 at the same dietary concentrations (0, 3, 10, or 30 ppm) and continued on these diets for up to 2 years. After 9 months, 10 mice per group were evaluated.

At 9 months, hepatocellular adenomas occurred in one or more male and female mice from each exposure group, with a significant increase in incidence in the group at 30:30 ppm. A hepatocellular carcinoma occurred in one female mouse in the group at 30:30 ppm.

After 2 years, the effect of adult exposure [conventional study of carcinogenicity] was determined by comparing the groups at 0:0, 0:10 and 0:30 ppm. The incidences of hepatocellular adenoma, carcinoma, and adenoma or carcinoma (combined) were significantly increased ($P \leq 0.01$) in mice at 0:10 and 0:30 ppm. While a single hepatocellular adenoma or carcinoma occurred in tumour-bearing control mice, multiple adenomas, carcinomas, or both adenomas and carcinomas were often present in exposed mice. The effects of perinatal exposure only were determined by comparing the groups at 0:0 and 30:0 ppm. The incidences of hepatocellular adenoma or carcinoma (combined) were significantly increased ($P \leq 0.001$) in mice at 30:0 ppm. The effects of perinatal exposure plus adult exposure were determined by comparing the groups at 0:10, 10:10 and 30:10 ppm, and the groups at 0:30 and 30:30 ppm. When mice were exposed to the lower adult dose, there was a significant increase in the incidence of hepatocellular adenoma and carcinoma in females, and of carcinoma in males (Chhabra *et al.*, 1993; NTP, 1993).

3.1.3 Initiation–promotion

In an initiation–promotion study, groups of 30 female CD1 mice were initiated with a skin application of 200 nmol of 7,12-dimethylbenz[*a*]anthracene (DMBA) in acetone, or acetone only. One week later, the mice received topical applications of 2 µg of 12-*O*-tetradecanoylphorbol-13-acetate (TPA) or 100 µg of Firemaster-BP6 in acetone, twice per week for 30 weeks. Firemaster-BP6 alone did not induce or promote

DMBA-initiated skin tumours (Berry *et al.*, 1978; Berry *et al.*, 1979).

Poland *et al.* (1982) investigated whether Firemaster FF-1 could promote *N*-methyl-*N*′-nitro-*N*-nitrosoguanidine (MNNG)-initiated skin tumours in female HRS/1 hairless mice, as part of a larger study examining the skin tumour-promoting activity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), PCBs, and polychlorinated dibenzofurans (PCDFs). At age 8 weeks, mice were given a single topical dose of MNNG (5 µmol in 50 µL of acetone), or the vehicle only. Mice were then given a topical dose of Firemaster FF-1 (2 mg in 50 µL of acetone) twice per week for 5 weeks, then 1 mg for 15 weeks; or 2,2′,4,4′,5,5′-hexabromobiphenyl (PBB-153) or 3,3′,4,4′,5,5′-hexabromobiphenyl (PBB-169), 20 µg or 50 µL of acetone, respectively, twice per week for 20 weeks. There were 26 mice in the MNNG + FF-1, PBB-153-only, and MNNG-only groups; and 20 mice in the MNNG + PBB-153, MNNG + PBB-169, PBB-169-only, and FF-1 only groups. Tumours were classified as skin papillomas. Firemaster FF-1 and PBB-169 increased the incidence and multiplicity of MNNG-initiated tumours. [Statistical analysis was not reported.]

3.2 Rat

See Table 3.2

3.2.1 Oral administration

The NTP studied the carcinogenic potential of PBBs when administered orally in rats (NTP, 1983). Groups of 50 male and female Fischer F344 rats were given Firemaster FF-1 at a dose of 0 (corn oil), 0.1, 0.3, 1.0, 3.0, or 10.0 mg/kg bw per day by gavage on five consecutive days per week for 6 months. The rats were then observed for an additional 24 months (lifetime observation). The incidence of hepatocellular carcinoma was significantly increased ($P < 0.01$) in males and females at 10 mg/kg bw per day – males, 7 out of 31 (23%) versus 0 out of 33 (controls), and females, 7 out of 20 (35%) versus 0 out of 20 (controls) – and in males at 3 mg/kg bw per day – 7 out of 33 (21%) versus 0 out of 33 (controls). There was also a significant increase ($P < 0.01$) in the incidence of cholangiocarcinoma in females at 10 mg/kg bw per day – 7 out of 20 (35%) versus 0 out of 21 (controls) – and a slight increase ($P = 0.06$) in males at 10 mg/kg bw per day – 2 out of 31 (6%) versus 0 out of 33 (controls). The incidences of hepatic neoplastic nodules in female rats at 3 mg/kg bw per day and higher were significantly increased ($P < 0.01$). There was no clear effect of treatment on the incidence of hepatic neoplastic nodules in males. Liver tumours were observed primarily in rats to which Firemaster FF-1 was given in doses sufficient to induce hepatic toxicity. An increased incidence of myelomonocytic leukaemia was also observed in male rats at 0.3 mg/kg bw per day. [The Working Group noted that the spectrum of neoplastic lesions in the liver was similar to that associated with exposure to PCB-126 and PCB-118 in NTP studies, and hypothesized that the effect observed could be due to PCB activity or the presence of impurities that had dioxin-like activity.]

In a series of studies, Kimbrough *et al.* (1981) dosed non-inbred female Sherman rats with Firemaster FF-1. In one study, groups of 65 female rats were given a single dose of PBBs at 1000 mg/kg bw by gavage and observed for 24 months. The incidence of hepatocellular (trabecular) carcinoma and hepatic neoplastic nodules [adenomas] was significantly increased – 24 out of 58 (41%) versus 0 out of 53 (controls) and 42 out of 58 (72%) versus 0 out of 53 (controls), respectively. In a second study, groups of 30 female rats were given Firemaster FF-1 at a dose of 100 mg/kg bw by gavage twice per week for two 3-week periods separated by approximately 10 weeks (total of 12 doses). After 24 months observation, the incidences of hepatocellular (trabecular) carcinoma and hepatic neoplastic nodules were significantly increased – 17 out of

Table 3.2 Studies of carcinogenicity with PBBs in rats

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence, (%) and/or multiplicity of tumours	Significance	Comments
Rat, F344/N (M, F) 29 mo Gupta *et al.* (1983a), NTP (1983), Silberhorn *et al.* (1990), EFSA (2010)	Firemaster FF-1 at 0 (corn oil), 0.1, 0.3, 1.0, 3.0, or 10.0 mg/kg bw per day, 5 d/wk by gavage for up to 25 wk, and then observed for 23 mo without exposure 51 rats/group; age 7–8 wk	*Males* Neoplastic nodules: 0/33, 0/39, 1/40 (2%), 4/31 (13%)*, 4/33 (12%), 1/31 (3%) Hepatocellular carcinoma: 0/33, 2/39 (5%), 0/40, 1/33 (3%), 7/33 (21%)*, 7/31 (23%)* Cholangiocarcinoma: 0/33, 0/39, 0/40, 0/31, 0/33, 2/31 (6%)* Myelomonocytic leukaemia: 3/33 (9%), 5/39 (13%), 8/40 (20%)*, 4/31 (13%), 2/33 (6%), 2/32 (6%) *Females* Neoplastic nodules: 0/20, 2/21 (10%), 0/21, 2/11 (18%), 5/19 (26%)*, 8/20 (40%)* Hepatocellular carcinoma: 0/20, 0/21, 0/21, 0/11, 3/19 (16%), 7/20 (35%)* Cholangiocarcinoma: 0/20, 0/21, 0/21, 0/11, 0/19, 7/20 (35%)* Myelomonocytic leukaemia: 5/20 (25%), 4/21 (19%), 4/21 (19%), 1/11 (9%), 2/19 (11%), 4/20 (20%)	*$P < 0.05$ *$P < 0.01$ *$P = 0.06$ $P < 0.01$ (trend) *$P < 0.05$ *$P < 0.01$ $P < 0.01$ (trend) *$P < 0.01$ $P < 0.01$ (trend) *$P < 0.01$ $P < 0.01$ (trend) NS	Purity, NR Dose-dependent decrease in survival in males; survival in males at ≥ 0.3 mg/kg bw was significantly less ($P < 0.01$) than controls. Other microscopic lesions included atypical foci and bile duct hyperplasia in liver. [The Working Group noted that the same spectrum of neoplastic lesions in the liver was seen in a long-term NTP study using PCB-126 or PCB-118 (see Section 3, *Monograph* on Polychlorinated Biphenyls in this Volume).]
Rat, Sherman (F) Study I: 23 mo Study II: 24 mo Study III: 22 mo Kimbrough *et al.* (1981), Silberhorn *et al.* (1990), EFSA (2010)	*Study I:* Single dose of 1000 mg/kg bw Firemaster FF-1 or corn oil (control); 65 rats/group; age 2 mo *Study II:* corn oil (control), or 100 mg/kg bw Firemaster FF-1 2×/wk every 3 wk, total of 12 doses; 30 rats/group; age 2 mo *Study III:* Single dose of 200 mg/kg bw Firemaster FF-1 or corn oil (control); 16 rats/group; age 4 mo	*Study I* Liver Trabecular carcinoma: 0/53, 24/58 (41%) Neoplastic nodules: 0/53, 42/58 (72%) *Study II* Liver Trabecular carcinoma: 0/25, 17/28 (61%)	 $P < 0.001$ $P < 0.001$ $P < 0.001$	Other non-neoplastic lesions included altered areas or foci, adenofibrosis and multinucleated hepatocytes in liver. Histological description of the neoplastic nodules was consistent with hepatocellular adenoma.

Table 3.2 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence, (%) and/or multiplicity of tumours	Significance	Comments
Rat, Sherman (F) Study I: 23 mo Study II: 24 mo Study III: 22 mo Kimbrough et al. (1981), Silberhorn et al. (1990), EFSA (2010) cont.)		Neoplastic nodules: 1/25 (4%), 24/28 (82%)	$P < 0.001$	
		Adenocarcinoma: 0/25, 1/28 (4%)	NS	
		Haemangioma: 1/25 (4%), 0/28	NS	
		Total malignant tumours: 0/25, 19/28 (68%)	$P < 0.001$	
		Study III		
		Liver: neoplastic nodules: 0/19, 5/16 (31%)	$P = 0.013$	
Transplacental and perinatal exposure				
Rat, F344/N (M, F) 2 yr Chhabra *et al.* (1993), NTP (1993)	Perinatal exposure (F_0): Firemaster FF-1 at 0, 1, 3, 10 ppm in feed from 60 d before breeding until weaning of the F_1 generation. Adult exposure (F_1): same diet as F_0 during gestation, lactation, and for 4 wk after weaning (age 8 wk); then FF-1 at 0, 3, 10, or 30 ppm, 7 d/wk for 104 wk. 60 rats/group F_0:F_1 – 0:0, 0:10, 0:30 ppm (adult exposure only) F_0:F_1 – 0:0, 10:0 ppm (perinatal exposure only) F_0:F_1 – 0:10, 3:10, 10:10 ppm (perinatal plus adult exposure) F_0:F_1 – 0:30, 10:30 ppm (perinatal plus adult exposure)	**F_0:F_1 – 0:0, 0:10, 0:30 ppm (adult exposure only)** *Males* Hepatocellular adenoma: 1/50 (2%), 10/49 (20%), 38/50 (76%)	$P = 0.002$ (0:10) $P < 0.001$ (0:30) $P < 0.001$ (trend)	Purity, NR Other microscopic changes included hepatocyte hypertrophy, eosinophilic focus, oval cell hyperplasia, hepatocyte cytoplasmic vacuolation and bile duct fibrosis.
		Hepatocellular carcinoma: 0/50, 2/49 (4%), 19/50 (38%)*	*$P < 0.001$ $P < 0.001$ (trend)	
		Hepatocellular adenoma or carcinoma (combined): 1/50 (2%), 12/49 (24%)*, 41/50 (82%)*	*$P < 0.001$ $P < 0.001$ (trend)	
		Females Hepatocellular adenoma: 0/50, 10/50 (20%), 38/50 (76%)	$P = 0.001$ (0:10) $P < 0.001$ (0:30) $P < 0.001$ (trend)	
		Hepatocellular carcinoma: 0/50, 2/50 (4%), 4/50 (8%)	NS	
		Hepatocellular adenoma or carcinoma (combined): 0/50, 12/50 (24%)*, 39/50 (78%)*	*$P < 0.001$ $P < 0.001$ (trend)	

Table 3.2 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence, (%) and/or multiplicity of tumours	Significance	Comments
Rat, F344/N (M, F) 2 yr Chhabra et al. (1993), NTP (1993) (cont.)		**F_0:F_1 – 0:0, 10:0 ppm (perinatal exposure only)**		
		Males		
		Hepatocellular adenoma: 1/50 (2%), 5/50 (10%)	NS	
		Females		
		Hepatocellular adenoma: 0/50, 0/50	NS	
		F_0:F_1 – 0:0, 0:10, 3:10, 10:10 ppm (perinatal plus adult exposure)	**Effect of perinatal exposure on the effect of adult exposure at 10 ppm (compared to 0:10)**	**Effect of total exposure on carcinogenicity (compared to 0:0)**
		Males		
		Hepatocellular adenoma: 1/50 (2%), 10/49 (20%), 13/50 (26%), 16/50 (32%)	NS	[$P < 0.01$ (3:10 and 10:10)]
		Hepatocellular carcinoma: 0/50, 2/49 (4%), 1/50 (2%), 1/50 (2%)	NS	NS
		Hepatocellular adenoma or carcinoma (combined): 1/50 (2%), 12/49 (24%), 14/50 (28%), 16/50 (32%)	NS	$P < 0.001$ (0:10, 3:10, 10:10)
		Females		
		Hepatocellular adenoma: 0/50, 10/50 (20%), 22/50 (44%), 35/50 (70%)	$P < 0.001$ (10:10)	$P < 0.001$ (0:10 and [3:10]) [$P < 0.0001$ (10:10)] [$P < 0.001$ (trend)]
		Hepatocellular carcinoma: 0/50, 2/50 (4%), 1/50 (2%), 8/50 (16%)	$P < 0.01$ (10:10)	[$P < 0.005$ (10:10)]
		Hepatocellular adenoma or carcinoma (combined): 0/50, 12/50 (24%), 22/50 (44%), 39/50 (78%)	$P = 0.03$ (3:10) $P < 0.001$ (10:10) $P < 0.001$ (trend)	$P < 0.001$ (0:10; 3:10; 10:10) [$P < 0.001$ (trend)]

Table 3.2 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence, (%) and/or multiplicity of tumours	Significance	Comments
Rat, F344/N (M, F) 2 yr Chhabra et al. (1993), NTP (1993) (cont.)		**F_0:F_1 – 0:0, 0:30, 10:30 ppm (perinatal plus adult exposure)**	**Effect of perinatal exposure on the effect of adult exposure at 30 ppm (compared with 0:30)**	**Effect of total exposure on carcinogenicity (compared with 0:0)**
		Males		
		Hepatocellular adenoma: 1/50 (2%), 38/50 (76%), 38/50 (76%)	NS	$P < 0.001$ (0:30 and [10:30])
		Hepatocellular carcinoma: 0/50, 19/50 (38%), 23/50 (46%)	NS	$P < 0.001$ (0:30 and [10:30])
		Hepatocellular adenoma or carcinoma (combined): 1/50, 41/50 (82%), 41/50 (82%)	NS	$P < 0.001$ (0:30 and 10:30)
		Females		
		Hepatocellular adenoma: 0/50, 38/50 (76%), 45/50 (90%)	NS	$P < 0.001$ (0:30) [$P < 0.0001$ (10:30)]
		Hepatocellular carcinoma: 0/50, 4/50 (8%), 22/50 (44%)	$P < 0.001$	[$P < 0.0001$ (10:30)]
		Hepatocellular adenoma or carcinoma (combined): 0/50, 39/50 (78%), 47/50 (94%)	$P < 0.05$	$P < 0.001$ (0:30 and 10:30)
Rat, Sherman (M, F) 24 mo Groce & Kimbrough (1984), EFSA (2010)	Pregnant females given corn oil or Firemaster FF-1 (200 mg/kg bw in corn oil) by gavage on d 7 and d 14 of pregnancy. Weaned pups (exposure through placenta and milk) assigned to: Approximately 50 pups/group	*Males*		Purity, NR Other recorded non-neoplastic lesions included foci or altered areas in liver, hepatic cysts, chronic nephrosclerosis, chronic nephritis, interstitial fibrosis and adenomatous hyperplasia in lung and testicular atrophy in males; and foci or altered areas and adenofibrosis in liver, cardiac interstitial fibrosis, chronic nephrosclerosis, interstitial fibrosis in lung, endometrial polyp and ovarian cyst in females.
		Hepatocellular (trabecular) carcinoma: 0/42, 4/41 (10%)	NS	
		Neoplastic nodules: 0/42, 2/41 (5%)	NS	
		Females		
		Hepatocellular trabecular carcinoma: 0/48, 3/51 (6%)	NS	
		Neoplastic nodules: 2/48 (4%), 9/51 (18%)	NS	

Table 3.2 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence, (%) and/or multiplicity of tumours	Significance	Comments
Initiation–promotion				
Rat, Sprague Dawley (F) 180 d Jensen *et al.* (1982), EFSA (2010)	Rats given NDEA at 10 mg/kg bw i.p., 24 h after partial hepatectomy (PH); then 30 d later fed diets containing PB at 500 ppm, or Firemaster BP-6, or PBB-153 at 10 or 100 ppm for 180 d. Controls included rats without PH or NDEA, but fed the same diets. Group 1: PH + NDEA Group 2: None Group 3: PH + NDEA + PB Group 4: PH + NDEA + 10 ppm PBB-153 Group 5: None + 10 ppm PBB-153 Group 6: PH + NDEA + 100 ppm PBB-153 Group 7: None + 100 ppm PBB-153 Group 8: PH + NDEA + 10 ppm BP-6 Group 9: None + 10 ppm BP-6 Group 10: PH + NDEA + 100 ppm BP-6 Group 11: None + 100 ppm BP-6 Three or six/group	Liver neoplastic nodules: 0/6, 0/3, 2/6 (33%), 3/6 (50%), 0/3, 5/6 (83%), 1/3 (33%), 6/6 (100%), 0/3, 6/6 (100%), 2/3 (66%)	$P < 0.05$ (groups 6, 8 and 10 vs group 1)	BP-6 purity, NR; PBB-153 purity, > 99.9% Rats given BP-6 or PBB-153 without PH or NDEA had few altered foci compared with those given PH or NDEA. PBB-153 increased the number of enzyme-altered foci. Limitations of the study included small number of rats and short duration (i.e. less than lifetime exposure).
Rat, Sprague Dawley (F) 480 d Jensen & Sleight (1986), EFSA (2010)	Single dose of NDEA at 10 mg/kg bw, 24 h after partial hepatectomy (PH); 30 d later given 0.1 mg PBB-153 or PBB-169 for 140 d, then basal diet for another 310 d Group 1: Basal diet Group 2: PH + NDEA Group 3: PBB-169 Group 4: PH + NDEA + PBB-169 Group 5: PBB-153 Group 6: PH + NDEA + PBB-153 Group 7: PBB-153 + PBB-169 Group 8: PH + NDEA + PBB-153 + PBB-169 6–12 rats/group	*All groups* Hepatocellular carcinoma: 0/6, 0/12, 0/6, 1/11 (9%), 0/6, 1/10 (10%), 0/6, 1/11 (9%) Hepatocellular nodules: 0, 0.11, 0, 0.25, 0, 1.94, 0, 3.85	$P < 0.05$ (group 6 and group 8)	PBB-153 purity, > 99%; PBB-169 purity, > 99% The combination of PBB-153 and PBB-169 caused a synergistic effect on the development of altered hepatic foci and hepatic nodules per cm^3 liver.

Table 3.2 (continued)

Species, strain (sex) Duration Reference	Dosing regimen, Animals/group at start	For each target organ: incidence, (%) and/or multiplicity of tumours	Significance	Comments
Administration with known carcinogens				
Rat, Sprague Dawley (F) 57 wk Schwartz *et al.* (1980)	Firemaster BP-6 at 50 ppm; after 4 wk, AAF added at 300 ppm for up to 57 wk. Group 1: Basal diet Group 2: BP-6 Group 3: AAF Group 4: BP-6 + AAF 8 or 12 rats/group; age at start, NR	Hepatocellular carcinoma: 0/8, 0/12, 3/8 (37%), 5/12 (42%) Cholangiocarcinoma: 0/8, 0/12, 1/8 (12%), 0/12 Mixed liver carcinoma: 0/8, 0/12, 1/8 (12%), 0/12 Mammary gland, adenocarcinoma: 0/8, 1/12 (8%), 3/8 (37%), 0/12 Mammary gland, cystadenocarcinoma: 0/8, 0/12, 4/8 (50%), 2/12 (17%) Ear duct gland, squamous cell carcinoma: 0/8, 0/12, 5/8 (62%), 1/12 (8%) Lung (metastatic tumours): 0/8, 0/12, 1/8 (12%), 1/12 (8%)		Purity of AAF and BP-6, NR Small numbers of rats and short observation period.

AAF, 2-acetylaminofluorene; bw, body weight; d, day; F, female; h, hour; M, male; MCL, mononuclear cell leukaemia; mo, month; NDEA, *N*-nitrosodiethylamine; NR, not reported; PBBs, polybrominated biphenyls; PH, partial hepatectomy; wk, week; yr, year

28 (61%) versus 0 out of 25 (controls), and 24 out of 28 (82%) versus 1 out of 25 (4%; controls). In a third study, groups of 16 female rats were given Firemaster FF-1 as a single dose at 200 mg/kg bw by gavage. After 22 months, the incidence of hepatic neoplastic nodules was significantly increased – 5 out of 16 (31%) versus 0 out of 19 (controls).

3.2.2 Transplacental and perinatal exposure

The NTP conducted long-term studies of toxicity and carcinogenicity in male and female F344/N rats given diets containing PBBs (Firemaster FF-1) to determine: (i) the effects of PBBs in rats receiving adult (F_1) exposure only from age 8 weeks for 2 years [conventional study of carcinogenicity]; (ii) perinatal (F_0) exposure only (dietary exposure of dams before breeding and throughout gestation and lactation) followed by control diet for 2 years; and (iii) the combined effects of perinatal and adult exposure (Chhabra *et al.*, 1993; NTP, 1993).

Groups of 60 female rats were exposed to Firemaster FF-1 at a dietary concentration of 0, 1, 3, or 10 ppm for 60 days before breeding. After breeding to previously unexposed males, exposure continued throughout pregnancy and lactation. Weaning occurred on postnatal day 28, and dietary exposure at these same concentrations continued until the pups were approximately age 8 weeks. Subsequently, groups of 60 male and 60 female pups (F_1) were given Firemaster FF-1 at the same dietary concentrations (0, 3, 10, or 30 ppm) and continued on these diets for up to 2 years.

After 2 years, the effects of adult exposure [conventional study of carcinogenicity] were determined by comparing the groups at 0:0, 0:10 and 0:30 ppm. The incidences of hepatocellular adenoma, and hepatocellular adenoma or carcinoma (combined) were all significantly increased in males and females of the groups at 0:10 and 0:30 ppm. The majority of male and female rats had multiple hepatocellular adenomas. The incidence of hepatocellular carcinoma was significantly increased in males at 0:30 ppm. Although the combined incidence of adenoma and carcinoma was similar for males and females, there were more carcinomas in males at 0:30 ppm (19 carcinomas) than in females (4 carcinomas). Multiple hepatocellular carcinomas occurred in seven males at 0:30 ppm. In the perinatal-only exposure study, the neoplastic effects of perinatal exposure were determined by comparing the groups at 0:0 and 10:0 ppm; marginal increases in the incidence of hepatocellular adenoma (1 out of 50, 5 out of 50) were noted in males. The effects of perinatal exposure plus adult exposure were determined by comparing the groups at 0:10, 3:10, and 10:10 ppm, and the groups at 0:30 and 10:30 ppm. The incidence of hepatic tumours in females was significantly greater than in those rats exposed only as adults. In females receiving varying concentrations at F_0 and a constant concentration of 10 or 30 ppm at F_1, the incidences of hepatocellular adenoma, and hepatocellular adenoma or carcinoma (combined), increased significantly with the concentration given at F_0. The incidence of mononuclear cell leukaemia in males and females in the groups exposed either as adults only or both perinatally and as adults generally was significantly elevated compared with untreated controls, most notably at the higher exposures (Chhabra *et al.*, 1993; NTP, 1993).

In a study in pregnant Sherman rats given Firemaster FF-1 at an oral dose of 200 mg/kg bw on days 7 and 14 of gestation, the incidences of neoplastic nodules and hepatocellular carcinoma were slightly increased (not significantly) in male and female offspring over the 24 months after treatment (Groce & Kimbrough, 1984).

3.2.3 Initiation–promotion

To determine whether PBB mixtures or individual congeners could serve as tumour promoters in a two-stage test for hepatocarcinogenesis, groups of three or six female Sprague-Dawley rats were given *N*-nitrosodiethylamine (NDEA) as a single intraperitoneal dose at 10 mg/kg bw, 24 hours after a 70% partial hepatectomy. After 30 days, the rats were fed a basal diet or a basal diet containing Firemaster BP-6 or PBB-153 [called "HBB" in the article] at a concentration of 10 or 100 ppm for 180 days. Diets were prepared by adding phenobarbital, Firemaster BP-6 or PBB-153 in corn oil to a basal diet. Controls included non-hepatectomized rats or rats not given NDEA. At 100 ppm, Firemaster BP-6 alone caused an increase (two out of three rats; not statistically significant) in the incidence of neoplastic nodules. In combination with partial hepatectomy and NDEA, diets that contained Firemaster BP-6 or PBB-153 were associated with significant ($P < 0.05$) promotion of neoplastic nodules: five out of six rats receiving PBB-153 at 100 ppm, and six out of six rats receiving Firemaster BP-6 at 10 ppm, and six out of six rats receiving Firemaster BP-6 at 100 ppm. Both Firemaster BP-6 and PBB-153 increased the number of enzyme-altered foci (Jensen *et al.*, 1982). [The limited numbers of animals and less-than-lifetime observation period in this study limited the conclusions that could be reached on carcinogenic potential.]

To determine the effect of individual PBB congeners on the enhancement of gamma-glutamyl transpeptidase (GGT)-positive altered hepatic foci and the development of hepatic nodules and carcinomas, groups of 6 or 12 female Sprague-Dawley rats were given a single dose of NDEA, 24 hours after a 70% partial hepatectomy. After 30 days, the rats were fed a basal diet, or the basal diet containing PBB-153 at 10 ppm, PBB-169 at 0.1 ppm, or PBB-153 (10 ppm) + PBB-169 (0.1 ppm) for 140 days, followed by basal diet for an additional 310 days. Rats were killed 170, 240 or 480 days after partial hepatectomy. Dietary exposure to the PBB congeners alone or in combination did not increase the incidence of hepatocellular carcinoma, hepatic nodules, or altered hepatic foci. However, PBB-153 alone or in combination with PBB-169 increased the development of altered hepatic foci and nodules in partially hepatectomized rats given NDEA. Rats that had not been hepatectomized and given NDEA, and that were fed the basal diet or the basal diet containing PBB congeners, had no or relatively few altered hepatic foci when compared with rats that received the same diets but had been partially hepatectomized and given NDEA (Jensen & Sleight, 1986).

3.2.4 Administration with known carcinogens

Groups of 8 or 12 female Sprague-Dawley rats were given diets containing 2-acetylaminofluorene (AAF) at a concentration of 300 ppm, Firemaster BP-6 at 50 ppm, or BP-6 + AAF, for approximately 1 year. Firemaster BP-6 significantly reduced the incidence of AAF-induced tumours at non-hepatic locations (mammary gland and ear duct), but did not affect the incidence of hepatic tumours. Ingestion of Firemaster BP-6 only did not increase the incidence of tumours when compared with untreated controls (Schwartz *et al.*, 1980). [Conclusions regarding the carcinogenic potential of Firemaster BP-6 were limited by the low number of animals per group and the less-than-lifetime observation period.]

3.3 Hamster

See Table 3.3

Table 3.3 Studies of carcinogenicity with PBBs in hamsters

Species, strain (sex) Duration Reference	Dosing regimen Animals/group at start	For each target organ: incidence (%) and/or multiplicity of tumours	Significance	Comments
Hamster, Syrian Golden (M) 273 d Wasito & Sleight (1989)	Initiated with NDEA as a single s.c. dose at 0 or 80 mg/kg bw, then fed diets containing BP-6 at 0 or 100 mg/kg for 140 days, after which basal diet was given until the end of the study W(d 273). Group 1: control Group 2: NDEA Group 3: NDEA + BP-6 Group 4: BP-6 30 hamsters/group	*Nasal cavity* Papilloma: 0/30, 1/30 (3%), 9/30 (30%), 0/30 Adenoma: 0/30, 2/30 (7%), 1/30 (3%), 0/30 Adenocarcinoma: 0/30, 7/30 (23%), 2/30 (7%), 0/30 Squamous cell carcinoma: 0/30, 0/30, 2/30 (7%), 1/30 (3%) Total nasal tumours: 0/30, 11/30 (37%), 15/30 (50%), 1/30 (3%) *Tracheal papilloma (multiplicity)*: 0, 26, 27, 0 (4.33, 1.6)	$P < 0.05$	Purity, NR Nasal tumours occurred at approximately the same incidence in hamsters given NDEA as in those given NDEA + BP-6.

bw, body weight; d, day; M, male; NDEA, *N*-nitrosodiethylamine; PBBs, polybrominated biphenyls; s.c., subcutaneous

Initiation–promotion

In an initiation–promotion study of carcinogenesis of the respiratory tract, groups of 30 male Syrian Golden hamsters were initiated with a single subcutaneous dose of NDEA at 0 or 80 mg/kg bw and were then (7 days later) fed a diet containing Firemaster BP-6 at 0 or 100 mg/kg for 140 days, followed by basal diet from day 140 until the end of the experiment at 273 days. Firemaster BP-6 slightly promoted the development of benign tracheal papilloma in hamsters. The multiplicity of tracheal papillomas, but not the incidence, was significantly increased in hamsters given NDEA + BP-6 compared with those given NDEA only. Tracheal papilloma was not seen in untreated hamsters or in hamsters fed a diet containing Firemaster BP-6 only. Nasal tumours (total) occurred at approximately the same incidence in hamsters given NDEA only or NDEA + BP-6. Adenomas occurred in the nasal cavity of two hamsters given NDEA only and in one hamster given NDEA + BP-6. Adenocarcinoma occurred in the nasal cavity of seven hamsters given NDEA only, and in two hamsters given NDEA + BP-6. Squamous cell carcinoma of the nasal cavity occurred in two hamsters given NDEA + PBBs and in one hamster given Firemaster BP-6 only (Wasito & Sleight, 1989).

4. Mechanistic and Other Relevant Data

4.1 Absorption, distribution, metabolism, and excretion

PBBs share several chemical and physical characteristics with their chlorinated analogues, including effective absorption and distribution, the higher brominated biphenyls distributing/re-distributing to fatty tissues. PBBs readily cross the placenta in several species (DiCarlo *et al.*, 1978; Ecobichon *et al.*, 1983). PBBs have estimated long half-lives in animal tissues, serum, and fat, ranging from 22 days to more than 69 weeks (Miceli & Marks, 1981; Ecobichon *et al.*, 1983), extending to years in humans (ATSDR, 2004). PBBs have the potential to greatly alter their own distribution, metabolism, and

excretion through at least two mechanisms: PBB congeners are potent and efficacious inducers of xenobiotic-metabolizing enzymes, for which they may also become substrates and inhibitors (see Section 4.3).

4.2 Genetic and related effects

Limited data on PBBs and genotoxicity were available to the Working Group (reviewed in Silberhorn *et al.*, 1990). Firemaster BP-6, Firemaster FF-1, and the individual congeners PBB-77, -153, -169, and -153 + -180 have been tested in assays for genotoxicity. All assays with commercial PBB mixtures or individual congeners gave negative results for genotoxicity in mammals, except one in which a more-than-additive mitotic arrest response was seen in the bone marrow of pregnant rats treated with Firemaster [not further specified] and colchine (Ficsor & Wertz, 1976).

Only three PBB congeners have been tested in bacterial assays for mutation (Silberhorn *et al.*, 1990), i.e. the 2-, 3-, and 4-bromobiphenyls (PBB-1, PBB-2, PBB-3). All results were negative with and without metabolic activation (Haworth *et al.*, 1983), except PBB-3 that gave positive results with activation from S9 (Kohli *et al.*, 1978).

4.3 Biochemical and cellular effects

4.3.1 Induction of xenobiotic metabolism and oxidative stress

PBBs, like their chlorinated analogues, are ligands for several cellular and nuclear receptors. The earliest description of PCBs as ligands was for the aryl hydrocarbon receptor (AhR) (Bandiera *et al.*, 1982). This binding preceeded the efficacious induction of a broad spectrum of xenobiotic-metabolizing enzymes, most noticeably certain cytochrome P450-dependent monoxygenases (CYPs). PCBs and PBBs increased the activity of CYP2Bs and microsomal epoxide hydrolase (Parkinson *et al.*, 1983), glutathione transferases (Schramm *et al.*, 1985), and UDP-glucuronosyltransferase (Ahotupa & Aitio, 1978).

Of the individual PBB congeners, like PCB congeners with the same substitution pattern, the best ligands for AhR are those isomers and congeners in which halogens are present in the *meta* and *para* positions of biphenyl, but without *ortho* halogens (Robertson *et al.*, 1982, 1983, 1984b). These PBBs are referred to as "coplanar" or "dioxin-like" congeners, typical examples of which are PBB-77, PBB-126, PBB-169. Other halogenated biphenyls, characterized by halogen substitution in the *ortho* and *para* positions of biphenyl (e.g. 2,2′,4,4′,5,5′-hexabromobiphenyl, PBB-153), activate the constitutive androstane receptor (CAR). PBBs in this group induce CYP2B1/2 and as such resemble phenobarbital in their mode of induction of cytochrome P450 (Robertson *et al.*, 1982, 1984b; Parkinson *et al.*, 1983). PBBs with one *ortho* bromine may be mixed-type inducers of CYPs, inducing CYP1A and CYP2B subfamily members (Robertson *et al.*, 1981, 1982).

Although there is great similarity in the modes of induction of cytochrome P450 by PCBs and by PBBs, in terms of potency and efficacy there are a few examples of qualitative differences, and many quantitative differences. 3,4,4′-Tribromobiphenyl (PBB-37) is strictly an inducer of CYP1A in rat liver, while its chloro analogue also induces CYP2B isoforms (Robertson *et al.*, 1982; Parkinson *et al.*, 1983). Andres and co-workers compared the modes of induction and potency of a series of 3,3′,4,4′-tetrahalobiphenyl congeners in which each chlorine atom was sequentially replaced with bromine; the brominated analogues were more potent and more efficacious inducers of cytochrome P450 and much more toxic (Andres *et al.*, 1983).

In a 16-day time-course, Firemaster BP-6 was more efficacious than Aroclor 1254 (both at a dose of 500 mg/kg bw) in repressing hepatic

selenium-dependent glutathione peroxidase activity (Schramm *et al.*, 1985). Given that the average relative molecular mass of Firemaster BP-6 is almost twice that of Aroclor 1254, Firemaster BP-6 had a greater effect at about half the molar dose.

4.3.2 Substrates and inhibitors of xenobiotic metabolism

The metabolic activation of lower halogenated biphenyls to electrophiles and their reaction with cellular substituents, such as proteins and DNA, the production of oxygen-centred radicals, and the biological and toxicological consequences of these reactions, have been explored extensively with individual PCBs and commercial PCB mixtures. However, the same level of attention has not been paid to the PBBs, although it may be assumed that many of the same principles/pathways apply (see *Monograph* on Polychlorinated Biphenyls, Section 4.2 and Section 4.6, in this Volume).

Mills and coworkers investigated the metabolism of PBBs by hepatic microsomes from male rats treated with 3-methylcholanthrene [CYP1A inducer]. The rate of metabolism in decreasing order was PBB-15 (fastest), followed by PBB-37, PBB-77, PBB-56, PBB-70, and PBB-49. The rate of metabolism by hepatic microsomes from male rats treated with phenobarbital [an inducer of CYP2B] was PBB-4 (fastest) followed by PBB-49, PBB-52, PBB-56, PBB-70, and PBB-101. Thus CYP1A preferentially metabolized congeners with adjacent non-halogenated *ortho* and *meta* carbon atoms, while CYP2B preferentially metabolized congeners with adjacent non-halogenated *meta* and *para* carbons on at least one ring (Mills *et al.*, 1985). Also, PBB-169 effectively inhibited the metabolism of PBB-77 at similar concentrations (Mills *et al.*, 1985).

4.3.3 Cell–cell communication and metabolic cooperation

There were three reports that Firemaster BP-6 and individual PBB congeners can inhibit cell–cell communication or metabolic cooperation (Trosko *et al.*, 1981; Tsushimoto *et al.*, 1982; Kavanagh *et al.*, 1987). Firemaster BP-6, and PBB-118, PBB-153, PBB-180, and PBB-194 were reported to exert a dose-related inhibition of metabolic cooperation at concentrations that were relatively non-toxic to cells (Trosko *et al.*, 1981; Tsushimoto *et al.*, 1982). Firemaster BP-6 and PBB-153 displayed dose-dependent inhibition of cell–cell communication (Kavanagh *et al.*, 1987). In contrast, PBB-77, PBB-126, and PBB-169, all three with a dioxin-like activity, were inactive as inhibitors of metabolic cooperation or cell–cell communication at non-cytotoxic concentrations (Tsushimoto *et al.*, 1982; Kavanagh *et al.*, 1987).

4.3.4 Initiation–promotion

Six publications described studies that assessed Firemaster BP-6 and individual PBB congeners (PBB-77, PBB-153, PBB-169, and the combination of PBB-153 and PBB-169) as initiators and promoters of preneoplastic lesions in two-stage models of hepatocarcinogenesis in female Sprague-Dawley rats. All studies found that Firemaster BP-6 and individual PBB congeners were weak initiators, producing a small number of preneoplastic foci when administered alone. In contrast, Firemaster BP-6 and PBB -77, PBB-153, and the combination of PBB-153 and PBB-169 were generally efficacious promoters following an initiation regimen of partial hepatectomy plus NDEA, while PBB-169 alone did not show promoting activity (Jensen *et al.*, 1982, 1983, 1984; Jensen & Sleight, 1986; Rezabek *et al.*, 1987; Dixon *et al.*, 1988).

4.3.5 Other biochemical and cellular effects

In contrast to the PCBs, the PBBs had not yet been investigated for estrogenicity and anti-estrogenicity via estrogen-receptor binding (Gierthy *et al.*, 1997), effects on calcium channels via activation of the ryanodine receptor (Wong *et al.*, 1997), ability to cause insulin release from cells in culture (Fischer *et al.*, 1996), their potency in lowering cellular dopamine levels (Chu *et al.*, 1995), and their ability to activate neutrophils to produce superoxide (Fischer *et al.*, 1998).

4.4 Organ toxicity

In studies of acute toxicity, especially with dioxin-like PBBs, pathological and biochemical changes in the liver are evident in a matter of days. In rats, for example, a single intraperitoneal dose of PBB-77 at 150 µmol/kg resulted in a statistically significant increase in liver weight in 24 hours, and a significant decrease in thymus weight in 4 days (Robertson *et al.*, 1991). Small distinct lipid droplets in hepatocytes were seen histopathologically as early as day 2, while a loss of cortical lymphocytes of the thymus was seen at day 4.

In a 30-day study, mice and rats were given either Firemaster FF-1 or an equal molar equivalent of PBB-153 (Gupta *et al.*, 1981). After 15 days, livers were enlarged due to hepatocyte swelling, fatty infiltration, and proliferation of the endoplasmic reticulum, in animals treated with 3 or 30 mg/kg, and these hepatocellular alterations persisted to 120 days at the highest dose. Firemaster FF-1 was more toxic than PBB-153 (Gupta *et al.*, 1981).

In a long-term study, rats and mice were given Firemaster FF-1 or BP-6 for 6 months (Gupta *et al.*, 1983b; NTP, 1983). Treated rodents showed decreased body-weight gain (despite no change in feed consumption), increased liver weight, and decreased thymus weight. Microscopic changes in the liver included hepatocellular swelling, disorganization, single-cell necrosis, fatty infiltration, and bile-duct proliferation. Levels of hepatic porphyrin were markedly increased, while serum levels of T4 (thyroxine) and T3 (triiodothyronine) were decreased (Gupta *et al.*, 1983b; NTP, 1983). After the 6 months of dosing, the animals were observed for an additional 23 or 24 months. Treated rats showed significantly higher incidence of atypical hepatocellular foci, neoplastic nodules, hepatocellular carcinoma, and cholangiocarcinoma (see Section 3).

Mild microscopic changes in the thyroid gland were also observed in the NTP study (NTP, 1983). Kasza and colleagues carried out a more detailed examination of the effects of PBBs in the rat thyroid. On microscopic (light and electron) examination after short-term dietary exposure, they found ultrastructural lesions consistent with diminished synthesis and secretion of thyroxine (Kasza *et al.*, 1978).

In a subsequent NTP study (NTP, 1993), the effects of exposure to Firemaster FF-1 were investigated in rats and mice exposed as adults, exposed only perinatally (dietary exposure of dams before breeding and throughout gestation and lactation), or exposed both perinatally and as adults. The adult-only exposures demonstrated that the major organ affected by toxicity associated with PBBs was the liver. At 9 months, rats had decreased body weight, hepatomegaly, non-neoplastic histopathological changes, mild anaemia, increased serum cholesterol, and decreased serum triglycerides (males only) (NTP, 1993).

Immunocompetence after exposure to PBBs has been investigated in rodents and birds (Vos & Van Genderen, 1973; Luster *et al.*, 1978), in cattle (Jackson & Halbert, 1974; Kateley & Bazzell, 1978), in swine (Howard *et al.*, 1980), and in humans (Bekesi *et al.*, 1979b, 1987). Exposure of rats to dioxin-like PBBs resulted in rapid loss of cortical thymocytes (Robertson *et al.*, 1991), as described above. In rats exposed to PBBs, the ability to mount an antibody response to an

antigen was impaired. Both cell-mediated and humoral immunity were affected in rats and mice (Vos & Van Genderen, 1973; Luster *et al.*, 1978). Farm cattle given fodder contaminated with PBBs developed a range of symptoms, including atrophic thymus, abnormal lymph nodes, and prolonged infections (Jackson & Halbert, 1974). In contrast, Kateley & Bazzell (1978) did not find evidence of immune system impairment in cattle exposed environmentally, or exposed accidentally to PBBs at much lower levels. In sows fed Firemaster BP-6 at a dose of 100 or 200 ppm during the second half of lactation, the lymphocyte mitogenic response was significantly reduced in piglets tested at age 4 weeks (Howard *et al.*, 1980). In Michigan-farm residents who had consumed food contaminated with PBBs, immune-function abnormalities in vitro were evident in 20–25% (Bekesi *et al.*, 1987) and 35–40% (Bekesi *et al.*, 1979b) of the residents examined.

Endocrine disruption

Several investigations have reported PBB-related effects in individuals exposed during the Michigan poisoning episode of the 1970s (see Section 1.4.4). Dietary exposure to PBBs was associated with an elevated occurrence of self-reported abnormal Pap tests in women; occurrence was lower in exposed women who had breastfed for more than 12 months (Jamieson *et al.*, 2011). Maternal exposure to PBBs was also associated with increased likelihood of a male birth (Terrell *et al.*, 2009) and with increased infant birth weights (Sweeney & Symanski, 2007).

Perinatal exposure of rats to PBBs diminished the effect of exogenously administered estradiol on uterine weight and uterine RNA content. PBBs increased the hepatic microsomal metabolism of estradiol, estrone, and ethynylestradiol in vitro (McCormack *et al.*, 1979; Bonhaus *et al.*, 1981).

4.5 Mechanistic considerations

PBBs are highly lipophilic, and bioconcentrate and bioaccumulate. In mammals, they are transferred through the placenta and in breast milk (McCormack *et al.*, 1981; Kimbrough, 1985; Jacobson *et al.*, 1989). PBBs are efficacious inducers of hepatic metabolism, accelerating the turnover (half-lives) of endogenous and exogenous compounds. An imbalance in metabolizing enzymes may lead to increased oxidative stress through at least three mechanisms, which have been observed with PCBs. Firstly, it has been demonstrated that the persistent induction of CYPs, in the absence of substrate, may lead to the production of reactive oxygen species (Schlezinger *et al.*, 1999, 2000). Secondly, an increase in or induction of certain metabolizing enzymes, especially CYPs and epoxide hydrolase, may steer the metabolism of endogenous and exogenous compounds towards more redox-reactive intermediates, estradiol, PCBs, etc., and increase redox cycling (CYP reductase, DT-diaphorase) (Twaroski *et al.*, 2001). Lastly, a reduction in antioxidants and antioxidant enzymes, such as selenium and selenium-dependent glutathione peroxidase, may cause an increase in oxidative stress through the lowering of antioxidant defenses (Schramm *et al.*, 1985; Twaroski *et al.*, 2001; Lai *et al.*, 2010, 2011).

PBBs display a variety of adverse effects, including immune-system suppression (Bekesi *et al.*, 1979b, 1987), disruption of normal hormone function (McCormack *et al.*, 1979; Bonhaus *et al.*, 1981) and disruption of cell–cell communication. The liver and the immune system are early targets of PBB toxicity. PBBs are weak initiators of rodent two-stage hepatocarcinogenesis and are efficacious promoters in this model system. PBBs produce lesions in the liver and in a variety of other tissues and organs. Other acute adverse biochemical and toxic effects of PBBs are no doubt mediated by the interactions of various PBBs with other sites and cellular receptors.

Much less research has been conducted on PBBs than on PCBs. Commercial PBB mixtures are associated with equivalent or greater toxicity than their chlorinated analogues (Matthews *et al.*, 1978; Andres *et al.*, 1983). It is likely that the congeneric PBBs exhibit their toxicity and disease potential via many of the same pathways as their chlorinated counterparts.

5. Summary of Data Reported

5.1 Exposure data

Polybrominated biphenyls (PBBs) are a class of aromatic compounds consisting of 209 congeners, in which 1–10 bromine atoms are attached to a biphenyl nucleus. The current nomenclature arranges the 209 congeners by increasing numbers of bromine atoms from 1 to 209. PBBs are not known to occur naturally. PBBs are chemically comparable to the polychlorinated biphenyls (PCBs), although the bromine atom is larger than the chlorine atom, and the carbon–bromine bond is weaker than that between carbon and chlorine. PBBs are characterized by low volatility, which decreases with increasing number of substituted bromine, and low solubility in water; they are chemically stable and persistent in the human body, although to a lesser extent than PCBs. Highly brominated PBB congeners tend to debrominate to less brominated congeners.

The analytical methods for detection of PBBs are similar to those for PCBs, but highly sensitive methods are required at low concentrations.

PBBs were produced primarily as flame retardants, as hexa-, octa- and decabromobiphenyls, with bromine content of up to 85% by weight. PBBs were also added to plastics as flakes (up to 10%), and not chemically incorporated into the polymers. Other uses were in coatings and lacquers, and in polyurethane foam.

PBBs have been detected primarily near the sites of production and use; however, detection in biota of remote areas shows that PBBs should be considered as global environmental pollutants. One major episode of human food contamination occurred in Michigan, USA, in which animal feed supplement was contaminated with a commercial PBB mixture. The highest exposure occurred from consuming dairy products from those farms that had received the contaminated feed. As a result of this accident in 1973–1974, PBB production soon ceased in the USA; by 2000, all known production had ceased globally. Workers involved in production were exposed to PBBs through inhalation or dermal contact. Some workers continue to be exposed today through e-waste dismantling and recycling.

Mixed polybromochlorobiphenyls (PXBs) are a class of aromatic compounds with a mixed content of chlorine and bromine atoms attached to the biphenyl nucleus. PXBs have been observed as minor contaminants in some commercial PCB or PBB mixtures, and maybe formed upon disposal of these products at high temperature. PXBs have been detected in environmental and biological samples.

5.2 Human carcinogenicity data

Human data on the carcinogenicity of PBBs were available primarily from follow-up of residents exposed to contaminated food following an industrial accident in Michigan, USA. In a nested case–control analysis, positive findings were observed for lymphoma and cancers of the digestive system combined (including liver, stomach, oesophagus, and pancreas). The cohort was unique, but small, and the risk estimates are imprecise.

5.3 Animal carcinogenicity data

PBBs have been evaluated using a variety of study designs in rats, mice and hamsters, ranging in duration from several months up to 2 years.

These include complete studies of carcinogenicity, studies of carcinogenicity involving transplacental and perinatal exposure, studies assessing promoting activity, using tumours or preneoplastic lesions as an end-point, a study of co-carcinogenicity, and a study of modification of iron metabolism.

Firemaster FF-1, a commercial mixture of PBBs, was tested for carcinogenicity in two studies by gavage or in the diet in male and female mice: FF-1 caused a significant increase in the incidence of hepatocellular carcinoma. In another study of carcinogenicity incorporating adult-only, perinatal-only, and adult-plus-perinatal exposures, Firemaster FF-1 caused significantly increased incidences of hepatocellular adenoma and carcinoma, and hepatocellular adenoma and carcinoma combined. There was also a positive trend for thyroid follicular-cell adenoma in male mice.

Firemaster FF-1 was tested for carcinogenicity in two oral gavage studies in male and female rats: FF-1 caused significantly increased incidences of hepatic neoplastic nodules, hepatocellular carcinoma and (rare) cholangiocarcinoma in male and female rats, and myelomonocytic leukaemia in male rats. In another study of carcinogenicity incorporating adult-only, perinatal-only, and adult-plus-perinatal exposures, Firemaster FF-1 exposure caused significantly increased incidences of hepatocellular adenoma and carcinoma, hepatocellular adenoma and carcinoma combined, and mononuclear cell leukaemia in male and female rats.

In Syrian golden hamsters, Firemaster BP-6 in the diet promoted the development of *N*-nitrosodiethylamine-initiated benign nasal papillomas in one study. Firemaster BP-6 did not promote 7,12-dimethylbenz[*a*]anthracene-initiated skin tumours in one study in mice.

PBB-153 had promoting activity in two studies of *N*-nitrosodiethylamine-induced rat liver carcinogenesis with hepatic nodules and altered hepatic foci as the end-points, but did not have promoting activity in one study of *N*-methyl-*N*′-nitro-*N*-nitrosoguanidine-induced mouse skin carcinogenesis.

PBB-169 had promoting activity in one study of *N*-methyl-*N*′-nitro-*N*-nitrosoguanidine-induced mouse skin carcinogenesis, but did not have promoting activity in one study of *N*-nitrosodiethylamine-induced rat liver carcinogenesis, although it enhanced the liver tumour promoting activity when administered together with PBB-153.

5.4 Mechanistic and other relevant data

PBBs are highly lipophilic compounds that biocncentrate and bioaccumulate in fatty tissues. PBBs are efficacious inducers of hepatic metabolism, accelerating the turnover (reducing the half-lives) of both endogenous and exogenous compounds. PBBs display a variety of adverse effects including suppression of the immune system and disruption of normal hormone function. PBBs are weak initiators of two-stage hepatocarcinogenesis in rodents, but they are efficacious promoters in these model systems. When administered to rodents by themselves and for longer periods of time, PBBs are carcinogens that produce tumours in the liver and in a variety of other tissues and organs.

While there is an extensive body of literature to assess the carcinogenicity of PCBs (see the *Monograph* on Polychlorinated Biphenyls in this Volume), their brominated analogues have received much less attention and study. Firemaster, a commercial mixture of PBBs, causes aryl hydrocarbon receptor-related toxicity equivalent to or greater than that of their chlorinated analogues. PBBs likely will exhibit their toxicity and disease potential via many of the same pathways as their chlorinated counterparts.

6. Evaluation

6.1 Cancer in humans

There is *inadequate evidence* in humans for the carcinogenicity of polybrominated biphenyls.

6.2 Cancer in experimental animals

There is *sufficient evidence* in experimental animals for the carcinogenicity of Firemaster FF-1.

There is *limited evidence* in experimental animals for the carcinogenicity of polybrominated biphenyl-153.

There is *inadequate evidence* in experimental animals for the carcinogenicity of polybrominated biphenyl-169 and Firemaster BP-6.

6.3 Overall evaluation

Polybrominated biphenyls are *probably carcinogenic to humans (Group 2A)* on the basis of mechanistic similarities to polychlorinated biphenyls.

Rationale for the mechanistic upgrade of polybrominated biphenyls to Group 2A:

- Polybrominated biphenyls share several chemical and physical characteristics with their chlorinated analogues.
- Polybrominated biphenyls are effectively absorbed and distributed, cross the placenta and are detected in milk.
- Polybrominated biphenyls have estimated long half-lives in animal tissues, serum and fat.
- Polybrominated biphenyl congeners are potent and efficacious inducers of xenobiotic-metabolizing enzymes.
- Individual polybrominated biphenyl congeners inhibit cell-to-cell communication or metabolic cooperation.
- Individual congeners PBB-77, PBB-153, PBB-169 are weak initiators and efficacious promoters of two-stage hepatocarcinogenesis.
- Individual polybrominated biphenyls, as for their chlorinated analogues, are ligands for several cellular and nuclear receptors.
- In studies of acute toxicity, pathological and biochemical changes in the liver and thymus are evident in a matter of days.
- In long-term studies, polybrominated biphenyls induce microscopic changes in rodent liver, described as hepatocellular swelling, disorganization, single cell necrosis, fatty infiltration and bile duct proliferation, and mild microscopic changes in thyroid glands.
- Reduced immunocompetence after polybrominated biphenyl exposure was found in rodents, birds, cattle, swine and humans.
- Perinatal exposure of rats to polybrominated biphenyls diminished the effect of exogenously administered estradiol on uterine weight and uterine RNA content. Polybrominated biphenyls increased the hepatic microsomal metabolism of estradiol, estrone and ethynylestradiol in vitro.
- Polybrominated biphenyl exposure in women was also associated with increased odds of a male birth.

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LIST OF ABBREVIATIONS

ABS	acrylonitrile-butadiene-styrene
AMAP	Arctic Monitoring and Assessment Programme
ANZECC	Australian and New Zealand Environment and Conservation Council
ARNT	aryl hydrocarbon receptor nuclear translocator
BHC	benzene hexachloride
BMI	body mass index
BOS	basic oxygen steelmaking
BrdU	bromodeoxyuridine
bw	body weight
CAR	constitutive androstane receptor
CAS	Chemical Abstracts Service
DEN	diethylnitrosamine
DHEA	dehydroepiandrosterone
DIPN	*N*-nitrosodiisopropanolamine
DLBCL	diffuse large B-cell lymphoma
DL-PCB	dioxin-like PCBs
DMBA	7,12-dimethylbenz[*a*]anthracene
ECNI	electron-capture negative ionization
EBV	Epstein–Barr virus
EBV-EA	Epstein–Barr virus early antigen
EFSA	European Food Safety Authority
EHEN	*N*-ethyl-*N*-hydroxyethylnitrosamine
EPA	Environmental Protection Agency
EROD	ethoxyresorufin *O*-deethylase
e-waste	electronic waste
GC-MS	gas chromatography–mass spectrometry
GGT	gamma-glutamyl transferase
GJIC	gap-junctional intercellular communication
GPC	gel permeation chromatography
GSH	glutathione
GST	glutathione *S*-transferase
OH-PCBs	hydroxylated PCBs
HR	hazard ratio
HRGC	high-resolution gas chromatography
HRMS	high-resolution mass spectrometry

IRR	incidence rate ratio
JEM	job-exposure matrix
LLE	liquid-liquid extraction
MDAB	3'-methyl-4-dimethylaminoazobenzene
MNNG	*N*-methyl-*N*'-nitro-*N*-nitrosoguanidine
NDEA	*N*-nitrosodiethylamine
NDL-PCBs	non-dioxin-like PCBs
NDMA	*N*-nitrosodimethylamine
NHANES	National Health and Nutrition Examination Survey
NHL	non-Hodgkin lymphoma
NIOSH	National Institute for Occupational Safety and Health
NK	natural killer
NNK	4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone
NTP	National Toxicology Program
PAH	polycyclic aromatic hydrocarbons
PBB	polybrominated biphenyl
PBDE	polybrominated diphenyl ether
PBMC	peripheral blood mononuclear cells
PBN	polybrominated naphthalene
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzodioxin
PCDF	polychlorinated dibenzofuran
PeCDF	2,3,4,7,8-pentachlorodibenzofuran
PEL	permissible exposure limit
PLE	pressurized liquid extraction
PXB	polybromochlorobiphenyl
PXR	pregnane-X receptor
RR	rate ratio
SIR	standardized incidence ratio
siRNA	small interfering RNA
SMR	standardized mortality ratio
SPE	solid-phase extraction
SHBG	steroid hormone-binding globulin
T3	triiodothyronine
T4	thyroxine
TCDD	2,3,7,8-tetrachlorodibenzo-*para*-dioxin
TWA	time-weighted average
TEF	toxic equivalence factor
TEQ	toxic equivalency
TNF	tumour necrosis factor
TSH	thyroid-stimulating hormone
TTR	transthyretin; thyroid hormone transport protein; thyroxine-binding protein
UDPGT	uridine diphosphoglucuronyl transferase

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IARC MONOGRAPHS

This volume of the *IARC Monographs* provides evaluations of the carcinogenicity of polychlorinated biphenyls and polybrominated biphenyls.

Polychlorinated biphenyls are a class of aromatic compounds comprising 209 congeners, each containing 1 to 10 chlorine atoms attached to a biphenyl nucleus. Technical products, which were manufactured to obtain a certain degree of chlorination, are mixtures of numerous congeners. These products were widely used as dielectric fluid in capacitors and transformers, and to a lesser extent in building materials. Although their production and use has been banned in most countries, these compounds are ubiquitous environmental pollutants, including in polar regions and the deep ocean, because they are persistent and bioaccumulate. Worldwide monitoring programmes have shown that polychlorinated biphenyls are present in most samples of human milk.

An *IARC Monographs* Working Group reviewed epidemiological evidence, animal bioassays, and mechanistic and other relevant data to reach conclusions as to the carcinogenic hazard to humans of polychlorinated biphenyls, of the subclass of dioxin-like polychlorinated biphenyls, and of polybrominated biphenyls.

Attachment E

Associations between Prenatal Exposure to Polychlorinated Biphenyls and Neonatal Thyroid-Stimulating Hormone Levels

Associations between Prenatal Exposure to Polychlorinated Biphenyls and Neonatal Thyroid-Stimulating Hormone Levels in a Mexican-American Population, Salinas Valley, California

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Background: Studies have reported that prenatal exposure to polychlorinated biphenyls (PCBs) may alter neurodevelopment in both humans and animals. Furthermore, prenatal exposure to some PCB congeners and commercial mixtures has been shown to decrease free and total thyroxine (T_4) blood levels in animals. Because thyroid hormones (TH) are essential for normal neurologic development, it has been suggested that the deleterious neurodevelopmental effect of PCBs may occur through TH disruption. PCBs may in turn affect TH levels by inducing the microsomal enzyme uridinediphosphate glucuronosyltransferase (UDP-GT), which is involved in TH elimination.

Objectives: Our goals were to group PCB congeners based on their potential to induce microsomal enzymes in animals, and to examine the relationship between neonatal TSH levels and prenatal exposure to PCB congeners grouped according to their structure and potential mechanisms of action.

Methods: We measured the concentration of 34 PCB congeners in serum samples collected from 285 pregnant women and the thyroid-stimulating hormone (TSH) levels in their children's blood collected shortly after birth.

Results: We found no association between the sum of PCB congeners, the toxic equivalents, or structure-based groupings (mono- or di-*ortho* substituted congeners), and TSH blood concentration. However, we found a positive association between the sum of congeners suspected to be UDP-GT inducers (more specifically cytochrome P450 2B inducers) in animals and neonatal TSH levels. In individual congener analyses, PCBs 99, 138, 153, 180, 183, 187, 194, and 199 were positively associated with neonatal TSH levels after adjustment for covariates. PCBs 194 and 199 remained significant after adjustment for multiple hypothesis testing.

Conclusions: Our results support grouping PCB congeners based on their potential mechanism of action of enzyme induction when investigating associations with TH. Findings also suggest that PCBs affect TH homeostasis even at the low background level of exposure found in the CHAMACOS (Center for the Health Assessment of Mothers and Children of Salinas) population.

Key words: cytochrome P450, enzyme inducers, *in utero*, microsomal enzymes, neonatal, polychlorinated biphenyls, prenatal, thyroid hormone, TSH, UDP-glucuronosyltransferase. *Environ Health Perspect* 115:1490–1496 (2007). doi:10.1289/ehp.9843 available via *http://dx.doi.org/* [Online 29 June 2007]

Polychlorinated biphenyls (PCBs) are synthetic chemicals that were widely used in electrical transformers, inks, plastics, and other consumer products. A total of 209 PCB congeners can be produced depending on the number of chlorine atoms and their position on the biphenyl structure. PCBs are lipophilic and persistent in the environment and bioaccumulate in animals and humans. Although PCBs have been banned in the United States since the 1970s, they can still be measured in most U.S. residents [Centers for Disease Control and Prevention (CDC) 2005].

Several epidemiologic studies have reported that prenatal exposure to PCBs is associated with poorer neurodevelopment in neonates, toddlers, and school-age children (Grandjean et al. 2001; Jacobson and Jacobson 1996; Jacobson et al. 1985, 1990; Koopman-Esseboom et al. 1996; Rogan and Gladen 1991; Rogan et al. 1986). These findings are consistent with animal studies in rodents and rhesus monkeys, which found that *in utero* exposure to PCBs was related to poorer discrimination reversal learning and spatial learning (Levin et al. 1988; Schantz et al. 1989; Widholm et al. 2004). Together with altered neuronal Ca^{2+} signaling (Wong et al. 1997) and reduced dopamine levels (Seegal et al. 1991), disruption of thyroid hormone (TH) homeostasis, which is essential for normal brain development, has been proposed as a potential mechanism for the deleterious neurodevelopmental effects of PCBs. In animals, hypothyroidism affects neuronal proliferation, migration, myelination, and synaptogenesis (Ibarrola and Rodriguez-Pena 1997; Nicholson and Altman 1972; Rami and Rabie 1990). In humans, maternal thyroid status may be of critical importance for fetal neurodevelopment (Haddow et al. 1999; Man and Serunian 1976; Pop et al. 1999), and iodine deficiency–related hypothyroidism is a known cause of cretinism, the leading preventable cause of mental retardation worldwide (Dunn 1993). Congenital hypothyroidism, associated with a variety of pathologies resulting in insufficient TH levels, can also lead to neurodevelopmental problems (de Vilder and Vulsma 2000; Kempers et al. 2006). For this reason, TH levels are routinely measured as part of neonatal screening programs so that TH supplements are administered promptly if necessary.

Several studies suggest that PCBs may disrupt TH levels. PCBs, and especially their hydoxylated metabolites (OH-PCBs), are structurally similar to thyroxine (T_4). Some PCB congeners reportedly induce the microsomal enzyme uridinediphosphate glucuronosyltransferase (UDP-GT), which catalyzes the glucuronidation of T_4 (Hood et al. 2003; Liu et al. 1995). Additionally, dioxin-like PCB congeners can bind to the aryl hydrocarbon receptor, resulting in the induction of the cytochrome P450 CYP1A1 as well as the UDP-GT isoenzyme UGT1A6 (methylcholanthrene-like inducers), which together with UGT1A1 is responsible for the glucuronidation of T_4 in rats (Visser et al. 1993). Other PCB congeners have a phenobarbital-like induction pattern which is characterized by the induction of CYP2B and UGT1A1 (Sugatani et al. 2001). PCBs inducing CYP1A and CYP2B are therefore likely to also induce UDP-GT.

In animals, *in utero* exposure to PCB mixtures as well as to individual PCB congeners decreases free and total T_4 blood levels. Morse et al. (1996) exposed rats to the commercial

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PCB mixture Aroclor 1254 from days 10 to 16 of gestation and found a dose-related reduction in circulating total and free T_4 in fetuses on gestation day 20 and pups on postnatal days 4 and 21. Similar results were obtained with PCB-77 in mice (Darnerud et al. 1996) and OH-PCBs in rats (Meerts et al. 2002).

Human studies suggest a relationship between PCBs and TH, but results differ depending on the PCB congeners studied, the grouping schemes used, and whether TH and PCB levels have been examined in the placenta, maternal, cord, or infant blood. For example, Osius et al. (1999) reported positive associations between PCB-118 and thyroid-stimulating hormone (TSH) both measured in children (n = 320) as well as negative associations between PCBs 138, 153, 180, 183, and 187, and the sum of seven congeners, and free triiodothyronine (T_3). Takser et al. (2005) found negative associations between PCBs 138, 153, 180, and the sum of 11 congeners, and total T_3 in 101 pregnant women, but did not find associations with TSH. In another study, the sum of PCB congeners measured in 160 maternal serum and breast milk samples was not associated with cord blood total T_4, free T_4, or TSH levels (Longnecker et al. 2000). Ribas-Fito et al. (2003) measured seven PCB congeners (28, 52, 101, 118, 138, 153, and 180) in 70 cord blood samples and found no significant association of their individual or summed concentrations with TSH in 3-day-olds. In the only study to measure TSH in neonates in relation to maternal PCB exposure, Koopman-Esseboom et al. (1994) reported significant positive correlations between the nonplanar PCB toxic equivalent (TEQ) measured in breast milk and TSH levels in 2-week-olds and between the planar PCB TEQ, the dioxin TEQ, and the PCB–dioxin TEQ, and infant TSH levels at 2 weeks and 3 months of age.

Animal studies suggest that PCB congeners differ in their mechanisms of action and in their toxicologic potencies (Desaulniers et al. 1997; Khan and Hansen 2003; Li et al. 2001) leading some researchers to develop mechanism-based congener groupings. One such grouping is based on congeners' dioxin-like properties using a method to weigh them by their toxic equivalencies relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) (Van den Berg et al. 2006). Wolff et al. (1997) proposed three groups of PCB congeners based on potential mechanisms of action, structure–activity considerations as well as occurrence in house dust and human samples: Group 1 included PCB congeners that are potentially estrogenic; group 2 included congeners that are potentially antiestrogenic, immunotoxic, and dioxin-like; and group 3 included microsomal enzyme inducers. To date, PCB congeners have not been grouped based on their potential to disrupt TH homeostasis.

Although other mechanisms may be involved, current evidence seems to support the hypothesis that the reduced T_4 levels caused by PCBs in animals occurs at least in part through the induction of UDP-GT. An increase in biliary T_4–glucuronide excretion has been reported following treatment of rodents with PCBs (Bastomsky 1974). Furthermore, administration of Aroclor 1254 to thyrodectomized rats implanted with osmotic pumps delivering T_4 resulted in a reduction of total T_4 and free T_4 by 75% and 70%, respectively, supporting an extrathyroidal mechanism of action (Barter and Klaassen 1992). Among all UDP-GT inducers tested in the study, UDP-GT activity was negatively correlated with serum total and free T_4 levels, supporting the hypothesis that induction of this microsomal enzyme contributes to the PCB-related reduction in circulating TH levels.

The purpose of the present investigation is twofold: to group PCB congeners based on their potential to induce microsomal enzymes in animals; and to examine the relation between neonatal TSH and prenatal exposure to PCB congeners grouped according to their structure and potential mechanisms of actions.

Methods

Participants. Data for this study were collected as part of the Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS), a longitudinal birth cohort study investigating environmental exposures and the health of pregnant women and children residing in the agricultural Salinas Valley, California. Pregnant women planning to deliver at Natividad Medical Center (NMC), a county hospital located in Salinas, California, and receiving prenatal care in this hospital or at one of five clinics of Clinica de Salud del Valle de Salinas were screened for enrollment between October 1999 and October 2000. Women were eligible to participate if they were ≥ 18 years of age, had completed < 20 weeks gestation, spoke English or Spanish, and were Medi-Cal eligible. A total of 601 women agreed to participate in the study, resulting in 538 live births. Excluded from these analyses were participants with insufficient serum volume for PCB analyses (n = 118), no locatable TSH data or only diagnostic information (n = 111), missing neonatal age at the time of blood draw (n = 22), and twins (n = 2). Women included in analyses (n = 285) were similar to those excluded except in that they had spent less time in the United States than excluded women. This study was approved by the University of California, Berkeley, Committee for the Protection of Human Subjects. All participants gave written informed consent before inclusion in the study.

Interviews. Participants were interviewed during pregnancy as well as shortly after delivery in English or Spanish by bilingual, bicultural staff. Sociodemographic information collected included maternal age, family income, the number of people supported by this income, country of birth, and number of years lived in the United States. Information on alcohol, tobacco, drug, and caffeine consumption as well as agricultural work was also collected.

Measurement of neonatal TSH. TSH is routinely measured by the California Department of Health Services Genetic Diseases Branch as part of the state's Neonatal Screening Program. Samples were collected by heel stick and deposited on a filter paper, which was left to dry at room temperature. Dried blood spots were then analyzed with a solid-phase, time-resolved sandwich fluoroimmunoassay (AutoDELFIA; PerkinElmer, Wellesley, MA) using a lanthanide metal europium (Eu) label. These data were abstracted from medical records. On average, blood spot samples were collected 25 hr after birth (range, 4–121 hr).

Measurement of PCBs in maternal serum. Blood samples were collected by venipuncture at the end of the second trimester (mean ± SD gestational age = 26.1 ± 2.9 weeks) and shortly before delivery. Delivery samples were only included in the few cases when the second-trimester sample was not collected (n = 19). Samples were processed at NMC and stored at –80°C until shipment on dry ice to the CDC in Atlanta, Georgia, for analysis. We measured a total of 34 PCB congeners (International Union for Pure and Applied Chemistry nos. 18, 28, 44, 49, 52, 66, 74, 87, 99, 101, 118, 128, 138, 146, 149, 151, 153, 156, 157, 167, 170, 172, 177, 178, 180, 183, 187, 189, 194, 195, 196, 199, 206, 209) by high-resolution gas chromatography/high-resolution mass spectrometry with isotope dilution quantification based on methods previously published (Barr et al. 2003). Quality control samples were included in each run. Simple substitution methods, which consist of assigning values when measurements are below the limit of detection (LOD) such as LOD/2 or LOD/√2, may bias results when detection frequencies are < 90–95% (Lubin et al. 2004). Values below the LOD were therefore randomly imputed from a log-normal distribution whose parameters were determined by maximum likelihood estimation. This method generally produces unbiased parameter estimates (Lubin et al. 2004). LODs for PCBs ranged between 0.01 and 1.92 ng/g lipids. Statistical analyses were restricted to PCB congeners with a detection frequency > 75% and included PCBs 18, 28,

44, 49, 52, 66, 74, 99, 101, 118, 138, 146, 153, 156, 180, 183, 187, 194, and 199.

Triglycerides and total cholesterol were determined using standard enzymatic methods (Roche Chemicals, Indianapolis, IN). Total blood lipid concentrations were then calculated using the method reported by Phillips et al. (1989). PCB values were lipid-adjusted for all analyses by dividing serum PCB concentrations by total blood lipid concentrations.

PCB groupings. PCB congeners were grouped according to previously proposed structure-based and mechanism-based groupings (Van den Berg et al. 2006; Wolff et al. 1997). Structure-based groupings were generated by summing the individual levels of mono-*ortho* (PCBs 28, 66, 74, 118, 156, 157, 167, and 189) and di-*ortho* substituted (PCBs 18, 44, 49, 52, 87, 99, 101, 128, 138, 146, 153, 172, 180, and 194) PCBs. The mechanism-based methods included the three groupings proposed by Wolff et al. (1997) and the TEQ method. We calculated TEQs using the World Health Organization's toxic equivalency factors (TEFs) (Van den Berg et al. 2006). In addition, we created *a priori* another mechanism-based grouping of PCBs according to their ability to induce UPD-GT, CYP1A [or 7-ethoxyresorufin-*O*-deethylase (EROD)] and CYP2B [or 7-pentoxyresorufin-*O*-dealkylase (PROD)] in mammals. PCB congeners likely to induce these enzymes were identified through a search in PubMed (http://www.ncbi.nlm.nih.gov/sites/entrez) and through referenced articles (Table 1). PCB congeners were considered enzyme inducers if a continuous linear or nonlinear dose response could be identified without otherwise overt toxic effects. Doses necessary to induce microsomal enzymes vary among animal species and strains tested (Boobis et al. 1990). We considered a dose–response relationship in at least one strain for UDP-GT, CYP1A, or CYP2B as an indication of possible UDP-GT induction in humans. PCB congeners in the final grouping included PCBs 52, 99, 101, 118 153, 156, 157, 167, 180, 183, 187, 189, 194, and 199. Because dioxin-like PCBs induce CYP1A, the enzyme inducers grouping included these congeners.

Statistical analyses. We used Pearson's correlations to evaluate the interrelationship of PCB congeners. We then used one-way analysis of variance (ANOVA) to compare PCB levels by demographic characteristics. We constructed multiple linear regression models to test the association of both individual PCB congeners as well as PCB groupings with neonatal TSH levels. Covariates considered in regression models included (categorized as shown in Table 2 or as indicated below): maternal age (continuously), race, country of birth, marital status (married or not), years of education, prepregnancy body mass index (BMI; continuously); cigarette, alcohol, and caffeine consumption during pregnancy (none vs. any); gestational age at time of blood collection for PCB analysis (continuously); and neonate's birth weight (continuously), sex, gestational age at birth (continuously), birth order (continuously), and age (in hours) at the time of heel stick (continuously). Covariates were selected for final models if they were related with the outcome ($p < 0.20$); final models included neonatal age at the time of heel stick, gestational age at birth, infant birth weight, sex, and mother's prepregnancy BMI.

We also considered the potential confounding effect of other environmental chemicals such as lead, organophosphate pesticides, and other organochlorine compounds including *o,p′*-dichlorodiphenyltrichloroethane (DDT), *p,p′*-DDT, *p,p′*-dichlorodiphenyltrichloroethylene (DDE), β- and γ-hexachlorocyclohexane, dieldrin, hexachlorobenzene, heptachlor epoxide, mirex, oxychlordane, and *trans*-nonachlor. Organochlorine compounds were measured concurrently with PCBs using the method described above and were lipid-adjusted. We determined exposure to organophosphate pesticides by averaging the concentration of dialkyl phosphate metabolites measured in urine by high-resolution gas chromatography–tandem mass spectrometry with isotope dilution quantification; these samples were collected twice during pregnancy (mean gestational age, 13 and 26 weeks) (Bradman et al. 2005; Bravo et al. 2004; Eskenazi et al. 2004). We measured lead in maternal and cord blood using graphite furnace atomic absorption spectrophotometry.

TSH levels surge at birth and decrease sharply within the first days of life, the period during which blood was collected for TSH analysis. Neonate's age at the time of blood collection (expressed in hours) was negatively associated with both PCBs and TSH in our data. $\log_{10}$(age) maximized the correlation between age at the time of blood collection and $\log_{10}$(TSH) and was entered as such in the models ($r = -0.6$, $p < 0.001$). As an alternative, we also age-standardized TSH levels based on data obtained from the neonatal screening program administered by the Genetics Disease Branch of the California Department of Health (http://www.dhs.ca.gov/pcfh/gdb/html/NBS/) ($n = 1{,}330{,}213$).

The concentrations of the different PCB congeners are usually highly intercorrelated, so the association of a specific grouping with neonatal TSH may be attributed to

Table 1. References supporting microsomal enzyme induction by individual PCB congeners in animals.

PCB congener	Microsomal enzymes		
	UDP-GT	CYP1A/EROD	CYP2B/PROD
42			Connor et al. 1995
47			Connor et al. 1995
52			Connor et al. 1995
77	Desaulniers et al. 1997; Seo et al. 1995; Visser et al. 1993	Chu et al. 1995; Morse et al. 1995; Seo et al. 1995	
99			Connor et al. 1995
101			Connor et al. 1995
105	Chu et al. 1998	Chu et al. 1998	Chu et al. 1998; Connor et al. 1995
118		Chu et al. 1995; Connor et al. 1995; Kuriyama et al. 2003	Connor et al. 1995
126	Craft et al. 2002; Seo et al. 1995; Van Birgelen et al. 1995	Chu et al. 1994; Craft et al. 2002; Desaulniers et al. 1999; Seo et al. 1995; Van Birgelen et al. 1995	
128		Lecavalier et al. 1997	
149			Li et al. 2001
153	Craft et al. 2002	Chu et al. 1996; Desaulniers et al. 1999	Connor et al. 1995; Craft et al. 2002; Desaulniers et al. 1999; Ikegwuonu et al. 1996
154			Connor et al. 1995
156	Van Birgelen et al. 1995	Van Birgelen et al. 1994	Van Birgelen et al. 1994
163			Connor et al. 1995
169	Morse et al. 1993	Morse et al. 1993	Morse et al. 1993
170			Connor et al. 1995
180			Connor et al. 1995
183			Connor et al. 1995
187			Connor et al. 1995
189			Connor et al. 1995
194			Connor et al. 1995
199			Connor et al. 1995

uncontrolled confounding by PCB congeners not included in models. Therefore, we also included the sum of those PCB congeners that were not part of any particular grouping in each model.

As described above, we ran multiple models to examine associations of neonatal TSH with individual PCB congeners. The Bonferroni adjustment method may be too conservative when a large number of tests are carried out. We therefore used the bootstrap-based single-step maxT multiple testing procedure proposed by Dudoit et al. (2004), which adjusts for multiple hypothesis testing while accounting for the correlation between exposures by estimating the joint distribution of test statistics (multtest package in R; R Foundation for Statistical Computing, Vienna, Austria).

Adding congeners assumes an equal potency for every component of a given grouping, which may not be appropriate. Thus, we also analyzed the data by using principal-component analysis to summarize both the group of enzyme inducers and congeners that have not been identified as enzyme inducers. Scores for each group's first component (defined as the loadings-weighted sum of PCB congeners) were then entered as the independent variable in a multiple linear regression model along with the covariates identified above.

Missing values for PCB congeners were imputed based on a nearest neighbor (Euclidian distance) method (impute package in R). Values for environmental exposures (including PCBs) and TSH were expressed on the $\log_{10}$ scale for statistical analyses. Analyses were performed with Intercooled STATA, version 8.1 (StataCorp., College Station, TX) and R, version 2.3.0 (R Foundation for Statistical Computing, Vienna, Austria).

Table 2. Demographic characteristics in a population of pregnant women and their children in the Salinas Valley, CA (n = 285).

		Sum PCBs (ng/g lipids)[a]	
	No. (%)	Geometric mean	95% CI
Maternal			
Age (years)			
18–24	142 (50)	54.1	49.2–59.5*#
25–29	89 (31)	59.9	53.1–67.5
30–34	35 (12)	72.9	60.2–88.3
35–45	19 (7)	87.5	72.3–105.9
Race			
White	3 (1)	38.9	9.7–156.3
Latina	275 (96)	59.9	55.9–64.2
Other	7 (2)	67.3	50.2–90.3
Education			
≤ 6th grade	118 (41)	55.7	50.1–62.0#
7–12th grade	100 (35)	59.4	53.0–66.5
≥ High school	67 (23)	68.5	60.1–78.1
Income (% poverty)			
< 100	162 (61)	59.3	54.3–64.7
100–200	96 (36)	60.5	53.7–68.2
> 200	9 (3)	49.1	29.9–80.4
Country of birth			
United States	34 (12)	61.9	50.0–76.5
Mexico	244 (86)	59.8	55.6–64.3
Other	7 (3)	52.0	34.5–78.6
Time in the USA (years)			
≤ 5	156 (55)	59.0	53.7–64.7
6–10	69 (24)	61.3	53.4–70.4
≥ 11	60 (21)	60.4	52.5–69.5
Parity			
0	103 (36)	61.6	54.8–69.2
≥ 1	183 (64)	58.8	54.2–63.9
Smoking during pregnancy			
Yes	12 (4)	68.7	48.5–97.3
No	274 (96)	59.5	55.5–63.7
Infant			
Sex			
Male	147 (52)	58.2	52.9–63.9
Female	138 (48)	61.6	56.0–67.8
Birth weight (g)			
< 2,500	9 (3)	66.8	49.9–89.4
2,500–3,500	150 (52)	58.6	53.3–64.4
> 3,500	126 (44)	60.8	55.0–67.2
Gestational age at birth (weeks)			
< 37	22 (8)	61.4	49.7–75.7
37–42	264 (92)	59.7	55.6–64.1
> 42	0 (0)	—	—

[a]PCBs with a detection frequency ≥ 75% were summed. *p < 0.05 ANOVA. #p < 0.05 linear trend by Pearson's correlation.

Results

Most participating women were young, Latina, born in Mexico, and had little education (Table 2). Almost all spoke Spanish at home (94%), and most were below the federal poverty line (61%) and had at least one household member working in agriculture (76%). There were slightly more male than female children (52 vs. 48%). Approximately 3% of the newborns had a low birth weight (< 2,500 g), and 8% were premature (< 37 weeks gestation).

TSH levels were within the reference range for all children (≤ 25 mIU/L) with a geometric mean of 5.7 mIU/L [95% confidence interval (CI), 5.3 to 6.1]. Gestational age at birth and birth weight were positively associated with TSH levels (data not shown). No other covariates were related to TSH levels.

The sum of those PCB congeners with a detection frequency > 75% increased with maternal age (p < 0.001) and with the number of years of education (p < 0.05; Table 3). Levels were also higher in women who smoked during pregnancy, but the difference was not statistically significant.

Only PCBs 183 (β = 0.11; 95% CI, 0.02 to 0.20) and 199 (β = 0.10; 95% CI, 0.01 to 0.20) were significantly positively related to neonatal TSH levels in univariate regressions (data not shown); however, as shown in Table 4, 6 of the 19 PCB congeners detected in > 75% of the samples—namely PCBs 101, 180, 183, 187, 194, and 199—were found to

Table 3. PCB levels (geometric means), detection frequencies, ranges, and LOD ranges in a population of pregnant women in the Salinas Valley, CA.

	No.	LOD range	Detection frequency (%)	Geometric mean	95% CI	Range
ΣPCBs[a] (ng/g)	285	0.02–1.92	100	59.8	56.0–64.0	15.3–323.7
TEQ (pg/g)	285	0.004–0.086	100	0.86	0.75–0.97	< LOD–5.17
Inducers[b] (ng/g)	285	0.03–1.44	100	38.9	36.3–41.6	10.0–250.3
Mono-*ortho*[c] (ng/g)	285	0.03–1.11	100	29.4	27.2–31.7	6.4–127.7
Di-*ortho*[d] (ng/g)	285	0.03–1.44	100	27.6	25.9–29.5	7.3–213.1
Wolff method						
Group 1[e] (ng/g)	285	0.03–1.92	100	8.3	7.6–9.1	0.4–62.9
Group 2[f] (ng/g)	285	0.03–1.11	100	13.2	12.3–14.1	2.1–69.1
Group 3[g] (ng/g)	285	0.04–1.09	100	8.4	7.7–9.1	0.6–138.0
Individual congeners						
PCB-18 (ng/g)	279	0.03–0.88	100	6.5	5.9–7.1	0.9–32.1
PCB-28 (ng/g)	285	0.03–0.79	100	17.4	15.8–19.1	3.1–91.7
PCB-44 (ng/g)	238	0.06–1.92	98.7	2.3	2.1–2.6	< LOD–11.4
PCB-49 (ng/g)	250	0.05–1.40	99.2	1.5	1.4–1.7	< LOD–7.9
PCB-52 (ng/g)	261	0.05–1.89	99.2	3.0	2.7–3.3	< LOD–12.4
PCB-66 (ng/g)	276	0.39–1.02	100	2.8	2.5–3.0	0.4–16.0
PCB-74 (ng/g)	276	0.04–1.11	100	4.1	3.8–4.4	0.7–20.8
PCB-99 (ng/g)	263	0.06–1.09	100	1.8	1.7–1.9	0.5–11.6
PCB-101 (ng/g)	241	0.06–1.44	94.6	0.9	0.8–1.1	< LOD–5.8
PCB-118 (ng/g)	270	0.05–0.86	99.6	3.4	3.2–3.7	< LOD–4.7
PCB-138 (ng/g)	263	0.03–0.65	100	2.5	2.3–2.7	0.2–30.9
PCB-146 (ng/g)	249	0.05–0.60	87.6	0.5	0.4–0.5	< LOD–14.7
PCB-153 (ng/g)	273	0.04–0.70	100	5.6	5.2–6.0	0.3–95.6
PCB-156 (ng/g)	270	0.07–0.55	85.2	0.4	0.4–0.5	< LOD–6.3
PCB-180 (ng/g)	231	0.06–0.96	100	1.5	1.4–1.7	0.3–30.0
PCB-183 (ng/g)	259	0.06–0.47	78.0	0.3	0.3–0.4	< LOD–8.2
PCB-187 (ng/g)	224	0.04–0.71	96.9	0.9	0.8–1.0	< LOD–38.3
PCB-194 (ng/g)	263	0.03–0.50	95.4	0.5	0.5–0.6	< LOD–8.6
PCB-199 (ng/g)	271	0.03–0.62	85.6	0.4	0.3–0.4	< LOD–7.5

[a]Sum of all PCBs with a detection frequency ≥ 75% (listed above). [b]Enzyme inducers include PCBs 52, 99, 101, 118, 153, 156, 180, 183, 187, 194, and 199. [c]Mono-*ortho* PCBs include PCBs 28, 66, 74, 118, and 156. [d]Di-*ortho* PCBs include PCBs 18, 44, 49, 52, 99, 101, 138, 146, 153, 180, and 194. [e]Group 1 includes PCBs 44, 49, 52, 101, 187, and 199. [f]Group 2 includes PCBs 66, 74, 118, 138, and 156. [g]Group 3 includes PCBs 99, 153, 180, and 183.

be significantly related to TSH levels, after adjustment for neonatal age at the time of blood draw (β = 0.08–0.14, $p < 0.05$). Three additional congeners, PCBs 99, 138, and 153, were significantly positively related to TSH after adjustment for all covariates (PCB-99: β = 0.11; 95% CI, 0.02 to 0.21; PCB-138: β = 0.09; 95% CI, 0.01 to 0.18 and PCB-153: β = 0.08; 95% CI, 0.00 to 0.17).

PCBs 194 and 199 remained significantly associated with TSH levels after adjustment for multiple hypothesis testing (PCB-194: β = 0.12; 95% CI, 0.01 to 0.24; PCB-199: β = 0.14; 95% CI, 0.02 to 0.25), whereas PCBs 101, 183, and 187 almost reached statistical significance (PCB-101: β = 0.09; 95% CI, –0.01 to 0.18; PCB-183: β = 0.13; 95% CI, –0.01 to 0.23; PCB-187: β = 0.09; 95% CI, –0.01 to 0.20).

Total PCB levels, structure-based groupings (mono-*ortho* and di-*ortho* substituted PCBs), and the TEQ of dioxin-like PCBs were not significantly associated with neonatal TSH levels (Table 4). However, the sum of PCBs specifically hypothesized to induce T_4-metabolizing enzymes was positively associated with neonatal TSH levels; each 10-fold increase in the sum of enzyme-inducing PCBs was associated with a 29% (95% CI, 2 to 62%) increase in TSH (computed from Table 4). The association was linear on a log–log scale, as shown in Figure 1. Including the sum of PCBs not found to be enzyme inducers in the same model or removing extreme values did not materially alter results (data not shown). The principal-component analysis–computed factor summarizing the serum level of potential enzyme inducers (accounting for 64% of the variance) was also significantly associated with neonatal TSH (β = 0.015, 95% CI, 0.005 to 0.024), whereas the factor representing noninducers (accounting for 63% of the variance) was not (β = 0.006, 95% CI, –0.005 to 0.017). Furthermore, the sum of congeners included in group 3 of the classification proposed by Wolff et al. (1997), which is also based on enzyme induction, was significantly associated with TSH levels (β = 0.11; 95% CI, 0.02 to 0.20). The fact that we found a significant association with the sum of all potential enzyme inducer PCBs but not with the TEQ suggests that CYP2B inducers were primarily associated with TSH. This was confirmed when we grouped PCBs by specific enzyme induced (CYP2B/PROD inducers: β = 0.11; 95% CI, 0.01 to 0.21).

Results above were similar whether neonate's age at the time of TSH measurement was controlled for by including the variable as a covariate in models or by standardizing with the use of an external population (California Department of Health Services, Genetic Diseases Branch). The method used to account for blood lipids (including it as a covariate or expressing PCBs on a lipid basis) did not appreciably alter results.

Table 4. Age-adjusted and fully adjusted associations between prenatal exposure to PCBs and neonatal TSH levels.

	Age-adjusted		Fully adjusted[a]	
	β	95% CI	β	95% CI
ΣPCBs[b]	0.05	–0.05 to 0.15	0.06	–0.05 to 0.16
TEQ	–0.01	–0.10 to 0.08	0.00	–0.10 to 0.09
Inducers[c]	0.09	–0.01 to 0.19	0.11	0.01 to 0.21
Mono-*ortho*[d]	0.01	–0.08 to 0.10	0.01	–0.08 to 0.10
Di-*ortho*[e]	0.08	–0.03 to 0.19	0.09	–0.02 to 0.20
Wolff method				
Group 1[f]	0.05	–0.04 to 0.14	0.06	–0.03 to 0.14
Group 2[g]	0.04	–0.07 to 0.14	0.05	–0.06 to 0.16
Group 3[h]	0.09	0.00 to 0.18	0.11	0.02 to 0.20
Individual congeners				
PCB-18	0.03	–0.05 to 0.10	0.03	–0.04 to 0.10
PCB-28	0.01	–0.06 to 0.08	0.02	–0.06 to 0.08
PCB-44	0.03	–0.05 to 0.10	0.03	–0.04 to 0.11
PCB-49	0.02	–0.06 to 0.09	0.02	–0.05 to 0.09
PCB-52	0.03	–0.05 to 0.11	0.03	–0.05 to 0.11
PCB-66	0.00	–0.08 to 0.08	0.01	–0.07 to 0.09
PCB-74	0.03	–0.07 to 0.12	0.03	–0.06 to 0.13
PCB-99	0.09	–0.00 to 0.18	0.11	0.02 to 0.21
PCB-101	0.08	0.02 to 0.15	0.09	0.03 to 0.16
PCB-118	0.01	–0.08 to 0.11	0.03	–0.07 to 0.13
PCB-138	0.07	–0.01 to 0.15	0.09	0.01 to 0.18
PCB-146	0.06	–0.02 to 0.14	0.07	–0.01 to 0.15
PCB-153	0.07	–0.01 to 0.15	0.08	0.00 to 0.17
PCB-156	0.04	–0.03 to 0.11	0.05	–0.03 to 0.12
PCB-180	0.08	0.00 to 0.15	0.09	0.01 to 0.17
PCB-183	0.12	0.05 to 0.19	0.13	0.05 to 0.20
PCB-187	0.09	0.02 to 0.16	0.09	0.02 to 0.17
PCB-194	0.11	0.03 to 0.19	0.12	0.04 to 0.20
PCB-199	0.14	0.07 to 0.22	0.14	0.07 to 0.22

[a]Models adjusted for neonatal age at time of heel stick for TSH measurement, gestational age at birth, infant birthweight, sex and mother's prepregancy BMI. [b]Sum of all PCBs with a detection frequency ≥ 75%. [c]Enzyme inducers include PCBs 52, 99, 101, 118, 153, 156, 180, 183, 187, 194, and 199. [d]Mono-*ortho* PCBs include PCBs 28, 66, 74, 118, and 156. [e]Di-*ortho* PCBs include PCBs 18, 44, 49, 52, 99, 101, 138, 146, 153, 180, and 194. [f]Group 1 includes PCBs 44, 49, 52, 101, 187, and 199. [g]Group 2 includes PCBs 66, 74, 118, 138, and 156. [h]Group 3 includes PCBs 99, 153, 180, and 183.

Discussion

Results from this study suggest that prenatal exposure to PCB congeners that induce CYP2B in animals is positively associated with TSH levels in children shortly after birth. In animals, UDP-GT (UGT1A1) is induced concurrently with CYP2B following exposure to phenobarbital-like compounds, which may explain the observed association (Sugatani et al. 2001). However, we found no association between levels of TSH and total PCBs, PCBs grouped by structure (mono- and di-*ortho*–substituted PCBs), or dioxin-like PCBs (TEQ). These findings were observed in the CHAMACOS population, which had low exposure to PCBs compared with the general U.S. population (CDC 2005). The median PCB-153 concentration, for instance, was 5.6 times lower in our participants than in the NHANES (National Health and Nutritional Examination Survey) sample (5.4 vs. 30.1 ng/g lipids).

Our findings differ from those of Koopman-Esseboom et al. (1994), who found associations between maternal dioxin-like PCBs (TEQ) and neonatal TSH levels, and of Wang et al. (2005), who reported associations between the placental dioxin/PCB TEQ and cord blood TSH levels. Although we may have underestimated the TEQ in our population because we did not measure the levels of two key dioxin-like PCBs (PCBs 126 and 169), the exposure level in the Dutch study appeared to be substantially higher (about 20 times) than in the current study (Longnecker et al. 2003). Our results also differ from those of previous studies that found no association of TSH levels with individual PCB congeners (Ribas-Fito et al. 2003; Takser et al. 2005). However, our findings are consistent with those of Longnecker et al. (2000), Takser

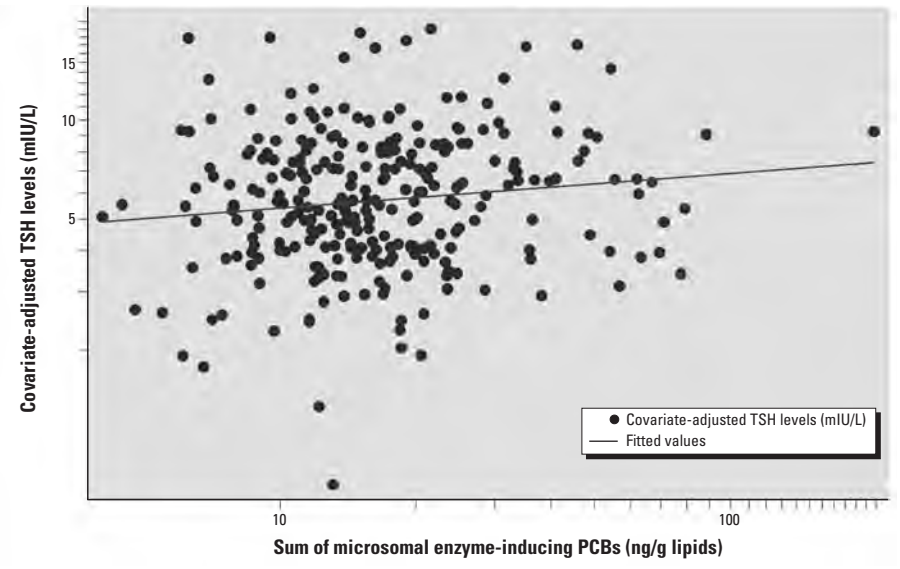

Figure 1. Association between covariate-adjusted TSH levels and the sum of microsomal enzyme-inducing PCB congeners in neonates ($n = 285$).

et al. (2005), and Ribas-Fito et al. (2003), who did not find an association between the sum of all PCB congeners measured and TSH levels in pregnant women, neonates, and cord blood. Our results also agree with the lack of association between the structure-based grouping of mono-*ortho*–substituted PCB congeners and TSH levels, as reported in two previous studies (Takser et al. 2005; Wang et al. 2005).

Serum concentrations of PCB congeners are highly intercorrelated, yet individual congeners may differ in their health effects and mechanisms of action. Except for a study by Wolff and Toniolo (1995), who investigated associations with breast cancer, we are not aware of any other study that grouped PCBs based on the mechanism of enzyme induction. To date, no study examining the potential of PCBs to disrupt TH has grouped congeners based on their potential to specifically disrupt TH.

One of the main strengths of this study is that we attempted to characterize *a priori* PCB congeners based on their potential to affect TH. We grouped PCBs based on evidence from animal studies suggesting the potential of specific PCB congeners to induce UDP-GT. We also considered the potential confounding effect of a large number of demographic and environmental covariates. Our findings were further supported by principal-component analysis, with the first factor summarizing enzyme-inducing congeners being significantly associated with neonatal TSH levels whereas the factor summarizing other congeners was not. Strengths also include the control for multiple hypothesis testing and our use of distribution-based imputation techniques for values below the LOD.

There are limitations to the method we propose, some of which stem from limited data. First, we could not find published data for seven of the 19 congeners commonly found in the CHAMACOS population (detection frequency > 75%). Also, our method does not consider other potential mechanisms of action by which PCBs could affect TH levels, including the binding of PCBs to transthyretin and displacement of T_4 (Chauhan et al. 2000), increased liver T_4 uptake and decreased pituitary sensitivity to thyroid-releasing hormone (Khan and Hansen 2003), altered T_4 and T_3 synthesis (Collins and Capen 1980), and inhibited thyroid gland response to TSH (Byrne et al. 1987). Summing the concentrations of PCB congeners also assumes equal potencies, which may not be appropriate. Summarizing the grouping with principal-component analysis–derived factors, though avoiding the equal potencies assumption of the summation method, still assumes no synergistic effect. An ideal grouping would consider all potential mechanisms of action, different relative potencies, and interactions. One strategy to improve our method might be to develop a TEQ-like weighing system (Van den Berg et al. 2006). Such a scheme would require more congener-specific data and would need to consider whether a simple linear additive model appropriately represents observed effects at environmentally relevant doses.

In summary, we report a positive association between neonatal TSH levels and prenatal exposure to PCBs reported to induce microsomal enzymes (specifically CYP2B) and suspected to induce UDP-GT in animals but not with the sum of all PCB congeners, or PCBs grouped according to their dioxin-like activity or structure. This is the largest study to date investigating prenatal exposure to PCBs and neonatal TSH. If replicated, our findings would support the hypothesis that not all PCB congeners disrupt thyroid hormones, and would argue against summing all PCB congeners. However, they would support grouping PCB congeners based on their potential mechanism of action of UDP-GT induction. Our results also suggest that PCBs affect TH homeostasis even at the low background level of exposure found in the CHAMACOS population. Although TSH remained within the reference range, previous animal and human studies suggest that maternal hypothyroxinemia (low free T_4 and normal TSH levels) during early pregnancy adversely affects neurodevelopment (Morreale de Escobar et al. 2000; Pop et al. 1999). Future studies should examine whether TH levels within the reference range in neonates may be related to neurodevelopment.

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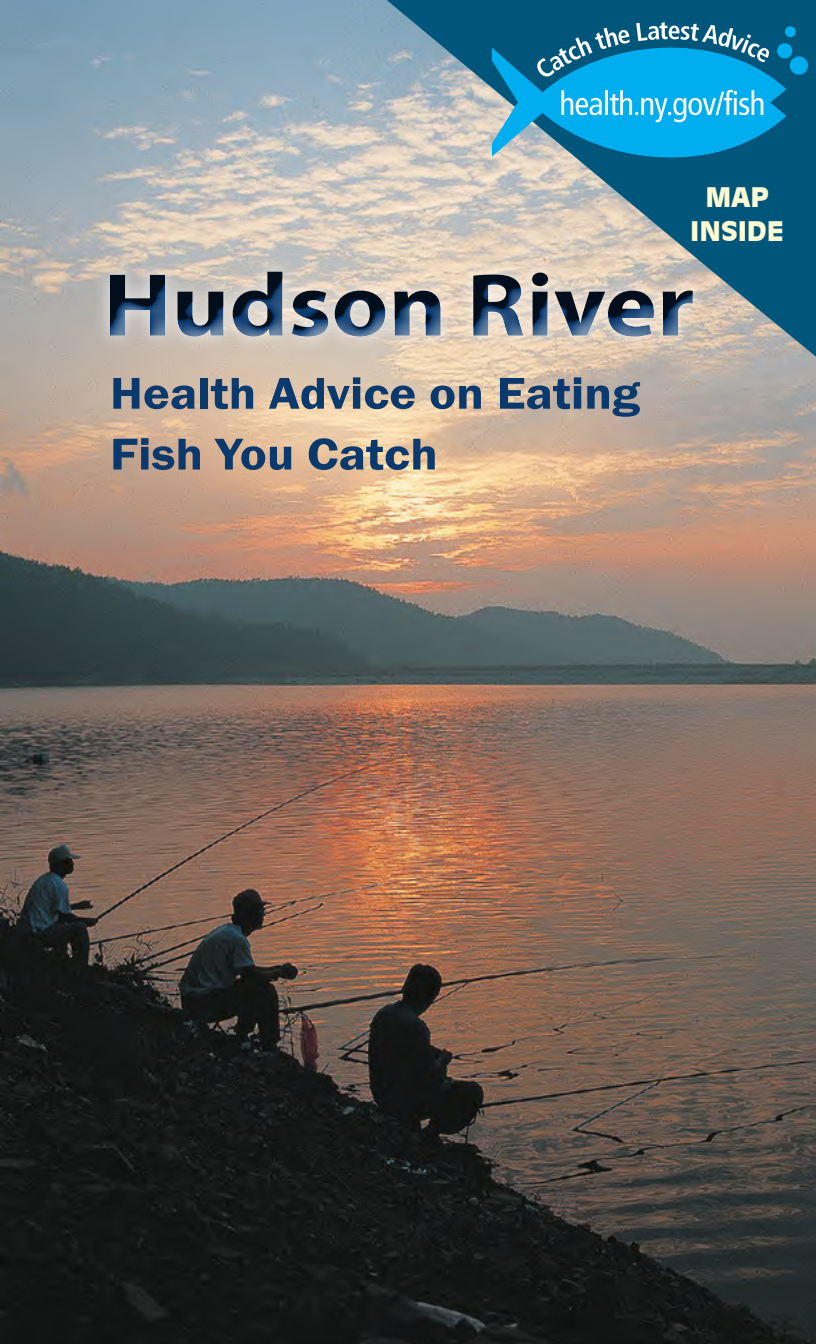
Attachment F

Hudson River Health Advice on Eating Fish You Catch, DOH



Hudson River

Health Advice on Eating Fish You Catch

Why We Have Advisories

Fishing is fun and fish are an important part of a healthy diet. Fish contain high quality protein, essential nutrients, healthy fish oils, and are low in saturated fat. However, some fish contain chemicals at levels that may be harmful to your health. The primary chemicals of concern in Hudson River fish are called polychlorinated biphenyls (PCBs) and they can build up in your body over time. Health problems that may result from eating fish with PCBs range from small changes in health that are hard to detect to effects on birthweight and cancer. (Visit www.health.ny.gov/fish for more info.) Eating Hudson River fish can be a concern because fish can have many thousands times more PCBs than the surrounding water.

New York State Department of Health (NYSDOH) offers advice about eating fish you catch. The health advice about which fish to eat depends upon:

Who You Are

Women of childbearing age (under 50) and children under 15 should not eat fish or crabs from the Hudson River from the Corinth Dam to the New York City Battery. Women who eat highly contaminated fish and become pregnant may have an increased risk of having children who are slower to develop and learn. Chemicals may have a greater effect on the development of young children or unborn babies. Also, some chemicals may be passed on in mother's milk.

Women beyond their childbearing years and men may face fewer health risks from PCBs. For that reason, the advice for women over age 50 and men over age 15 allows them to eat more kinds of sportfish and more often, particularly in the lower Hudson.

Where You Fish

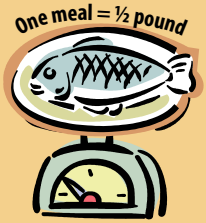
The advice on eating Hudson fish depends upon where on the river you fish. The Hudson River around Hudson Falls has been more affected by industrial chemicals. **In general, fish from the lower Hudson are less contaminated.** For example, from the Federal Dam at Troy to the Rip Van Winkle Bridge at Catskill, no one is advised to eat striped bass. However, south of Catskill, men and older women can eat up to one striped bass meal a month.

The Hudson River advice also applies to its tributaries and connected waters if there are no dams, falls, or barriers to stop the fish from moving upstream. This is because chemicals remain in fish when they move from one waterbody to another. If you are not sure about possible fish barriers near waters where you are fishing, see the DEC information on the back of this brochure.

What You Catch

Some species (kinds of fish) have higher levels of chemicals than others. In general, smaller fish are less contaminated than larger, older fish of the same species. PCBs are also found at higher levels in the fat of fish. **Reduce PCBs by trimming, skinning, and cooking your catch as shown in the diagram on the back of brochure.**

Certain species with a lot of fat, like catfish and eels, should be avoided because they have high levels of PCBs.



Visit www.health.ny.gov/fish for the latest fish advisory information

Upstream of South Glens Falls Dam

Visit www.health.ny.gov/fish for this advice or see the Northern Hudson River brochure.

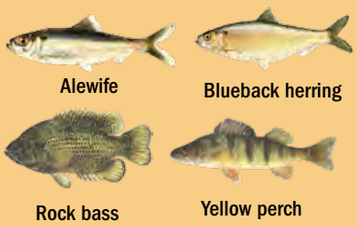
Upper Hudson

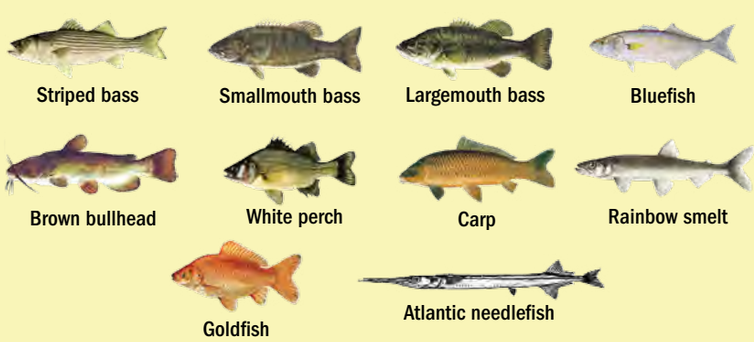
From South Glens Falls Dam to Federal Dam at Troy

Do not eat fish from the South Glens Falls Dam to the Federal Dam at Troy.

From Baker's Falls to the Federal Dam at Troy, New York's State Department of Environmental Conservation's "catch and release" regulations apply.

Take No Fish. Eat No Fish.

Mid Hudson From Federal Dam at Troy to Rip Van Winkle Bridge at Catskill	Men over 15 and Women over 50	Women under 50 and Children under 15
 Alewife, Blueback herring, Rock bass, Yellow perch	Up to 1 meal/month	DON'T EAT
All other fish from the Mid Hudson (including Striped bass and Walleye)	DON'T EAT	DON'T EAT

Lower Hudson From Rip Van Winkle Bridge at Catskill to the NYC Battery	Men over 15 and Women over 50	Women under 50 and Children under 15
 Walleye, White catfish, Channel catfish, American eel*, Gizzard shad **DEC regulations prohibit taking American eel for food from the Hudson River*	DON'T EAT	DON'T EAT
 Striped bass, Smallmouth bass, Largemouth bass, Bluefish, Brown bullhead, White perch, Carp, Rainbow smelt, Goldfish, Atlantic needlefish	Up to 1 meal/month	DON'T EAT
 Blue crab Do not eat the tomalley ("green stuff," mustard, hepatopancreas) or reuse cooking water	Up to 6 crabs/week	DON'T EAT
All other species	Up to 4 meals/month	DON'T EAT

Don't forget that specific advice applies to tributaries and connected waters if there are no dams, falls, or barriers to stop the fish from moving upstream.

The Hudson River Fish Advisory Outreach Project

The NYSDOH Hudson River Fish Advisory Outreach Project has a goal that all anglers and others who eat fish from the Hudson River know about, understand, and follow the advisories.

New York State Fish Advisories

www.health.ny.gov/fish
To be a Hudson River partner, call (518) 402-7530 or 1-800-458-1158
email HRFA@health.ny.gov
www.health.ny.gov/hudsonriverfish

New York State Fishing

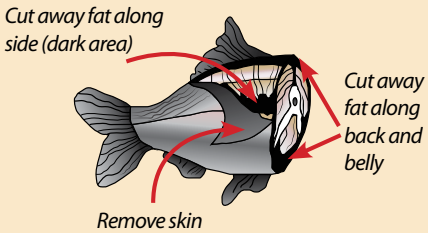
Department of Environmental Conservation (DEC)
Visit www.dec.ny.gov/outdoor/fishing.html; (518) 402-8920

Fish from Stores and Restaurants

Visit www.fda.gov/fishadvice or www.epa.gov/fishadvice

Cut the Fat to Cut PCBs

Follow the advice below to reduce PCBs by nearly one half.



- After trimming as shown, broil, grill, or bake the fish on a rack so that fat drips away.
- Do not pan-fry the fish or use drippings to make stock or sauce.

Do not eat the "green stuff" (tomalley) in crabs or use the cooking liquid.

Attachment G

First Five Year Review Comments

Hudson River Sloop Clearwater
Natural Resources Defense Council
Riverkeeper
Scenic Hudson

May 4, 2012
Judith Enck, Regional Administrator
US EPA Region 2294 Broadway
New York, NY 10007

Dear Judith:

We would like to thank you for your recent decision to provide a short extension of the completion deadline for the mandatory, statutory 5-Year Review of the Hudson River's PCB remediation site, and we truly appreciate your willingness to respond to the concerns we shared with you in our letter dated April 2, 2012.

However, we remain troubled that the Hudson's first 5-Year Review will be limited in scope and fails to allot the time customary for this process, especially in regards to document review and stakeholder participation. Given the scope and complexity of the Remedy and the ongoing issues raised by an array of governmental and non-governmental stakeholders, we believe that a six month completion deadline would allow for an adequate review. Importantly, Section VIII of the 5-Year Review report is supposed to contain "a discussion of unresolved concerns or items raised by support agencies and the community."[1]

Under both CERCLA's statutory language and long-standing EPA policy, a cornerstone of the 5-Year Review process is that cleanup projects must be responsive to current conditions, new information, and technological advances. The 5-Year Review is intended to broadly assess a remedy and ensure that it is designed and implemented to achieve the intended protectiveness for human health and natural systems. Both the law and guidance clearly anticipate that new understandings and advanced removal methods will be incorporated during remediation to ensure the protectiveness of a remedy.

Several federal and state natural resource agencies, along with municipal governments and respected independent scientists, have raised serious concerns including a) the discovery that the Hudson's PCB contamination is much greater than originally assumed and its implication for the remedy's protectiveness b) the effect of this greater contamination on restoration and recovery options, c) the lack of adequate monitoring protocols for sediment and benthic fauna, d) additional exposure pathways that may impact the Remedial Action Objectives. Current quantitative and qualitative analyses not available at the time of the Record of Decision (ROD) or Consent Decree (CD) support these concerns. These and other issues must be included within the scope of the 5-Year Review and examined in conjunction with the project's initial assumptions and predictions, to determine the long-term protectiveness of the remedy. The resulting data will also serve to inform the adaptive-management framework under which the Remedial Action Work Plans (RAWPs) for each successive year of Phase 2

[1] OSWER No. 9355.7-03B-P pg.3-7

dredging are implemented. This is also consistent with the Peer Review Panel recommendation that "in a project of the complexity and duration of the Hudson River PCBs Site cleanup, EPA needs to be able to adapt to new information and make or require changes through adaptive management in order to achieve the expected benefits of the project."

Given the overall requirements and standards involved in this review process, we hope additional time will be devoted to ensure this examination accomplishes all critical components of USEPA's Comprehensive Five-Year Review Guidance documents. Accordingly, as part of that process we have summarized below specific concerns and issues that should be considered as USEPA determines the protectiveness of the remedy.

Impact of Greater PCB Contamination Levels on Protectiveness of Remedy

The amount of PCB toxins expected to remain in the Hudson at the end of the cleanup is a primary trigger for Superfund's' 5-Year Review requirements[2] and is foundational to Remedial Action Objectives (RAOs) in the ROD. However, actual conditions found during in-the-water operations revealed that high levels of PCB contamination are much deeper and more widely distributed than originally assumed. We believe that accurately determining both the depth and areal extent of contamination is a priority issue that must be examined in order to answer the three questions that frame the Hudson's first Five-Year Review. This would be entirely consistent with provisions in the ROD that directed the USEPA to conduct sampling that "will cover both target areas as well as the areas outside the current target area boundaries. In this manner, EPA will produce a current contamination map of the Site on which to finalize its target area selection."[3]

Study Issues

- The discovery of much greater PCB contamination during Phase 1 requires a more comprehensive identification of the vertical and horizontal distribution of toxic sediment for Phase 2. Two significant unknowns are the distribution of contaminated sediment outside of the Dredge Area Delineations (DAD) and how the greater contamination of unremediated areas may reduce the protectiveness of the remedy if not addressed.
- Re-analyze the sediment transport model with the new contamination data to determine the likelihood that unremediated PCBs outside the current DAD would recontaminate the site after dredging is completed.

Impacts of Projected Post-Remedy Contamination Levels on Protectiveness

In addition, federal agencies tasked with completing the Natural Resource Damage Assessment (NRDA) and implementing a restoration plan after the Superfund cleanup is completed have identified, an estimated 136 acres of highly toxic sediment in River Sections 2 & 3 that will be left unremediated in the current remedy, but which the ROD anticipated would be much less highly contaminated than it actually is. This amount of contamination

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[2] CERCLA § 121(c), "If the President [or his delegate, in this case the EPA Administrator] selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site, the President shall review such remedial action no less often than each five years after the initiation of such remedial action to assure that human health and the environment are being protected by the remedial action being implemented."

[3] Responsiveness Summary; Hudson River PCBs Site Record of Decision: Response to Master Comment 605 pg. 4-2.

will continue to impair human health and wildlife recovery, can limit the restoration of the river and may be a source of recontamination to dredged areas if not addressed during the present cleanup.

Study Issues

- Review the results of the federal agencies' analyses and conclusion that, due to greater PCB contamination than assumed under the ROD, response action is not expected to achieve all target cleanup levels in the timeframe expected and therefore an important Remedial Action Objective (RAO) may not be achieved.
- Both federal and state support agencies responsible for the maintenance and health of the river's economic and environmental resources have offered sound guidance to address this issue. USEPA and GE should determine how to incorporate these recommendations into the design and Remedial Action Work Plan (RAWP) in successive years of the cleanup.
- Monitoring of pre- and post-remedy sediment concentrations are not adequate to determine the protectiveness of the remedy, especially in river sections 2 and 3, where recent data estimates that post-remediation PCB concentrations (in the river section as a whole, not limited to the areas within the DAD) will be five times higher than predicted by the USEPA models.
- There is no unlimited use/unrestricted exposure for Phase 1 dredge areas, specifically CU-1, which includes the Ft. Edward yacht basin where sediment redeposition over the remedy cap will impede full use and unrestricted access. Further remediation action should be examined and implemented.
- Future RAWPs should include navigational dredging as part of the dredging design as there will otherwise, be no unlimited use/unrestricted exposure for the navigational channel in the entire 40-mile active remedy area of the Superfund site. This will continue to impede the New York State Canal Corporation from executing its constitutionally mandated dredging responsibility for the Champlain Canal.
- Re-examination of untargeted hot-spots should be conducted in river sections 2 & 3 as under the current approach, there will be no unlimited use/unrestricted exposure for human, wildlife and NRD restoration activities in the Phase 2 dredge area.

Predicting Protectiveness of Remedy for Fish Tissue Concentrations.

Reducing fish tissue levels of PCBs is a major cleanup level parameter in the ROD, but it is no longer clear that the current remedy will meet the timelines projected in the ROD for fish tissue level reductions.

The "protectiveness" provisions in the ROD target the attainment of a fish PCB concentration of 0.4 mg/kg – which was deemed protective of the average adult who consumes one fish meal from the Upper Hudson every two months – within the entire upper Hudson River within 20 years of active remediation. A target PCB fish concentration of 0.2 mg/kg was expected to be attained in River Section 2 within 32 years of active remediation.

The ROD's target reductions in cancer risk correspond to these fish tissue concentrations and timelines; however, other examinations of sediment concentrations, like those described by the Federal Trustees, indicate these targets will not be reached in the timeframe anticipated in the ROD and imply further remediation of heavily contaminated sediment may be necessary.

Study Issues

- Bioaccumulation model assumptions of contaminant concentrations have not been updated to reflect the new sediment contaminant data and projections of fish tissue PCB concentrations are systematically over-optimistic relative to observed values. Re-analysis of this fundamental model with the new sediment contamination data is required to assess the short- and long-term likelihood of the remedy's protectiveness.
- The peer review panel recommended further development of the bioaccumulation model to improve its accuracy for the Hudson River system. A status update should be provided and plans for further model development should be developed.
- Since the ROD, the science of human health risk assessment has evolved, with respect to the use (or misuse) of the "average adult male" as a metric for evaluating risks of exposure to contaminated fish and shellfish. EPA should evaluate the protectiveness of the remedy, for all affected human populations and sub-populations, in light of current best practices for scientific risk assessment.

Institutional Controls and Fish Advisories

Institutional controls are currently inadequate to prevent ongoing overconsumption of contaminated fish (e.g., fish advisories are not preventing subsistence anglers from eating the fish). For example, a 2010 Angler Survey performed by Clearwater along the Peekskill waterfront from Annsville Creek to Verplanck as part of a Community-Based Environmental Justice Inventory reports higher levels of contaminated fish consumption, especially by Environmental Justice populations, than previous surveys. This indicates that far more public education and better signage is needed to effectively prevent this route of exposure to PCBs.[4]

Study Issues

- Review current institutional controls, assess efficacy, and develop enhanced control strategies to improved public awareness and behavior, and minimize exposure in communities.

Optimizing Habitat Reconstruction

The ROD and all subsequent decisions projected a cleanup that substantially reduces PCB contamination in the water and soil and a remedy that leaves behind an environment capable of supporting diverse marine communities that will help heal the river after active remediation is completed. The habitat recovery work is intended to reestablish marine vegetative beds and habitats damaged by dredging operations and residual PCB contamination. However, adjustments to dredge area slopes, backfill sediment profiles and selection of plants must be appropriate for natural and native regeneration to occur. In addition, USEPA should adequately identify, and ensure the repair and restoration of, unique natural resources of the riverine system, like benthic invertebrate populations, that may suffer severe damage during active remediation.

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[4] Citizen's for Equal Environmental Protection (CEEP), Hudson River Sloop Clearwater and Peekskill Environmental Justice Council, Community-Based Environmental Justice Inventory for the City of Peekskill, Dec. 21010 www.clearwater.org/wp-content/images/2011/03/CBEJI_FINAL-_DRAFT-1-30-11-for-printing.pdf

Study Issues

- The five-year review should evaluate pre- and post-dredge habitat assumptions and address state and federal natural resource agency concerns in regards to habitat reconstruction during remediation.

Protectiveness for Human Health

Recent studies by the NYS Department of Health have investigated additional dimensions of public health impacts from PCB exposure, including non-cancer risks and non-consumption exposure pathways. These initial results warrant further assessment of the remedy's protectiveness for human health.

Study Issues

- Review the protectiveness of the remedy in light of the potential for airborne exposure and the larger amount of contamination to remain in place post-remedy.
- New York State's Department of Health Reference Doses (RfDs) for Chronic Oral Exposure has not changed but the USEPA Integrated Risk Information System[5] (IRIS) is currently assessing noncancerous risks from PCBs. The Review should develop a plan for incorporating any new guidance into the remedy as it becomes available.

Protectiveness With Respect to Other Remedial Action Objectives

The goals of the ROD include compliance with ARARs, reduction of cancer and non-cancer health risks to humans through exposure pathways other than fish consumption (such as through primary and secondary contact), reducing the inventory (mass) of PCBs in sediments that are or may be bioavailable, minimizing the long-term downstream transport of PCBs in the river, and compliance with federal and state water quality standards.

Study Issues

- Review the effectiveness of the remedy with respect to all of the ROD's objectives.

Environmental Conditions and Extreme Events

A significant type of site-condition highlighted in 5-Year Review guidance documents is whether the site was subject to a 100-year flood after the remedy was selected. The Upper Hudson experienced this level of flooding in 2011, which scoured PCBs from the unremediated river bottom and sent elevated PCB loads downstream and also was subject to storm events that forced a halt to dredging operations twice. Climate change science also teaches that the frequency of such storms will increase in the coming decades. .

Study Issues

- Review engineering standards of cap and habitat reconstruction and designs in light of the multiple events already experienced by the site and projections for increasing frequency and intensity of storm/flooding events due to climate change.
- Review sediment transport models to determine the likelihood that unremediated PCBs outside the current DAD would recontaminate the site after dredging is completed, under projected future climate conditions.

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[5] USEPA's Integrated Risk Information System (IRIS) is a human health assessment program that evaluates information on health effects that may result from exposure to environmental contaminants.

Returning the economic and ecological potential of the Hudson River to communities long denied these benefits is our highest priority. A measured and focused review of the PCB project will help ensure a cleanup that is responsive and protective in both the short and long-term.

Ned Sullivan, President
Scenic Hudson

Paul Gallay, President
Riverkeeper

Jeff Rumpf, President
Hudson River Sloop Clearwater

Lawrence Levine, Senior Attorney
Natural Resources Defense Council

Cc: Admin. Lisa Jackson (EPA)
Hudson Valley Congressional Delegation
Deputy Sec. Energy & Envir. Bob Hallman (NYS Office of Governor)
Asst. Dep. Sec. Envir. Basil Seggos (NYS Office of Governor)
Asst. Admin. Mathy Stanislaus (EPA)
Asst. Admin. Cynthia Giles (EPA)
Cmr. Joe Martens (DEC)
Dir. Brian Stratton (NYS Canal Corps)
Asst. Cmr. Eugene Leff (DEC)
Eric Schniederman (NY AG)
Brian Donohue (DOJ)

Bcc: Wendi Weber
Robert Foley
Robert Haddad
Tom Brosnan
Kevin Farrar
Joe Moloughny
John Davis (NYS AG's office)

Attachment H

Petition to USEPA

PETITION TO THE UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Petition for Evaluation and Expansion of Remedial Action Selected in the 2002 Record of Decision for the Hudson River PCBs Site	))))	*Submitted December 17, 2015, to Judith Enck, EPA Region 2 Administrator*

Pursuant to the Petition Clause contained in the First Amendment of the United States Constitution,[1] the Administrative Procedure Act,[2] and the Comprehensive Environmental Response, Compensation, and Liability Act ("CERCLA" or the "Superfund Act"), Hudson River Sloop Clearwater, the Natural Resources Defense Council, Riverkeeper, Scenic Hudson, and the Sierra Club, Atlantic Chapter (collectively, "Petitioners") petition the U.S. Environmental Protection Agency ("EPA") to take immediate action to ensure protection of the Hudson River and the health and safety of millions of New Yorkers.

Beginning in 2009, pursuant to a 2006 consent decree with EPA implementing the agency's 2002 Record of Decision, the General Electric Company ("GE") carried out dredging operations in the Upper Hudson in an effort to remove sediments laden with the PCBs that it had dumped into the river for decades. In October of 2015, GE announced that it had completed the dredging program, which EPA declared a success, and on November 12, 2015, EPA approved the decommissioning of the dewatering facility and other critical infrastructure that had supported the dredging operations. This approval constituted a de facto determination by EPA that the dredging remedy selected in the Record of Decision had been satisfactorily completed and that this remedy is protective of human health and the environment.

This de facto determination was arbitrary and capricious. It was made in the face of compelling evidence that the PCBs remaining in the Hudson constituted a real and continuing danger and that the completed dredging had not resulted in conditions that were sufficiently protective of human health and the environment. This was the conclusion reached by the National Oceanic and Atmospheric Administration ("NOAA") in a recent technical analysis regarding the efficacy of EPA's cleanup plan for PCB-contaminated sediments in the Hudson River. This analysis directly contradicted EPA's previous assessments—specifically, in the 2012 Five Year Review—that the dredging remedy was fully and adequately protective. Ultimately, the NOAA analysis concluded that high levels of PCBs will remain in the river, and in Hudson River fish, generations longer than expected unless further dredging is conducted.

EPA ignored this analysis and NOAA's conclusions. Instead, it hued to the position it had taken for more than five years—namely, that the limited dredging GE was required to undertake would

[1] U.S. Const. amend. I (prohibiting laws "abridging freedom of speech, or of the press; or the right of the people peaceably to assemble, and to petition the Government for a redress of grievances"). The right to "petition for redress of grievances" has been described by the U.S. Supreme Court "among the most precious of the liberties safeguarded by the Bill of Rights." *United Mine Workers of America, Dist. 12 v. Illinois State Bar Ass'n*, 389 U.S. 217, 222 (1967).

[2] 5 U.S.C. § 555(e).

still be sufficient to timely achieve the remedial health and safety targets. With this position in mind, the agency blessed the termination of the dredging program, labeling it a "success."

In this and in other respects described below, EPA also violated its non-discretionary duty under CERCLA to ensure that selected remedies are protective of human health and the environment—which includes a responsibility to consider substantial new evidence bearing on the issue. As matters stand now, the agency's de facto approval of the termination of the GE dredging program and its refusal to order additional dredging cannot be sustained.

However, EPA has recently signaled that it may revisit its conclusions by undertaking an accelerated five-year review. Given the agency's history of unwillingness to analyze critical new information bearing on the sufficiency of the remedy, Petitioners have serious concerns as to whether any review at this point would be truly objective. Still, such a review would give EPA an opportunity to take corrective action by undertaking a comprehensive, in-depth evaluation of the dredging remedy that is capable of assessing whether it is truly protective. This evaluation, at a minimum, must include full consideration of the NOAA analysis, other concerns raised by the Trustees and the public, and the issues raised herein. Additionally, EPA must also give serious consideration to new scientific research demonstrating the potential human health harms posed by chronic exposure to airborne forms of PCBs—an exposure pathway specifically not addressed by EPA's current remedy—as well as recent evidence of the failure of longstanding fish consumption advisories to protect human health in the interim.

The review must be transparent, thorough, and inclusive, with ample provision for meaningful participation by interested agencies and the public. Moreover, to the extent that this evaluation demonstrates the selected remedy will not meet EPA-established targets for human health and safety within the requisite timeframes, EPA must take all appropriate action—including expansion of the dredging remedy—to protect human health and the environment.

IDENTITY AND INTEREST OF PETITIONERS

Petitioners are a group of five not-for-profit environmental organizations, all of which have a strong connection to New York's environment and the Hudson River. They include the Hudson River Sloop Clearwater, Natural Resources Defense Council, Riverkeeper, Scenic Hudson, and the Sierra Club, Atlantic Chapter. Each of these organizations has a long-standing interest in the health and ecological well-being of the Hudson River, including an interest in ridding the River of PCB contamination. Petitioners' respective statements of interest are included in Exhibit A to this Petition.

BACKGROUND

I. PCBs in the Hudson River Pose a Significant Threat to Human and Animal Health

Polychlorinated Biphenyls ("PCBs") are manmade, bioaccumulative persistent-organic-pollutants that are known to cause a wide variety of adverse health effects. As EPA states:

> PCBs have been shown to cause cancer in animals. PCBs have also been shown to cause a number of serious non-cancer health effects in animals, including effects on the immune system, reproductive system, nervous system, endocrine system and other health effects. Studies in humans provide supportive evidence for potential carcinogenic and non-carcinogenic effects of PCBs. [3]

Exposures to PCBs can occur through consuming contaminated food or water, direct skin contact, or breathing contaminated air.[4] Non-cancer risks from exposure to PCBs likely include, among others: dermal and ocular lesions; liver and kidney disorders; reduced birth weight, conception rates, and live birth rates; persistent and significant deficits in neurological development, including visual recognition, short-term memory and learning; and developmental problems due to interference with thyroid hormone levels.[5]

Because PCBs do not readily break down in the environment and accumulate in animal fat and other tissue when ingested, PCB contamination of river sediments can spread throughout the food chain from low level river bottom fauna to fish, birds, and land animals (including, of course, humans).[6]

As recent anglers surveys have shown, consumption of fish from the Hudson River remains a major health concern for New Yorkers, despite the existence of longstanding New York Department of Health ("NYSDOH") fish consumption advisories. In 2012, for example, the Cornell Cooperative Extension performed a survey of over 300 anglers, finding that approximately 11% of those surveyed ate Hudson River fish.[7] In 2013, NYSDOH presented preliminary results of its own angler survey showing even higher consumption percentages (near 50%), also noting that awareness of fish consumption advisories in the more populated and linguistically diverse Lower Hudson was about half of what it was in the Mid and Upper Hudson regions.[8]

Further, within the last decade, a growing body of research has highlighted the severity of the potential risks from "volatilized" or airborne PCBs, which have been associated with certain chronic illnesses—such as cancer, cardiovascular disease, hypertension, and diabetes—even at relatively low levels. [9]

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[3] EPA, Health Effects of PCBs (accessed Dec. 16, 2015) [hereinafter "Health Effects of PCBs"] http://www.epa.gov/epawaste/hazard/tsd/pcbs/pubs/effects.htm.

[4] EPA, *Polychlorinated Biphenyls (PCBs)*, CAS Number 1336-36-3, *available at* http://www3.epa.gov/epawaste/hazard/wastemin/minimize/factshts/pcb-fs.pdf.

[5] *See* Health Effects of PCBs.

[6] *See* EPA, Polychlorinated Biphenyls (accessed Dec. 16, 2015) *available at* http://www3.epa.gov/epawaste/hazard/tsd/pcbs/about.htm.

[7] *See* NYSDOH, *Hudson River Fish Advisory Outreach Project Update*, 5 (Sep. 19, 2013), *available at* http://www.hudsoncag.ene.com/files/Hudson%20Fish%20Health%20Advice%20Outreach%20091913.pdf.

[8] *See Id.* at 6, 20; Hudson River PCBs Community Advisory Group, *Hudson CAG Meeting Summary*, 5-6 (Sep. 19, 2013), *available at* http://www.hudsoncag.ene.com/files/Final%20Meeting%20Summary_Sept192013.pdf.

[9] *See* M. Kouznetsova et al., *Increased Rate of Hospitalization for Diabetes and Residential Proximity of Hazardous Waste Sites*, 115(1) Envtl. Health Perspectives 75 (Jan. 2007); Alexander Sergeev & David Carpenter, *Hospitalization Rates for Coronary Heart Disease in Relation to Residence Near Areas Contaminated with Persistent Organic Pollutants and Other Pollutants*, 113(6) Envtl. Health Perspectives 756 (Jun. 2005).

II. EPA Determines that the Removal of PCB-Contaminated Sediments in the Hudson River Is Necessary to Protect Human Health and the Environment

Between 1946 and 1976, GE dumped millions of pounds of PCBs into the Hudson River from two manufacturing plants located in Fort Edward and Hudson Falls, New York. Because of the resulting pollution, EPA declared a nearly 200-mile stretch of the river—from roughly 40 miles north of Albany to the Battery in New York City—a federal Superfund site in 1984. The site was, and remains, one of the largest in the country.

Given its sheer size, EPA divided the Hudson River Superfund Site into separate parts or "operable units" for the purpose of developing a remedial plan for each distinct unit. The focus of this Petition is the remedial plan for Operable Unit 2, which targets contaminated sediments located within the river.[10]

In 2002, EPA issued its Record of Decision ("ROD") for Operable Unit 2 regarding the need and feasibility of action to address contaminated river sediments.[11] In it, EPA concluded that active remediation was "necessary to protect the public health or welfare and the environment" due to the "health hazards associated with human ingestion of [Hudson River] fish, as well as the ecological risks associated with ingestion of fish by birds, fish and mammals."[12]

To address these hazards, the ROD established site-specific remedial action objectives for remediation of in-river sediments ("RAOs"), also setting defined numeric performance targets known as preliminary remediation goals ("Remediation Goals") for acceptable levels of PCBs in fish.[13] The ultimate numeric goal was 0.05 mg/kg of PCBs in fish fillet—a level at which it was expected that an adult could eat a half-pound meal a week safely.[14] The ROD additionally set interim Remediation Goals of 0.2 mg/kg (one meal every month) and 0.4 mg/kg (one meal every two months).[15] For the protection of Hudson River wildlife that also consume fish, such as mink and otter, similar Remediation Goals were established.[16]

In addition to fish-related targets, the RAOs also called for: (1) the reduction of PCB levels in sediment in order to meet the applicable or relevant and appropriate requirements ("Applicable

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[10] Other operable units include: Operable Unit 1 (1984 ROD remedy for Remnant Deposits 2-5); Operable Unit 3 (1999 EPA removal of 4,400 tons of contaminated sediments from Rodger's Island); and Operable Unit 4 (yet to be determined remedy for remediation of floodplains). EPA, *First Five-Year Review Report for Hudson River PCBs Superfund Site*, 1 (Jun. 1, 2012) [hereinafter "FYR"] *available at* http://www3.epa.gov/hudson/pdf/Hudson-River-FYR-6-2012.pdf.

[11] EPA, *Hudson River PCBs Site, New York: Record of Decision* (Feb. 2002) [hereinafter "ROD"] *available at* http://www.epa.gov/hudson/RecordofDecision-text.pdf.

[12] *Id.* at 49.

[13] *Id.* at 50.

[14] *Id.*

[15] *Id.*

[16] *Id.* For river otter, "the piscivorous mammal calculated to be at greatest risk from PCBs at the Site," the risk-based PRG was set at 0.3 to 0.03 mg/kg of PCBs in largemouth bass. For mink, another species known to be sensitive to PCBs, the target range was from 0.7 to 0.07 mg/kg of PCBs in spottail shiner. Other species were considered, but no target ranges were specified as it was determined that they were "at less risk than the river otter." *Id.* The ROD also set a goal of "[r]educ[ing] the inventory (mass) of PCBs in sediments that are or may be bioavailable." *Id.* at 51.

Requirements") for surface water; [17] and (2) the minimization of the long-term flow of PCBs that daily run over the Federal Dam in Troy, NY and down through the Lower Hudson River. The RAOs did not, however, include any targets for air quality because of EPA's finding at the time that "[a]ir exposure was not expected to present a significant risk to human health."[18]

In order to accomplish the RAOs, the ROD evaluated five remedial alternatives—two non-active remedies and three active remedies. The non-active remedies considered were a "no action" alternative and a "monitored natural attenuation" ("Natural Attenuation") alternative, the latter of which assumed some future control of the PCBs then still entering the Hudson ecosystem from the contaminated plant sites. The active remedies proposed capping and/or dredging of contaminated sediments, followed by natural attenuation,[19] but only as applied to the northernmost forty miles of the Superfund site—from the plant sites to the Federal Dam in Troy (the "Upper Hudson River"). The roughly 150 miles of the Hudson Superfund Site below Troy, designated as the "Lower Hudson River," was "not . . . identified for active remediation" on the assumption that active remediation in the Upper Hudson would sufficiently "reduce[] risks to humans and ecological receptors living in and near the Lower Hudson River."[20]

All three active remedial alternatives outlined in the ROD (two calling for dredging and one for capping) divided the Upper Hudson into three distinct sections of unequal length—River Sections 1 and 2 (approximately 10 miles in length combined) and River Section 3 (approximately 30 miles in length)—with varying cleanup standards for each triggered by the amount of "Tri+"[21] PCBs found in surface sediment.[22] The major animating principle behind all three active alternatives was simple: remove or sequester enough PCBs in surface sediments, so the PCBs would no longer get into the water column or the food chain where they would harm people and wildlife.

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[17] For the Hudson River site the federal Applicable Requirements are: 0.5 µg/L total PCBs for drinking water (maximum contaminant level under the Safe Drinking Water Act); 1 ng/L for the Ambient Water Quality Criterion; and 0.014 µg/L for the criteria continuous concentration Federal Water Quality Criterion in freshwater and 0.03 µg/L in saltwater. The New York State Applicable Requirements are: 0.09 µg/L total PCBs for protection of human health and drinking water sources; and 0.12 ng/L for protection of wildlife; 0.001 ng/L for the protection of the health of human consumers of fish. ROD at 50-51.

[18] *Id.* at 26. As explained above, new scientific studies on the potential harms from chronic exposure to lower-chlorinated forms of PCBs undermine this conclusion.

[19] *Id.* at 56-62.

[20] *Id.* at 2.

[21] The remedial alternatives discussed in the ROD target "Tri+" PCBs, defined as PCB molecules with 3 to 10 chlorine atoms, based upon the finding that "that the Tri+ PCB concentration ranged from 98 to 100 percent of the total PCB concentration in fish collected." *Id.* at 24, n. 1. Total PCB levels in the Upper Hudson, however, were roughly 2-4 times higher than the Tri+ levels. *See* Jay Field et al., *Hudson River Remedy: Unremediated PCBs and the Implications for Restoration* (2011) [hereinafter "Unremediated PCBs Trustee Poster"], *available at* http://www.fws.gov/contaminants/restorationplans/HudsonRiver/docs/Battelle1_Field.final1.pdf.

[22] For example, the "REM 3/10/Select" alternative—which EPA ultimately selected—called for the dredging and removal of contaminated sediments: in areas in River Section 1 with a surface concentration of greater than 3 g/m^2 of "Tri+" PCBs; in areas in River Section 2 with a surface concentration more than 10 g/m^2 of Tri+ PCBs; and in select "hot spots" in River Section 3. Similarly, the "CAP 3/10/Select" remedy called for capping of those same sediments respectively, and the "REM 0/0/3" remedy called for removal of contaminated sediments in River Sections 1, 2, and 3 in areas with surface concentrations of Tri+ PCBs of greater than 0 g/m^2, 0 g/m^2, and 3 g/m^2, respectively. *See* ROD at 56-62.

Since consumption of fish was the major exposure pathway of concern, the ROD acknowledged that "[t]he time to reach target PCB concentrations in fish was a *primary factor* in comparing remedial alternatives."[23] Although EPA recognized the limited interim protection provided by longstanding NYSDOH fish consumption advisories,[24] it also found that these "controls do not protect ecological receptors."[25] Further, it found that "human health risk reduction relies on *knowledge of and voluntary compliance with* the consumption advisories and fishing restrictions," having earlier recognized that "fish consumption advisories are not fully protective of human health due to gaps in compliance."[26] Accordingly, expeditious reduction of PCBs in fish was critical to selection of the remedy and in ensuring the protection of human health and the environment.

Indeed, timing was a key factor in EPA's rejection of the non-active alternatives as not sufficiently protective. Relying on computer models designed to predict the short-and-long-term concentrations of PCBs in Hudson River sediment, water, and fish,[27] the agency concluded that the No Action and Natural Attenuation remedial alternatives were "not sufficiently protective of human health and the environment" because: (1) the Natural Attenuation alternative would "*take at least twenty years longer than the selected remedy* to reach target levels in fish tissue in River Sections 1 and 2;" and (2) both non-active alternatives would not sufficiently remedy the "unacceptably elevated" levels of PCBs in the Upper Hudson as well as "the continued degradation of the sediments and surface water quality . . . *for at least several decades longer* than any of the active remedial alternatives."[28]

In contrast, EPA found that all of the active remedial alternatives were "substantially more protective," primarily because of "the shorter time required to reach fish PCB target levels under those alternatives."[29] While the agency found all three active remedies to be sufficiently

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[23] *Id.* at 66 (emphasis added).

[24] The Department of Health advisories caution that all children under 15 and women under 50 should never eat any fish from any section of the river, and that no one should ever eat fish from the Upper Hudson. Men over 15 and women over 50 are advised that they may safely eat some select species of fish in the Mid and Lower Hudson on a occasional basis. NYSDOH, *Hudson River: Health Advice on Eating Fish You Catch*, 6-12 (undated), *available at* https://www.health.ny.gov/publications/2794.pdf.

[25] ROD at 104.

[26] *Id.*; EPA, *Hudson River PCBs Reassessment RI/FS Phase 3 Report: Feasibility Study* (Dec. 2000) (emphasis added), *available at* http://www3.epa.gov/hudson/fs000001.pdf.

[27] ROD at 26. EPA predictions for PCB fish tissue reduction timeframes were the product of a series of interconnected modeling efforts. The "backbone" of these efforts was the Upper Hudson River Toxic Chemical Model ("HUDTOX"), which "forecasted PCB concentrations in water and sediment" in the Upper Hudson River. EPA, *Revised Baseline Modeling Report*, ES-2 (Jan. 2000) *available at* http://www3.epa.gov/hudson/rbmr-bk1&2-chpt1-5.pdf. Outputs from HUDTOX were used as inputs in a number of bioaccumulation models, including the FISHRAND model, which ultimately predicted long-term trends in PCB fish tissue concentrations under the various remedial alternatives. *Id.* at ES-2 to ES-3.

[28] ROD at 102, 108 (emphasis added). It is not explicit in the ROD what years EPA predicted each of the remedies would commence and end. EPA does note, however, that both the CAP 3/10/Select and REM 3/10/Select remedies would take 6 years to complete, and that 2011 would be "the year following the completion of dredging" for the CAP 3/10/Select remedy. ROD at 75, 82. Confusingly, however, the ROD states that all three active remedial alternatives would achieve the 0.4 mg/kg target "within 5 years of completion of dredging (before or by 2013)," ROD at 103, and the FYR notes that "2012 [is] the year after completion of the remedy as simulated by the model." FYR at 28. For the purposes of this petition, it is assumed that EPA predicted 2010 as the year that dredging under the REM 3/10/Select remedy would be completed.

[29] ROD at 104.

protective, it ultimately selected the REM 3/10/Select alternative, which contemplated removal of sediments with PCB surface concentrations of greater than 3 g/m^2 and10 g/m^2 in River Sections 1 and 2, respectively, and select hotspots in River Section 3. EPA anticipated that the REM 3/10/Select Remedy would meet the 0.4 mg/kg target within 2 years of completion of the remedy and the 0.2 target within 14 years.[30] Although EPA predicted that the selected remedy would not meet the final 0.05 mg/kg target within the model timeframe for all three river sections, it did find that sediments in River Section 3 would achieve this goal within 41 years.[31] Importantly, EPA assumed that meeting the 0.05 mg/kg Remedial Goal in River Section 3 would indicate that this goal would likewise "be attained in the majority of the Lower Hudson River, due to [its] lower initial concentration of Site-related PCBs."[32]

III. Post-ROD Sampling Demonstrates that PCB Contamination in the Upper Hudson Is Significantly Greater and More Persistent than Thought

Shortly after issuance of the ROD, EPA conducted the most comprehensive sampling done in the Upper Hudson up until that point as part of the remedial design process, with over 9,000 sediment core samples taken from 2002 to 2005 (the "RD Sampling/Analysis Program"). The results of this sampling program demonstrated that EPA had vastly underestimated the size and persistence of PCB contamination in the Upper Hudson.[33]

Significantly, EPA later determined that PCB surface concentrations in the Upper Hudson were not only "3 times higher than predicted by the [EPA] model," but that the rate of natural attenuation was also much slower.[34] Indeed, while it had earlier "conjectured that the contaminated sediments were 'being buried,'" it later admitted, "the reality is much different."[35] While EPA interpreted these discoveries as "further impetus for the [implementation of the] remedy,"[36] it did not publically consider at the time whether this information would prevent the selected remedy from achieving the RAOs and Remediation Goals identified in the ROD.

In October of 2005, EPA and GE agreed to enter into a consent decree, which was approved by the U.S. District Court for the Northern District of New York in November of 2006, for implementation of the 3/10/Select Remedy ("2006 Consent Decree").[37] The 2006 Consent Decree did not substantially alter the scope of the selected remedy, but it did split it into two phases: Phase I, a small scale dredging pilot followed by a peer-reviewed evaluation ("Phase 1 Evaluation"); and Phase II, full implementation of the remedy.

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[30] *Id.* at 73.
[31] *Id.* at 71.
[32] *Id.* at 103.
[33] *See* Jay Field et al., *Evaluation of Natural Recovery Models for Sediment in the Upper Hudson River* (Feb. 2009), *available at* https://casedocuments.darrp.noaa.gov/northeast/hudson/pdf/Battelle09_Field_NatRecovery_508.pdf.
[34] EPA, *Hudson River PCBs Site EPA Phase 1 Evaluation Report*, ES-18 (Mar. 2010) [hereinafter "Phase 1 Evaluation"] *available at* http://www3.epa.gov/hudson/pdf/2010-03-15_Phase_1_Evaluation_Report_Text.pdf.
[35] *Id.* at I-53.
[36] *Id.* at I-4.
[37] *See U.S. v. Gen. Elec. Co.*, 460 F. Supp. 2d 395, 404 (N.D.N.Y. 2006) [hereinafter "2006 Consent Decree"], *available at* http://www3.epa.gov/hudson/consent_decree/consent_decree.pdf.

Phase I dredging began in 2009, and, consistent with the findings during the RD Sampling/Analysis Program, greater than expected volumes of PCBs were encountered. As part of the Phase 1 Evaluation, conducted in 2010, EPA explained that, because of a greater than expected depth of contamination, the amount dredged in each designated dredging area or "certification unit" was "nearly double the originally planned volume."[38]

While EPA optimistically observed that, as a result, Phase 1 dredging "removed more PCB mass and sediment volume than called for in the ROD,"[39] other observers expressed concern that, likewise, greater-than-expected loads of PCBs would also remain *outside* of the identified dredging areas.[40]

Importantly, the three "natural resources trustees" for the Superfund site—the National Oceanic and Atmospheric Administration ("NOAA"), the U.S. Fish and Wildlife Service ("USFWS"), and the New York Department of Environmental Conservation ("DEC")[41]—commented during the Phase 1 Evaluation that:

> PCB contamination in surface sediment is higher, more widespread, and closer to the surface than anticipated in the ROD. PCBs in the sediments are not being buried and are not declining at the rates predicted. In fact, River Section 2 is as contaminated as River Section 1. However, the cleanup triggers for the surface in River Sections 2 and 3 are approximately 75-90 ppm total PCBs, i.e., three times higher than for River Section 1. The Trustees analysis indicates that average PCB concentration in the top 2 inches of the sediment in River Section 2 and River Section 3 after dredging *will be approximately five times higher than the models predicted.*[42]

Similarly, the final report of the Phase 1 Evaluation peer review panel also heavily criticized EPA's pre-ROD modeling, finding that:

- "[the pre-ROD] models are outdated and inadequate to accurately project [Natural Attenuation] and post-dredge fish recovery rates.
- Neither EPA nor GE has sufficient data or a credible tool to project recovery."[43]

Accordingly, the peer review panel emphasized the importance of conducting new modeling "designed to predict surface sediment concentrations, fish PCB uptake, and long-term recovery

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[38] Phase 1 Evaluation at ES-4.
[39] *Id.* at II-3.
[40] Indeed even EPA noted that in areas capped during Phase 1 that "sediments were left behind that contained more PCBs than permitted by the ROD or the Residuals Standard in CU's 1, 2, 4, 5, 6, 7, and 8." *Id.* at II-58.
[41] NOAA, USFWS, and DEC are acting on behalf of the Department of Commerce, the Department of Interior, and the State of New York respectively. The trustees are responsible for calculating GE's outstanding natural resources damages for the site—a distinct basis of liability under CERCLA.
[42] NOAA, USFWS, & DEC, *Trustee Comments on Phase 1 Evaluation Reports for the Hudson River* (Apr. 26, 2010) (emphasis added) [hereinafter "Trustee Phase 1 Comments"], *available at* https://casedocuments.darrp.noaa.gov/northeast/hudson/pdf/Hudson_trustee_letter_re._Phase%201_Evaluation.pdf.
[43] Todd Bridges et al., *Hudson River PCBs Site: Peer Review of Phase 1 Dredging - Final Report*, 13 (Sep. 10, 2010), *available at* http://www3.epa.gov/hudson/pdf/hudsonriverphase1dredgingreport_final.pdf.

for the entire river," also recommending that the results of this modeling be made available for peer review.[44] This call for updated and more accurate modeling was also echoed by NOAA[45] and some of the Petitioners.[46]

In response, EPA "agree[d] that a new model with strong predictive capabilities," would be helpful for "adaptively managing the project to a successful conclusion."[47] But it did not agree to conduct additional modeling by itself. Instead, it noted that GE had developed its own computer model that "may be a useful foundation for this effort," promising "to complete a detailed, thorough evaluation of the model," but without allowing time for peer review.[48] Although this work was supposed to take "6-9 months,"[49] EPA has never publicly released the results of this effort, to the extent that it was actually undertaken.

Several months later, Dr. Robert Haddad, Chief of the NOAA Assessment and Restoration Division, wrote to EPA, warning that "the impacts of maintaining the current course of action is clear and troubling to NOAA."[50] Pointedly, Dr. Haddad stated that the implementation of the remedy, as planned, would leave the equivalent to "[a] series of Superfund-caliber sites" in the Upper Hudson, thereby frustrating restoration and recovery efforts and "result[ing] in the high likelihood of remediated areas becoming recontaminated."[51] In order to "achieve the original risk-based goals of the ROD," the letter urged EPA to "apply[] River Section 1 surface criteria to River Sections 2 and 3."[52]

Despite these concerns, EPA refused to alter the basic scope of the dredging required under the selected remedy, and Phase 2 dredging began in 2011.

IV. The Initial Five Year Review Was Inadequate

In 2012, EPA performed a statutorily mandated five-year review to "ensure that implemented remedies [at the Hudson River Superfund Site] protect public health and the environment and . . . function as intended by the Site decision documents" (the "Five-Year Review").[53] The review process provided EPA with a clear opportunity to address longstanding concerns regarding the

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[44] *Id.* at 37.

[45] *See* Jay Field, *Comments to the Hudson River Engineering Performance Standards Peer Review Panel* (May 5, 2010), *available at* https://casedocuments.darrp.noaa.gov/northeast/hudson/pdf/HRPeerRev_Comments_JField_20100505.pdf.

[46] *See* Letter from Hudson River Sloop Clearwater, Natural Resources Defense Council, Riverkeeper, Scenic Hudson, to Judith Enck, EPA (May 4, 2012) *available at* http://www3.epa.gov/hudson/pdf/CorrespondenceReceived_FiveYearReview_HudsonRiverPCBs.pdf.

[47] Letter from Walter Mugdan, EPA, to Dr. Stephen Garon, SRA International, entitled "EPA Response to Draft Hudson River EPS Peer Review Report" (Aug. 27, 2010), *available at* http://www3.epa.gov/hudson/pdf/EPA_Comments8-27-2010.pdf.

[48] *Id.*

[49] *Id.*

[50] Letter from Dr. Robert Haddad, NOAA, to Robert Sussman, EPA, entitled "Phase 2 Remediation, Hudson River PCB Superfund Site" (Dec. 2, 2010) [hereinafter "Haddad Letter"], *available at* http://www3.epa.gov/hudson/pdf/CorrespondenceReceived_FiveYearReview_HudsonRiverPCBs.pdf.

[51] *Id.*

[52] *Id.*; *see also* Unremediated PCBs Trustee Poster. This would result in the removal of approximately 136 additional acres of highly contaminated sediment from the Hudson River. *Id.*

[53] FYR at 1; *see also* 42 U.S.C. § 9621(c).

impact of greater-than-expected PCB volumes on remedial effectiveness by performing its own detailed analysis.

The review, however, was cursory at best—completed within a mere 60 days of the announcement it would be conducted, a timeframe which also included the public comment period.[54] Significantly, the Five-Year Review did not include updated computer modeling analyzing the impact of the large volumes of PCBs discovered post-ROD. Instead, it attempted to roughly estimate the future effects of the planned dredging on fish tissue using the RD Sampling/Analysis Program data, under the apparent assumption that the removal of "concentrations of [PCBs] in the surface sediments" would have a proportional effect on the "[PCB] reduction in fish body burden."[55]

Using the expected *percentage* reduction of PCBs—rather than analyzing the *total* amount of PCBs that would be left in surface sediments as compared to the ROD predictions—EPA estimated that while in River Section 2 it would take about 10 years longer to reach fish tissue targets, in River Sections 1 and 3, the remedy would actually perform "better than previously anticipated (or at least comparabl[y])."[56] Based upon these findings, among others, EPA concluded that the 3/10/Select Remedy would still "be protective of human health and the environment" upon completion.[57]

V. New Computer Modeling Analysis by NOAA Concludes that the EPA Sediment Remedy Will Fail to Achieve the Health and Safety Targets Established in the ROD

Although it is Petitioners' understanding that EPA has not performed any new computer modeling since issuing the ROD, new modeling analysis was performed by NOAA and released earlier this year. This model assesses the impact of the greater-than-expected volumes of PCBs discovered post-ROD on remedial effectiveness, and its findings critically undercut EPA's conclusion that the selected remedy, as designed, will meet the health and safety targets outlined in the ROD.[58] It is Petitioners' understanding that this analysis is currently undergoing peer review.

In essence, the new analysis uses a computer model that emulates EPA's pre-ROD modeling, but unlike that earlier effort, the new "model emulation" includes updated data from the RD Sampling Analysis/Program. Based on that data, NOAA calculated that EPA had likely overestimated the rate of natural recovery by a factor of 6—with the EPA pre-ROD modeling

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[54] *See* EPA, Notice of "U.S. Environmental Protection Agency Conducting First Five-Year Review of Implemented Actions at the Hudson River PCBs Superfund Site" (2012) *available at* http://www3.epa.gov/hudson/pdf/adhudsonriver_fyrnotice2012.pdf.

[55] FYR at 27-28.

[56] FYR at 33.

[57] FYR at iii.

[58] Jay Field et al., *Revisiting Model Projections of Lower Hudson River Fish PCBs Using Model Emulation and Recent Data*, 9 (Aug. 20, 2015) [hereinafter "NOAA Analysis"] *available at* https://casedocuments.darrp.noaa.gov/northeast/hudson/pdf/CSF2015_AUG20_LHR_Fish_final_dist.pdf. A presentation explaining the new NOAA Analysis is available here.

estimate at 8%, and the post-ROD actual observed rate at 1.3%.[59] Using a conservative 3% decay rate and the updated sediment sampling information, the model emulation predicts that:

- post-remedial PCB concentrations in the Upper Hudson River sediments will exceed previous EPA model predictions *by a factor of 3-to-5 times*; and

- achieving the Remediation Goals for PCB fish tissue concentrations in the Lower Hudson River would take *several decades longer* than expected.[60]

For example, while EPA predicted white perch just below the Troy Dam would achieve the 0.4 mg/kg PCB target almost immediately after completion of the remedy, the NOAA analysis predicts that this target will likely not be met for *another 44 years*.[61] Similarly, the time to achieve the 0.2 mg/kg PCB target would take *another 67 years*.[62]

Ultimately, the NOAA analysis concludes that, because EPA was unaware of the true extent of contamination when conducting its pre-ROD computer modeling, "[a]ttainment of EPA's Remedial RAOs for fish in the [Lower Hudson] will take longer than predicted" and that "[a]dditional removal of PCB-contaminated sediment in the [Upper Hudson] [is] needed to achieve reductions in [Lower Hudson] fish PCBs anticipated in the ROD."[63]

VI. EPA Allows Closure of GE's Remedial Activities Despite New Evidence that the Remedy Will Not Be Protective of Human Health and the Environment

In advance of the completion of the final season of dredging under the REM 3/10/Select Remedy, GE presented EPA with a plan for decommissioning of the dewatering facility used for processing of contaminated sediments during dredging.[64] EPA released the plan for public comment, initially giving the public a mere two weeks to comment, during which Petitioners commented that EPA's consideration of a plan to dismantle cleanup infrastructure was premature given the unanswered concerns regarding the adequacy of the remedy—specifically, those raised by the RD Sampling/Analysis Program data, new NOAA analysis, and new information about the harmfulness of volatilized PCBs that was not previously considered by EPA.[65]

Likewise, the federal trustees also submitted comments, recommending that, because of their "overarching concern about the protectiveness of the remedy, the extended time it will take our trust resources [in the Hudson] to recover, as well as the impacts demobilization might have on

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[59] *Id.* at 10.
[60] *See id.* at 9, 31, 36.
[61] *Id.* at 31.
[62] *Id.*
[63] *Id.* at 36.
[64] *See* GE, *Phase 2 Sediment Processing Facility Demobilization and Restoration Plan: Hudson PCBs Superfund Site* (Sep. 2015), *available at* http://www3.epa.gov/hudson/pdf/ph2_hr_spf_demob_and_restorationplan_text_tables_figures091115.pdf.
[65] *See* Hudson River Sloop Clearwater et al., *Public Comments on GE's Draft Phase 2 Sediment Processing Facility Demobilization and Restoration Plan* (Sep. 28, 2015), *available at* https://d3n8a8pro7vhmx.cloudfront.net/campaignforacleanerhudson/pages/26/attachments/original/1443621485/Coalition_Comments_on_Phase_II_Demobilization_and_Restoration_Plan_9.28.15.pdf?1443621485.

restoration opportunities," EPA "postpone action on the demobilization plan until a new Five-Year Review is conducted to ensure that the remedy is protective of human health and the environment."[66]

On October 1, 2015, EPA held what it explained was its final Community Advisory Group meeting addressing the dredging component of the REM 3/10/Select Remedy. At that meeting, several of Petitioners raised concerns about the adequacy of the selected remedy earlier-raised in comments and elsewhere, including concerns that the longstanding state fish consumption advisories are not adequately functioning to protect human health. In particular, Petitioners highlighted the new NOAA analysis, also asking EPA whether it had a current estimate of whether and when the Remedial Goals would be met.[67] EPA responded that no such estimate was possible unless and until it: (1) conducted additional computer modeling incorporating post-ROD data; or (2) reviewed 5-7 years of fish data after closure of the remedy to ascertain, after the fact, whether or not dredging had been a success. EPA also stated that additional computer modeling before certification of completion of the remedy would likely be infeasible, strongly indicating that it would take no significant new actions to assess the efficacy of the remedy before certifying the remedy as complete.

However, without regard to its apparent inability to predict remedial efficacy, and despite the serious concerns raised by multiple parties, EPA issued a written statement on the same day stating "[t]he Hudson River PCB Superfund dredging project has been a success."[68] The statement also criticized the NOAA analysis for relying on "old" data—namely, the RD Sampling/Analysis Program results earlier used by EPA to support its conclusions in the Five-

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[66] NOAA and USFWS, *Comments on the Phase 2 Sediment Processing Facility Demobilization and Restoration Plan Hudson River PCBs Superfund Site, Revised September 2015*, 1, 2 (Sep. 28 ,2015), *available at* http://www.fws.gov/contaminants/restorationplans/hudsonriver/docs/Hudson%20River%20Fed%20Trustee%20Comments%2009282015_Final%20signed.pdf.

[67] Additionally, Petitioners raised concerns regarding GE's departure from the required sampling protocol that it used in fish tissue sampling for roughly a 10-year period (from 2004 to 2014) and its possible effects on EPA's conclusions during the Five-Year Review. The Federal Trustees also raised these concerns in their comments on the dewatering facility demobilization plan, stating that:

> "[t]he change in protocol requires a thorough analysis and report out of conclusions from that study . . . Comparison studies between rib-in and rib-out fillet method in brown bullhead, yellow perch, white perch and striped bass are also necessary to understand the impacts the change in the processing protocol had on wet weight and lipid normalized PCBs for these four species, which are analyzed as part of the baseline monitoring and remedial action monitoring program to determine remedy effectiveness.
>
> This information is critical for updating Tables 3 and 4 (wet and lipid normalized fish PCBs) of the [Five-Year Review] and for EPA to reassess remedial protectiveness. On the basis of new information about the higher pre-remedial concentrations, decreased rate of natural recovery in Hudson River sediments, measured concentrations of PCBs in white perch supporting the lower decay rate, decades of delay in achieving RAO fish objectives, and the issues surrounding changes in fish filleting protocol, the Federal Trustees believe such a review is justified, and that until it is completed, any action on the [demobilization] [p]lan must be put on hold."

Id. at 4.

[68] EPA, *Statement From EPA on Hudson River Cleanup* (Oct. 1, 2015), *available at* http://www3.epa.gov/hudson/pdf/statement_hudson_october_1_final.pdf.

Year Review. On October 5, 2015, GE announced that it had completed dredging in the Hudson River pursuant to the REM 3/10/Select Remedy, also describing the project as a success.[69]

On November 12, 2015, without addressing the concerns of Petitioners or the federal trustees, EPA approved GE's plans to dismantle the dewatering facility. This action effectively constituted approval of the completion of GE's dredging operations and confirmed the agency's conclusion in its October 1, 2015, statement that the dredging project has been a success.[70]

ARGUMENT

I. EPA Has Failed to Ensure that the Selected Sediment Remedy Is Protective of Human Health and the Environment

A. EPA Has a Duty to Ensure that the Remedy Is, and Remains, Protective of Human Health and the Environment

CERCLA requires EPA to respond to the threat of toxic pollution where it may endanger human health and the environment. Where EPA determines that a hazardous substance at a Superfund site "may present an imminent and substantial danger to the public health and welfare," it must "select appropriate remedial actions" that it "deems necessary to protect the public health or welfare or the environment."[71]

In order to identify and implement "remedies that are protective of human health and the environment," CERCLA requires that EPA establish site-specific remedial action objectives, including concrete and quantifiable remediation goals.[72] All remedial actions selected by the agency must "attain a degree of cleanup . . . which assures protection of human health and the environment,"[73] and the success or failure of a remedy under this standard is measured by its ability to actually achieve the action objectives and the remediation goals.[74]

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[69] *See* GE, *GE Completes Hudson River Dredging* (Oct. 5, 2015), http://www.hudsondredging.com/2015/10/05/ge-completes-hudson-river-dredging/.

[70] *See* EPA, *EPA Statement on Approval of PCB Processing Facility Demobilization and Restoration Plan for Hudson River Cleanup* (Nov.12, 2015), *available at* http://www3.epa.gov/hudson/pdf/DemobPlan_ApprovalStatement_Final.pdf.

[71] 42 U.S.C. §§ 9604(a)(1), (c)(4), 9621(a), (b)(1). EPA may also select a removal action or take other response measures that it deems appropriate. 42 U.S.C. § 9604(a)(1), (2). As EPA found remedial action to be necessary here, however, this petition will focus on EPA's remedial obligations.

[72] *See* 40 C.F.R. § 300.430(a)(1)(i), (e)(2)(i); 42 U.S.C. § 9621(b)(1).

[73] 42 U.S.C. § 9621(d)(1).

[74] *See* 42 U.S.C. § 9621(c), (d)(1); EPA, *Interim Guidance for Evaluation of Federal Agency Demonstrations that Remedial Actions are Operating Properly and Successfully Under CERCLA Section 120(h)(3)*, (Aug. 1996), http://www2.epa.gov/fedfac/guidance-evaluation-federal-agency-demonstrations-remedial-actions-are-operating-properly-and#intro ("completion of a remedial action is defined by the attainment of specific cleanup levels or performance goals that are specified in a decision document, such as a Record of Decision"); *see also, e.g.*, U.S. Dep't of Energy, *Guide to Ground Water Remediation at CERCLA Response Action and RCRA Corrective Action Sites*, 7-10 (Oct. 1995), *available at* http://homer.ornl.gov/sesa/environment/guidance/gw/grndh2o.pdf ("The suitability and performance of any completed or ongoing ground water remedial action should be evaluated with respect to the objectives of those actions (e.g., . . . attainment of cleanup levels)").

These objectives and goals, however, do not become permanently fixed upon issuance of a ROD. Indeed, even after a ROD is finalized, EPA has a duty to consider significant new information and analysis that substantially supports the need to alter a response action,[75] and to take appropriate additional action where necessary to ensure its protectiveness.[76] In cases where EPA "selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site"—as is the case with the Hudson River PCB Superfund Site—CERCLA requires EPA to conduct review of the remedial action every five years (or sooner) in order to evaluate the protectiveness of the remedy as implemented.[77] Because it is EPA's responsibility to ensure that the remedy is protective,[78] where the evaluation shows that the action objectives and/or remedial goals may not be met, EPA must determine what additional review or action is needed.[79] Where the "review shows that a remedy is no longer protective of human health and the environment," EPA must ensure that "additional action [is]evaluated *and taken* to mitigate the threat."[80]

In the present case, the threat posed by GE's PCBs in the Hudson River to the health of New Yorkers and the state's environment is unambiguous. As EPA concluded in the ROD, the significant health and ecological risks associated with the ingestion of PCB-laden fish made active remediation "necessary to protect the public health or welfare and the environment."[81]

To eliminate this threat, EPA developed specific RAOs and Remediation Goals to be achieved by the selected alternative—the REM 3/10/Select Remedy. The selection of the REM 3/10/Select Remedy was premised on its ability to achieve these criteria within a reasonably prompt timeframe.[82] It was the ability to meet these targets within that timeframe that defined the adequacy of the REM 3/10/Select Remedy, and it was and is EPA's duty to ensure that they are met in order to ensure protection of human health and the environment.

B. EPA Has Failed to Ensure the Protectiveness of the REM 3/10/Select Remedy

EPA has chosen to move forward with the closure of the REM 3/10/Select Remedy despite the clear implications of its own post-ROD sediment sampling data and years of repeated warnings by other state and federal agencies that the remedy, as designed, will fail. Under these circumstances, the agency has—time and again—acted arbitrarily and capriciously, and acted contrary to law, by failing to ensure the protectiveness of the remedy as demanded by CERCLA.

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[75] *See* 40 C.F.R. § 300.825(c).

[76] *See* Proposed Rule for National Oil and Hazardous Substances Pollution Contingency Plan, 53 FR 51394, 51430; EPA, *Comprehensive Five-Year Review Guidance*, OSWER Directive 9355.7-03B-P, 4-11 (Jun. 2001) [hereinafter "FYR Guidance"] *available at* http://semspub.epa.gov/work/11/128607.pdf.

[77] 42 U.S.C. § 9621(c).

[78] *See* EPA, *Recommended Evaluation of Institutional Controls: Supplement to the 'Comprehensive Five Year Review Guidance'*, http://www.progressivereform.org/articles/Institutional_Controls_Guidance_091311.pdf ("EPA . . . is legally responsible for making the protectiveness determination during the [Five Year Review]").

[79] FYR Guidance at 4-9, 4-12.

[80] 53 FR 51394, 51430 (emphasis added); *See also id.* at 4-11 ("Follow-up actions should be completed to ensure long-term protectiveness of the remedy, or to bring about protectiveness of a remedy that is currently not protective.").

[81] ROD at 49.

[82] *See* ROD at 102-05.

Almost immediately after issuance of the ROD, sediment sampling conducted during the Sampling Analysis/Program demonstrated the equivalent of a bombshell—namely, that the extent of PCB contamination in the Upper Hudson was *2-3 times* larger and significantly more persistent in surface sediments than ever anticipated. Indeed, by EPA's own admission during the Phase 1 Evaluation, despite earlier agency assumptions regarding natural sequestration of contaminated sediments, "the reality [wa]s much different."[83]

While the implications of this new "reality" on the adequacy of the remedial plan should have been duly evaluated at the earliest possible juncture, they were not. Moreover, EPA persisted in burying its head in the sand despite years of collecting commentary and analysis spelling out the concerns and consequences raised by these post-ROD-discovered PCBs. These observations came not only from the public and interested environmental organizations, such as Petitioners, but from EPA's own Phase 1 Evaluation peer review panel and the three state and federal agencies that have been studying the effects of PCBs on the Hudson River's ecosystem for the past 15 years in their role as the site's "trustees." Further, concerns have been raised at every major step in the remedial process—from the Phase 1 Evaluation to the Five-Year Review to comments on the recent plan for decommissioning of critical cleanup infrastructure.

The thrust of these concerns has been most plainly and emphatically outlined by the two federal trustees—NOAA and USFWS. Both during and after the Phase 1 Evaluation, they sounded alarms about the bulk of PCBs slated to be left in the river, characterizing them as "equivalent to a series of Superfund-caliber sites."[84] Of paramount concern was the trustees' estimate that remedial surface concentrations of PCBs would be "approximately five times higher than [pre-ROD] models predicted" [85] because it is primarily through surface sediments that PCBs migrate into water and wildlife, thereby heightening the exposure risk to people. These concerns were so pointed that, in an unusual move, the Chief of NOAA's Assessment & Restoration Division wrote to EPA warning that EPA's "current course of action is clear and troubling" and urging additional dredging in order "to achieve the original risk-based goals of the ROD."[86]

EPA has never indicated an intention to expand the scope of dredging in response to these significant concerns, and further, as far as the public is aware, it has never seriously attempted to perform its own thorough analysis to either verify or refute them. In 2010, EPA promised to investigate the implications of the post-ROD-discovered contamination by examining computer modeling then being conducted by GE, but this effort was apparently later abandoned without explanation. In 2012, EPA missed another opportunity for earnest assessment, eschewing additional modeling or other detailed analysis, and instead relying on an apparent back-of-the-envelope calculation to support its conclusion that nothing was seriously wrong with its remedy.

That thinly supported conclusion is now directly and convincingly challenged by the detailed modeling analysis of its sister agency, NOAA. To Petitioner's understanding, the NOAA analysis represents the only truly thoroughgoing attempt since the ROD to assess the impact of the vast amounts of PCBs discovered after its issuance on remedial effectiveness, and,

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[83] Phase 1 Review at I-53.
[84] *See* Unremediated PCBs Trustee Poster; Haddad Letter.
[85] Trustee Phase 1 Comments.
[86] Haddad Letter.

unsurprisingly, it concludes the obvious. Specifically, it finds that the 3/10/Select Remedy will not achieve the RAOs or Remediation Goals for the Lower Hudson—confirming that post-remedial surface concentrations of PCBs will be *3-5 times higher* and demonstrating that fish tissue targets will take *several generations longer* than EPA anticipated in the ROD. Ultimately, the NOAA analysis concluded that "[a]ttainment of EPA's Remedial RAOs for fish in the [Lower Hudson] will take longer than predicted" and that "*[a]dditional removal of PCB-contaminated sediment in the [Upper Hudson] [is] needed to achieve reductions in [Lower Hudson] fish PCBs anticipated in the ROD.*"[87]

This conclusion originates from a leading federal scientific agency and is endorsed by USFWS. Not only are these the two federal agencies that have the greatest general expertise in fisheries matters, they also have been conducting detailed study of the effects of PCBs specifically on the Hudson River ecosystem for well over a decade. As such, the conclusions of the NOAA analysis could well have been taken as definitive on the issue of the adequacy—or rather, inadequacy—of the 3/10/Select Remedy. At the very least, they should have led EPA to revisit, in detail and in depth, its operating assumption that all was well with the Hudson and no further dredging is needed. Instead, EPA criticizes the data used by NOAA as "old" in a brief press statement, without any apparent intention to publicly clarify or support its thinking, and seemingly ignoring the fact that this "old" data is the same that undergirds its conclusions in the 2012 Five-Year Review.

Accordingly, in light of the conclusions of the NOAA analysis—and the years of concerns raised by other state and federal agencies and its own Phase 1 peer review panel—EPA's de facto determination regarding the sufficiency of the REM 3/10/Select without further adequate review is arbitrary and capricious and in violation of its obligation to ensure the protectiveness of selected remedial actions.

II. EPA Must Immediately Undertake a Thorough and Adequate Review of the Protectiveness of the REM 3/10/Select Remedy

EPA must immediately undertake an in-depth evaluation of the protectiveness of the REM 3/10/Select Remedy, including an objective evaluation of the NOAA analysis and the opportunity for full participation by interested agencies, public comment, and peer review. Further, if that review confirms, as the Trustees have asserted, that the REM 3/10 Select Remedy will not meet the RAOs or Remedial Goals within the relevant timeframes, EPA *must* take appropriate action by expanding the scope of the remedial action to require further PCB removal.[88]

These actions must be done immediately and not deferred until the next scheduled five-year review on April 23, 2017.[89] As EPA's own guidance provides "[f]ive-year reviews may be conducted earlier or more frequently than every five years, if needed, to ensure the protection of

[87] NOAA Analysis at 36 (emphasis added).
[88] *See* 42 U.S.C. § 9621(d)(1) (EPA must ensure degree of cleanup that, at minimum, "assures protection of human health and the environment").
[89] FYR at 40.

human health and the environment."[90] That is certainly the case here, and as EPA has signaled, it is now considering undertaking such a review. This review may now provide EPA with the opportunity to discharge its responsibility to ensure remedial protectiveness.

Performance of a five-year review as a pro-forma exercise, however, is not sufficient. In order to adequately discharge its duty to ensure that the REM 3/10/Select Remedy is protective of human health and the environment, EPA must do what it has not done for the past 13 years—namely, adequately and thoroughly analyze the mounting evidence that its remedy, as designed, fails.

More specifically, any review now must do what EPA's own five-year review guidance provides: it must analyze all information that has "come to light that could call into question the protectiveness of the [REM 3/10/Select] [R]emedy" and ascertain whether "the remedy is functioning as intended."[91] Given the circumstances and history of this case, however, an effective review must also include close collaboration with EPA's sister agencies (including NOAA and USFWS), be exceptionally transparent, and also include ample opportunity for participation from members of the public (such as Petitioners) and peer review.

Further, EPA must consider *all* relevant new information including, but not limited to, the NOAA model emulation and conclusions, the potential harms presented by continued exposure to low levels of lower-chlorinated forms of volatilized or airborne PCBs, and the failures of longstanding fish consumption advisories to protect human health.

Importantly, satisfactory review must be coupled with swift and appropriate action, including expansion of the REM 3/10/Select Remedy if it is determined that the remedy will not timely meet ROD goals.[92] Indeed, Petitioners underscore that action now—before certification of remedial completion—is critical given the terms of the 2006 Consent Decree. It is Petitioners' understanding that under that document, the agency's covenants not to sue GE for additional administrative or injunctive-like relief in the Upper Hudson in areas *outside* of the remedial areas designated for dredging is not triggered *until certification.*[93] Thus, EPA may have much greater latitude to act *now* in discharging its duty to protect human health and the environment than if it waits to address the issue after certification. In short, EPA must fully discharge its obligations under CERCLA before it binds its own hands.

In the meantime, EPA should direct GE to halt any further demobilization or restoration activities with respect to the dewatering and related facilities until it has the opportunity to conduct a full and adequate review.

CONCLUSION

For the reasons set forth above, EPA's de facto determination regarding the sufficiency of the REM 3/10/Select Remedy is arbitrary and capricious and in violation of the agency's duty to ensure the protectiveness of selected remedies. EPA must take immediate corrective action by adequately evaluating the sufficiency of its REM 3/10/Select Remedy in a thorough,

[90] FYR Guidance at 1-4.
[91] *Id.* at 4-1.
[92] *See id.* at 4-12.
[93] *See* 2006 Consent Decree at ¶¶ 98(f), 99(b).

transparent, and inclusive review that includes all relevant new information, and, upon such evaluation, require continued dredging by GE to remove additional PCB-contaminated sediments as necessary to adequately protect human health and the environment. Further, EPA must not certify completion of the remedy until this evaluation has been conducted and the agency assures the protectiveness of the REM 3/10/Select Remedy.

Petitioners further respectfully request that EPA respond to this Petition within 90 days of its receipt by the agency. If no response is received within 90 days, the Petitioners will take that as a denial of the Petition, and reserve all rights to take any available and appropriate action in response.

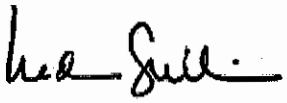
Dated: December 17, 2015

Respectfully submitted,

Daniel Raichel
Staff Attorney
NRDC

Peter Gross
Executive Director
Hudson River Sloop Clearwater

Paul Gallay
President and Hudson Riverkeeper
Riverkeeper



Ned Sullivan
President
Scenic Hudson

Roger Downs
Conservation Director
Sierra Club, Atlantic Chapter

cc:
Gina McCarthy, EPA Administrator
Mathy Stanislaus, Assistant Administrator for EPA's Office of Solid Waste and Emergency Response

EXHIBIT A

Petitioner **Hudson River Sloop Clearwater, Inc.** ("Clearwater") is a 501[c][3] not-for-profit corporation with its offices located at 724 Wolcott Ave Beacon, NY 12508. Clearwater is a member-supported environmental education organization with approximately 5,000 active members, many of whom reside in the Hudson Valley. Clearwater operates the Hudson River Sloop known as "Clearwater," through which it provides environmental education and experience for tens of thousands of New Yorkers. The organization is dedicated to, among other things, protecting and cleaning up the Hudson River and educating the public, including young people, about the unique environmental resources of the River and the Hudson River Valley. Clearwater was instrumental in supporting the passage of the Clean Water Act, and over the years, along with the other petitioners, has led the public effort to clear the Hudson of PCB contamination and restore the environmental and economic health of the River. Clearwater's members regularly enjoy the Hudson River.

Petitioner **Natural Resources Defense Council, Inc.** ("NRDC") is an international, non-profit membership organization headquartered in New York, and committed to the preservation, protection, and defense of the environment, public health, and natural resources. With over 30,000 members in New York State, NRDC has been active since its founding in 1970 on environmental and land use issues affecting New York's local communities—including watershed protection, brownfields redevelopment, and smart growth and zoning. NRDC has also been a key advocate for the cleanup of PCBs from the Hudson River for more than four decades. Thousands of NRDC members live near the Hudson River and thousands more regularly visit, work, and play along it.

Petitioner **Riverkeeper, Inc.** ("Riverkeeper") is a 501(c)(3) not-for-profit corporation with its offices located at 20 Secor Road, Ossining, New York 10562. Riverkeeper is a member-supported watchdog environmental organization with approximately 4,000 active members, many of whom reside in the Hudson Valley. Riverkeeper is dedicated to, among other things, defending the Hudson River and its tributaries, protecting and restoring the unique environmental resources of the Hudson River and the Hudson River Valley, and fostering proper management of such environmental and natural resources. Over the years, Riverkeeper, along with the other Petitioners, has led the public effort to clear the Hudson of PCB contamination and restore the environmental and economic health of the River. Riverkeeper's members regularly enjoy the Hudson River for various recreational, educational, and other such purposes and can be expected to continue to use the river for such purposes in the future.

Petitioner **Scenic Hudson, Inc.** is a regional, non-profit membership organization headquartered in Poughkeepsie, New York, and committed to the preservation, protection, and defense of the environment, public health, and economic sustainability of the Hudson River Valley. Scenic Hudson works to protect and restore the Hudson River as an irreplaceable national treasure and a vital resource for residents and visitors. A crusader for the Valley since 1963, today it is the largest environmental group focused on the Hudson River Valley. Scenic Hudson combines land acquisition, support for agriculture, citizen-based advocacy and sophisticated planning tools to create environmentally healthy communities, champion smart economic growth, open up riverfronts to the public and preserve the valley's inspiring beauty and natural resources. To date Scenic Hudson has created or enhanced more than 65 parks, preserves and historic sites up and down the Hudson River and conserved over 35,000 acres. Scenic Hudson has over 25,000 supporting members, most of whom reside in the counties

located along the Hudson River. Scenic Hudson supporting members are regular users of the Hudson River for boating and other recreational activities.

Petitioner **Sierra Club Atlantic Chapter** is a 501[c][3] not-for-profit corporation with its offices located at 353 Hamilton Street Albany, NY 12210. The Atlantic Chapter is a volunteer led environmental organization of 40,000 members statewide dedicated to protecting New York's air, water and remaining wild places. Sierra Club members have been active for decades in advocating for the cleanup and restoration of the Hudson River—including the organizing of "fishing for Justice" programs that have educated communities of color and subsistence fishermen about the river's ecology and the risks that come from eating PCB contaminated fish.

Attachment I

Letter from Judith Enck to Petitioners
Mar. 16, 2016

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
290 BROADWAY
NEW YORK, NY 10007-1866

MAR 16 2016

Mr. Roger Downs
Conservation Director
Sierra Club Atlantic Chapter
353 Hamilton Street
Albany, NY 12210

Mr. Paul Gallay
President
Riverkeeper
20 Secor Road
Ossining, New York 10562

Mr. Dave Conover
Interim Executive Director
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724 Wolcott Ave
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Mr. Dan Raichel
Staff Attorney
Natural Resources Defense Council
40 West 20th Street
New York, NY 10011

Mr. Ned Sullivan
President
Scenic Hudson
One Civic Center Plaza, Suite 200
Poughkeepsie, NY 12601

Dear Mr. Downs, Mr. Gallay, Mr. Conover, Ms. Moran, Mr. Raichel and Mr. Sullivan:

This letter is in response to the December 17, 2015, *Petition to the United States Environmental Protection Agency* from five of your organizations which requests, among other things, that the U.S. Environmental Protection Agency immediately begin a second Five Year Review (the "Second Five Year Review") pursuant to Section 121(c) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended ("CERCLA"), 42 U.S.C. § 9621(c), addressing the protectiveness of the remedial action for the Hudson River PCBs Site. The Petition further requests that EPA require General Electric Company to perform additional dredging if, as a result of that review, EPA determines that the remedy is not protective. In addition to responding to the Petition, this letter also responds to your January 22, 2016, letter to Assistant Administrator Stanislaus and me, and to the February 23, 2016, correspondence to me from four of your organizations regarding the Second Five Year Review. Previously, on December 18, 2015, Assistant Administrator Stanislaus and I responded to issues that were raised by five of your organizations in a December 10, 2015, letter to EPA, and that were similar to the concerns raised in the Petition and the January 22 letter.

Our responses to issues raised in the Petition and your recent correspondence are as follows:

Schedule for the Second Five Year Review

Both the Petition and your January 22 letter request that EPA "immediately" begin conducting the Second Five Year Review, with your January 22 letter suggesting that the review preferably should begin no later than early February 2016. In our February 22, 2016, letter, we informed you that EPA did in fact initiate the Second Five Year Review in February when it internally commenced activities that are part of the review. Such activities included the initial scoping of the fish, sediment and water data collection to be performed in 2016. In addition, on February 24, 2016, EPA held a workshop meeting with the Community Advisory Group ("CAG") to discuss the scope of the Operation, Maintenance and Monitoring ("OM&M") program for the Site, which will include the collection of fish, sediment and water column data. Attendees of the workshop included representatives of the federal natural resource trustees, New York State, and many of your organizations.

EPA plans to hold a public workshop with the CAG in late April of 2016 to discuss the Second Five Year Review. In addition, EPA anticipates finalizing the OM&M plan in April or May of this year. Sediment, water and fish samples for the Second Five Year Review will be collected and analyzed beginning in the spring and continuing over the summer, although the schedule for the Second Five Year Review will not allow for the inclusion of data from samples collected after the summer of 2016. Our goal remains to issue the Second Five Year Review report for public comment in late 2016 or early 2017, prior to the April 23, 2017, due date for the report. We look forward to receiving additional input from the CAG, including your groups, regarding the Second Five Year Review, and also to closely coordinating on the review with New York State and the federal trustees.

Certification of Completion of the Remedial Action

Both the Petition and your January 22 letter strongly urge EPA not to certify GE's completion of the remedial action until after EPA completes the Second Five Year Review. Please note that the government will follow the requirements of the consent decree between the United States and GE with respect to certifying completion of the remedial action. Insofar as the timing of the certification process is concerned, and as we explained in our December 18, 2015, letter, EPA does not at this time believe that there is sufficient time for all of the consent decree's certification prerequisites to be met prior to April 23, 2017, particularly because some remedial action work, including habitat reconstruction and decommissioning of the Fort Edward facility, will not likely be completed before mid- to late 2016. The completion of those activities will then trigger a series of steps under the consent decree that must be undertaken before EPA could be in a position to certify GE's completion of the remedial action. We do not expect there to be sufficient time to complete those steps prior to the summer of 2017, if not later, which would be at least several months after the due date for the Second Five Year Review report.

Operation, Maintenance and Monitoring Plan

Your January 22 letter suggests that EPA's review of GE's OM&M Plan should not delay the data collection needed for the Second Five Year Review. We agree.

Timing of First "Workshop"

EPA agrees with your comment in the January 22 letter that the first workshop for the Five Year Review should occur prior to the late summer of 2016. As noted above, EPA's current schedule calls for holding a public workshop with the CAG in late April of 2016. Members of the public will be invited to attend.

Coordination between EPA Region 2 and EPA Headquarters

Your January 22 letter states that your organizations "trust that" EPA Region 2 and EPA's Office of Land and Emergency Management ("OLEM," formerly EPA's Office of Solid Waste and Emergency Response) will continue to coordinate with regard to the schedule and substance of the Second Five Year Review. EPA Region 2 and OLEM will coordinate closely throughout the Second Five Year Review.

Next Steps

There are a number of statements in the Petition with which we disagree, but to which EPA does not believe it is necessary to respond at this time. Instead, we believe our collective and cooperative focus should be on the Second Five Year Review, in which EPA will conduct a new evaluation of the remedy's protectiveness in accordance with CERCLA, and for which EPA will continue to provide opportunities for public involvement. The Second Five Year Review will incorporate sediment, water, and spring fish data to be collected in 2016, and take into consideration NOAA's analyses that underpin many of the concerns in the Petition regarding the cleanup work to date. With regard to the topics suggested in the February 23 letter for the Second Five Year Review, please note that the review will include evaluations of the projected time frames for reducing PCB concentrations in Upper and Lower Hudson River fish, and also will consider questions raised (such as in your February 23 correspondence) about potential risks from PCBs that volatilize from the river. With respect to the fish consumption advisories, prior creel surveys already have shown that anglers do not always follow the advisories. We note that EPA will continue to coordinate with the New York State Department of Environmental Conservation and New York State Department of Health as they consider adjustments to the consumption advisories, as appropriate. As we said in our December 18, 2015, letter, if after the Second Five Year Review EPA determines that the remedy is not protective, EPA will consider what additional actions are needed.

We appreciate and share your organizations' work and strong interest in the success of this project. EPA is committed to ensuring that the remedy is protective of human health and the

environment, and looks forward to the five year review process.

Sincerely,

Judith A. Enck

Judith A. Enck
Regional Administrator

cc: Basil Seggos, Acting Commissioner
New York State Department of Environmental Conservation

Wendi Weber, Regional Director
Northeast Region
U.S. Fish & Wildlife Service

Tony Penn, Chief
Assessment & Restoration Division
Office of Response and Restoration
National Oceanic & Atmospheric Administration

Attachment J

Contaminated Sediment Remediation Guidance for Hazardous Waste Sites

Contaminated Sediment Remediation Guidance for Hazardous Waste Sites

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United States Environmental Protection Agency
Office of Solid Waste and Emergency Response

EPA-540-R-05-012
OSWER 9355.0-85
December 2005

ADDITIONAL COPIES

The *Contaminated Sediment Remediation Guidance for Hazardous Waste Sites* is available to download from EPA s Superfund program Web site at http://www.epa.gov/superfund/resources/sediment/guidance.htm. Hard copies of the document can be obtained at no charge by contacting by contacting EPA s National Service Center for Environmental Publications (NSCEP) at (800) 490-9198 or ordered via the Internet at http://www.epa.gov/nscep/ordering.htm.

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ACKNOWLEDGMENTS

Initial drafts of this document were prepared by an Inter-Agency workgroup led by the U.S. Environmental Protection Agency (EPA) Office of Emergency and Remedial Response [now Office of Superfund Remediation and Technology Innovation (OSRTI)]. In addition to EPA, the workgroup included representatives from the following organizations:

National Oceanic and Atmospheric Administration (NOAA)
U.S. Army Corps of Engineers (USACE)
U.S. Fish and Wildlife Service (USFWS)

Representatives of other organizations contributed to the document by commenting on early drafts. These included the following:

Environment Canada
U.S. Navy
U.S. Geological Survey
U.S. Department of Energy
Oregon Department of Environmental Quality
Massachusetts Department of Environmental Quality
Wisconsin Department of Natural Resources

The following individuals led subgroups to draft various sections of the document or otherwise contributed substantially to the overall character of the guidance:

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Executive Summary

In 2004, the U.S. Environmental Protection Agency (EPA) released the *Updated Report on the Incidence and Severity of Sediment Contamination in Surface Waters of the United States: National Sediment Quality Survey*, which identifies areas in all regions of the country where sediment may be contaminated at potentially harmful levels (U.S. EPA 2004a). Contaminated sediment can significantly impair the navigational and recreational uses of rivers and harbors in the U.S. [National Research Council (NRC) 1997 and 2001] and can be a contributing factor in many of the 3,221 fish consumption advisories nationwide (U.S. EPA 2005a). As of 2004, EPA had decided to take action to clean up contaminated sediment at approximately 140 sites, including federal facilities, under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and additional sites under the Resource Conservation and Recovery Act [(RCRA), U.S. EPA 2004a]. The remedies for more than 60 sites are large enough that they are being tracked at the national level. Many other sites are being cleaned up under state authorities, other federal authorities, or as voluntary actions.

This document provides technical and policy guidance for project managers and management teams making remedy decisions for contaminated sediment sites. It is primarily intended for federal and state project managers considering actions under CERCLA, although technical aspects of the guidance are also intended to assist project managers addressing sediment contamination under RCRA. Many aspects of this guidance also will be useful to other governmental organizations and potentially responsible parties (PRPs) that may be conducting a sediment cleanup. Although aspects related to site characterization and risk assessment are addressed, the guidance focuses on considerations regarding feasibility studies and remedy selection for contaminated sediment. The guidance is lengthy, and users may wish to consult sections most applicable to their current need. To help in this process, a short summary of each of the eight chapters is provided below. Sediment cleanup is a complex issue, and as new techniques evolve, EPA will issue new or updated guidance on specific aspects of contaminated sediment assessment and remediation. Links to guidance and additional information about contaminated sediments at Superfund sites are available at http://www.epa.gov/superfund/resources/sediment.

Chapter 1, Introduction, describes the general backdrop for contaminated sediment remediation and reiterates EPA s previously issued Office of Solid Waste and Emergency Response (OSWER) Directive 9285.6-08, *Principles for Managing Contaminated Sediment Risks at Hazardous Waste Sites* (U.S. EPA 2002a). Other issues addressed in Chapter 1 include the role of the natural resource trustees, states, Indian tribes, and communities at sediment sites. Where there are natural resource damages associated with sediment sites, coordination between the remedial and trusteeship roles at the federal, state, and tribal levels is especially important. In addition to their role as natural resource trustees, certain state cleanup agencies and certain Indian tribes or nations have an important role as co-regulators and/or affected parties and as sources of essential information. Communities of people who live and work adjacent to water bodies containing contaminated sediment should be given understandable information about the safety of their activities, and be provided significant opportunities for involvement in the EPA s decision-making process for sediment cleanup.

Chapter 2, Remedy Investigation Considerations, introduces investigation issues unique to the sediment environment, including those related to characterizing the site, developing conceptual site models, understanding current and future watershed conditions, controlling sources, and developing cleanup goals. Especially important at sediment sites is the development of an accurate conceptual site

model, which identifies contaminant sources, transport mechanisms, exposure pathways, and receptors at various levels of the food chain. Project managers should consider the role of a sediment site in the watershed context, including other potential contaminant sources, key issues within the watershed, and current and reasonably anticipated or desired future uses of the water body and adjacent land. Important parts of site characterization and remedy selection include the identification and, where feasible, control of significant continuing sources of contamination and an accurate understanding of their contribution to site risk and potential for recontamination. It is also generally important that remedial action objectives, remediation goals, and cleanup levels are based on site-specific data and are clearly defined. At most Superfund sites, chemical-specific remediation goals should be developed into final sediment cleanup levels by weighing the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) balancing and modifying criteria.

In addition, Chapter 2 introduces issues relating to sediment mobility and contaminant fate and transport, and modeling at sediment sites. In most aquatic environments, surface sediment and associated contaminants move over time. An important part of the remedial investigation at many sediment sites is a site-specific assessment of whether movement of contaminated sediment (surface and subsurface), or of contaminants alone, is occurring or may occur at scales and rates that will significantly change their contribution to risk. For example, is significant sedimentation of cleaner sediment burying contaminated sediment, and, if so, how quickly, and is erosion likely to re-expose those contaminants in the future? An accurate assessment of sediment mobility and contaminant fate and transport can be one of the most important factors in identifying areas suitable for monitored natural recovery (MNR), in-situ caps, or near-water confined disposal facilities (CDFs). Evaluation of alternatives should include consideration of disruption from man-made (anthropogenic) causes such as propeller scour and natural causes such as floods and ice scour. Generally, this evaluation should include the 100-year flood and other events with a similar probability of occurrence. Project managers should make use of the variety of field and laboratory measurement methods available for evaluating site characteristics. For example, the shear stress necessary to erode sediment or the increase in exposure of biota that might be expected from any contaminants transported to surface water from ground water.

Where appropriate, project managers also should make use of numerical models for predicting future conditions at a site. There is a wide range of models, from simple to complex, which can be applied to contaminated sediment sites. Where numerical models are used, verification, calibration, and validation should be typically preformed to yield a scientifically defensible study. While quantitative uncertainty analyses can be performed for watershed loading and food web models, at the current time they cannot be generally performed for fate and transport models. However, frequently a sensitivity analysis can be used to identify the model parameters that have most impact on model results, so that the project team can ensure that these parameters are well constrained by site data.

Chapter 3, Feasibility Study Considerations, supplements existing EPA guidance by offering sediment-specific guidance about developing alternatives, applying the NCP remedy selection criteria, identifying applicable or relevant and appropriate requirements (ARARs), evaluating effectiveness and permanence, estimating cost, and using institutional controls. Major alternatives include dredging and excavation, in-situ capping, and MNR. Innovative lab and field testing of in-situ treatment in the form of reactive caps or sediment additives are underway and may be useful in the future. Due to the limited number of cleanup methods available for contaminated sediment, generally project managers should evaluate each of the three potential remedy approaches (sediment removal, capping, and MNR) at every

sediment site. At large or complex sites, project managers have found that alternatives that combine a variety of approaches are frequently cost effective. Pursuant to CERCLA section 121, all final remedial actions at CERCLA sites must be protective of human health and the environment, and must comply with ARARs unless a waiver is justified. Developing accurate cost estimates is an important part of evaluating sediment alternatives. Project managers should evaluate capital costs, operation and maintenance costs (including long-term monitoring), and net present value. When evaluating alternatives with respect to effectiveness and permanence, it is important to remember that each of the three potential remedy approaches may be capable of reaching acceptable levels of effectiveness and permanence, and that site-specific characteristics should be reviewed during the alternatives evaluation to ensure that the alternative selected will be effective in that environment. Institutional controls are frequently evaluated as part of sediment alternatives to prevent or reduce human exposure to contaminants. Common types of institutional controls at sediment sites include fish consumption advisories, commercial fishing bans, and waterway use restrictions. In some cases, land use restrictions or structure maintenance agreements have also been important elements of an alternative.

Chapter 4, Monitored Natural Recovery, describes the natural processes that should be considered when evaluating MNR as a remedy, and briefly discusses enhanced natural recovery through thin-layer placement of sand or other material. MNR is a remedy that typically uses known, ongoing, naturally occurring processes to contain, destroy, or otherwise reduce the bioavailability or toxicity of contaminants in sediment. An MNR remedy generally includes site-specific cleanup levels and remedial action objectives, and monitoring to assess whether risk is being reduced as expected. Although a no action decision may also include monitoring, in this case the monitoring is intended to ensure that an already-acceptable level of risk is maintained (e.g., that deeply buried contaminants are not re-exposed by erosion). Although burial by clean sediment is often the dominant process relied upon for natural recovery, multiple physical, biological, and chemical mechanisms frequently act together to reduce risk. Evaluation of MNR should be usually based on site-specific data, including multiple lines of evidence such as decreasing trends of contaminant levels in fish, in surface water, and in sediment. Project managers should evaluate the long-term stability of the sediment bed and the mobility of contaminants within it. Contingency measures should be included as part of a MNR remedy when there is significant uncertainty that the remedial action objectives will be achieved within the predicted time frame. Generally, MNR should be used either in conjunction with source control or active sediment remediation.

In addition, Chapter 4 discusses the potential advantages and limitations of MNR. In most cases, the two key advantages of MNR are its relatively low implementation cost and its non-invasive nature. While costs associated with site characterization and modeling can be extensive, the costs associated with implementing MNR are primarily associated with monitoring. Because no construction or infrastructure is needed, it is generally much less disruptive to human communities and the ecosystem than active remedies. Two key limitations of MNR may be that it generally leaves contaminants in place without engineered containment and that it can be slow in reducing risks in comparison to active remedies. As with any risk reduction approach that takes a period of time to reach remediation goals, remedies that include MNR frequently rely upon institutional controls, such as fish consumption advisories, to control human exposure during the recovery period. At most sites, some people will disregard advisories despite best efforts to communicate risk, and advisories have no ability to reduce ecological exposures.

Chapter 5, In-Situ Capping, summarizes the major capping technologies and describes the site conditions that are important to understand in evaluating the feasibility and effectiveness of in-situ

capping. In-situ capping refers to the placement of a subaqueous covering or cap of clean material over contaminated sediment that remains in place. Caps are generally constructed of clean sediment, sand, or gravel, but can also include geotextiles, liners, or the addition of material, such as organic carbon, to attenuate the flux of contaminants into the overlying water. Depending on the contaminants and sediment conditions present, a cap is generally designed to reduce risk through the following primary functions: 1) physical isolation of the contaminated sediment sufficient to reduce exposure due to direct contact and to reduce the ability of burrowing organisms to move contaminants to the cap surface; 2) stabilization of contaminated sediment and erosion protection of sediment and cap sufficient to reduce resuspension and transport of contaminants into the water column; and 3) chemical isolation of contaminated sediment sufficient to reduce exposure from dissolved contaminants that may be transported into the water column.

In addition, Chapter 5 discusses the potential advantages and limitations of in-situ capping. One advantage of in-situ capping is that it can quickly reduce exposure to contaminants. Also, compared to sediment removal it normally requires both less infrastructure in terms of material handling, dewatering, and disposal and is typically less disruptive to people in local communities. Compared to MNR, the potential for erosion and transport of contaminants is typically much lower. However, contaminated sediment is still left in place in the aquatic environment where contaminants could be exposed or dispersed if the cap is significantly disturbed or if contaminants move through the cap in significant amounts. Another potential limitation to in-situ capping may be that in some situations a preferred habitat may not be provided by the surficial cap materials which may be needed for erosion control.

Chapter 6, Dredging and Excavation, describes dredging technologies (conducted under water) and excavation technologies (typically conducted after water is diverted or drained). The chapter describes some of the key components involved in a sediment dredging or excavation remedy and describes site conditions that may be important when evaluating the feasibility and effectiveness of these remedies. A dredging or excavation alternative should include an evaluation of all phases of the project, including removal, staging, dewatering, water treatment, sediment transport, and sediment treatment, reuse, or disposal. Transport and disposal options for contaminated sediment are sometimes complex and controversial and should be investigated and discussed with stakeholders early in the project. In some cases, specialized methods of operation or equipment may be needed to minimize resuspension of sediment and transport of contaminants. Project managers should make realistic, site-specific predictions of residual contamination (i.e., contamination that remains within or adjacent to the dredged area after dredging) based on pilot studies or data from comparable sites. Where residuals are a concern, thin layer placement/backfilling, MNR, or capping may also be needed.

In addition, Chapter 6 discusses potential advantages and limitations of contaminated sediment removal by dredging and excavation. One of the principal advantages of dredging and excavation is often that, if they achieve cleanup levels for the site, they may result in the least uncertainty regarding future environmental exposure to contaminants because the contaminants are removed from the aquatic ecosystem and disposed in a controlled environment. Another potential advantage of removing contaminated sediment rather than managing it in place is that it may leave more flexibility regarding future use of the water body. Although dredging remedies at sites with bioaccumulative contaminants usually include fish consumption advisories for a period of time after sediment removal, other types of institutional controls that might be needed to protect a cap or a layer of natural sedimentation are usually not necessary. The principal limitations of sediment removal are that it is usually more complex and costly than in-situ management, and that the level of uncertainty associated with estimating residual

contamination can be high at some sites. The need for transport, storage, treatment (where applicable), and disposal facilities may lead to increased impacts on communities. In some parts of the country, disposal capacity may be limited in existing municipal or hazardous waste landfills and it may be difficult to site new local disposal facilities. Another limitation may include the potential for contaminant losses during dredging through resuspension, and to a generally lesser extent, through other processes such as volatilization during excavation, transport, treatment, or disposal. Finally, similar to in-situ capping, dredging or excavation typically includes at least a temporary destruction of the aquatic community and habitat within the remediation area.

Chapter 7, Remedy Selection Considerations, discusses risk management decision making, the NCP s remedy selection framework, including considering sediment remedies and comparing net risk reduction, considering alternatives that include institutional controls, and considering a no-action decision. Where a remedy is necessary, the best route to overall risk reduction depends on a large number of site-specific considerations, some of which may be subject to significant uncertainty. Any decision regarding the specific choice of a remedy for contaminated sediment should be based on a careful consideration of the advantages and limitations of each available approach and a balancing of trade-offs among alternatives. This chapter includes two summary tables to help with this comparison process: one describes site characteristics and conditions especially conducive to each of the three potential remedy approaches for sediment (MNR, capping, and dredging), and the other lists examples of key differences between the three potential remedy approaches with respect to the NCP s nine remedy selection criteria. Documenting and communicating how and why remedy decisions were made are especially important at complex sites. The concept of comparing net risk reduction may assist in the remedy selection process by providing a framework for considering elements of alternatives which may reduce risk and elements which may allow risk to continue or temporarily increase. When considering remedies that include institutional controls, project managers should consider what entities possess the legal authority, capability and willingness to implement the control.

EPA s policy has been and continues to be that there is no presumptive remedy for any contaminated sediment site, regardless of the contaminant or level of risk. At many sites, but especially at large sites, a combination of sediment cleanup methods may be the most effective way to manage the risk. The remedy selection process for sediment sites should include a clear analysis of the uncertainties involved, including uncertainties concerning the predicted effectiveness of various alternatives and the time frames for achieving cleanup levels and, if possible, remedial action objectives. The uncertainty of factors very important to the remedy decision should be quantified, so far as this is possible. Where it is not possible to quantify uncertainty, sensitivity analysis may be helpful to determine which apparent differences between alternatives are most likely to be significant.

Chapter 8, Remedial Action and Long-Term Monitoring, provides a recommended approach to developing an effective monitoring plan at contaminated sediment sites. The chapter presents sample measures of sediment remedy effectiveness, in terms of remedy performance and risk reduction. A fully successful sediment remedy typically is one where the selected sediment chemical or biological cleanup levels have been met and maintained over time, and where all relevant risks have been reduced to acceptable levels based on the anticipated future uses of the water body and the goals and objectives stated in decision documents. The chapter also presents the key steps in designing and conducting a monitoring program at a sediment site, introduces some of the monitoring techniques available for physical, chemical, and biological measurements, and summarizes some of the factors to consider when

monitoring remedies including MNR, in-situ capping, or dredging/excavation. A monitoring plan typically can be important for all types of sediment remedies, before, during and after remedial action. The development of monitoring plans should follow a systematic planning process that identifies monitoring objectives, decision criteria, endpoints, and data collection and interpretation methods. Project managers should ensure that adequate baseline data are available for comparison to monitoring data after a remedial action and that adequate background data are available, including any continuing off-site contaminant contributions. Monitoring before, during, and after sediment remediation generally will help not only to answer site-specific questions but to contribute to a better understanding of remedy performance at the national level.

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8.0 REMEDIAL ACTION AND LONG-TERM MONITORING

1.0 INTRODUCTION

1.1 PURPOSE

This document provides technical and policy guidance for project managers and management teams making risk management decisions for contaminated sediment sites. It is primarily intended for federal and state project managers considering remedial response actions or non-time-critical removal actions under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), more commonly known as Superfund. Technical aspects of the guidance are also intended to assist project managers addressing sediment contamination under the Resource Conservation and Recovery Act (RCRA). Many aspects of this guidance may also be useful to other governmental organizations and potentially responsible parties (PRPs) that are conducting a sediment cleanup under CERCLA, RCRA, or other environmental statutes, such as the Clean Water Act (CWA) or the Water Resource Development Act (WRDA). This guidance may also be useful to members of the community and their technical representatives.

This guidance also provides information to the public and to the regulated community on how EPA intends to exercise its discretion in implementing its regulations at contaminated sediment sites. It is important to understand, however, that this document does not substitute for statutes EPA administers nor their implementing regulations, nor is it a regulation itself. Thus, this document does not impose legally binding requirements on EPA, states, or the regulated community, and may not apply to a particular situation based upon the specific circumstances. Rather, the document suggests approaches that may be used at particular sites as appropriate, given site-specific circumstances. EPA made many changes to this document based on public comment and external peer review of draft documents. Even though the document is now final, however, EPA welcomes public comments on the document at any time and will consider those comments in any future revisions to the document which EPA may make without public notice.

Guidance presented in this document can be applied to contaminated sediment in a wide variety of aquatic environments, including rivers, streams, wetlands, ponds, lakes, reservoirs, harbors, estuaries, bays, intertidal zones, and coastal ocean areas. Sediment in wastewater lagoons, detention/sedimentation ponds, on-site storage/containment facilities, or roadside ditches is not addressed. This guidance addresses both in-situ and ex-situ remedies for sediment, including monitored natural recovery (MNR), in-situ capping, and dredging and excavation. However, because the science and practice of sediment remediation are rapidly evolving, project managers are encouraged to test innovative approaches (e.g., including in-situ treatment options) that are beyond those discussed here, which may also effectively reduce risk from contaminated sediment.

Consideration of materials deposited in floodplains, whether called soil or sediment, is an important factor in reducing risk in aquatic environments. Much of the general approach recommended in this guidance can be applied to contaminated floodplains, although the technical considerations are written with aquatic sediment in mind. Control of upland soils and other upland source materials is also critical to reducing risk in aquatic environments, but in general, existing guidance should be used for these materials [e.g., the U.S. Environmental Protection Agency s (EPA s) *Soil Screening Guidance: Users Guide* (U.S. EPA 1996a)]. However, where floodplain soils may be a source of contamination to surface water or sediment, the fate and transport of contaminants in the soil should be evaluated.

The emphasis of this guidance is on evaluating alternatives (e.g., the feasibility study stage of the Superfund process) and remedy selection, although the guidance presents some of the key remedial investigation issues at sediment sites. Following this introductory chapter, the guidance provides sediment-specific issues to consider during remedial investigations (see Chapter 2) and feasibility studies (see Chapter 3), followed by chapters concerning the three potential remedy approaches for sediment management (see Chapter 4, Monitored Natural Recovery; Chapter 5, In-Situ Capping; and Chapter 6, Dredging and Excavation). This guidance then presents information on selecting sediment remedies (see Chapter 7); and on monitoring sediment sites (see Chapter 8).

1.2 CONTAMINATED SEDIMENT

For the purposes of this guidance, contaminated sediment is soil, sand, organic matter, or other minerals that accumulate on the bottom of a water body and contain toxic or hazardous materials at levels that may adversely affect human health or the environment (U.S. EPA 1998a). Contaminants adsorbed to soil or in other forms may wash from land, be deposited from air, erode from aquatic banks or beds, or form from the underwater breakdown or buildup of minerals (U.S. EPA 1998a). Contaminated sediment may be present in wetlands, streams, rivers, lakes, reservoirs, harbors, along ocean margins, or in other water bodies. In this guidance, water body generally includes all of these environments. Some contaminants have both anthropogenic (or man-made) sources and natural sources (e.g., many metals and some organic compounds). This guidance addresses management of contaminants present above naturally occurring levels that may cause an unacceptable risk to humans or to ecological receptors.

Examples of primary and secondary sources of contaminants in sediment are included in Highlight 1-1.

Highlight 1-1: Potential Sources of Contaminants in Sediment

- Direct pipeline or outfall discharges into a water body from industrial facilities, waste water treatment plants, storm water discharges, or combined sewer overflows
- Chemical spills into a water body
- Surface runoff or erosion of soil from floodplains and other contaminated sources on land, such as waste dumps, chemical storage facilities, mines and mine waste piles, and agricultural or urban areas
- Air emissions from power plants, incinerators, pesticide applications, or other sources that may be transferred to a water body through precipitation or direct deposition
- Upwelling or seepage of contaminated ground water or non-aqueous phase liquids (NAPL) into a water body
- Direct disposal from docked and dry-docked ships, or release of contaminants from in-water structures and over-water structures or ship maintenance facilities

Organic contaminants in sediment typically adsorb to fine sediment particles and exist in the pore water between sediment particles. Metals also adsorb to sediment and may bind to sulfides in the sediment. The relative proportion of contaminants between sediment and pore water depends on the type of contaminant and the physical and chemical properties of the sediment and water. Pore water in sediment generally is interconnected with both surface water and ground water, although the degree of

interconnection may change from place-to-place and with flow changes in ground water and surface water.

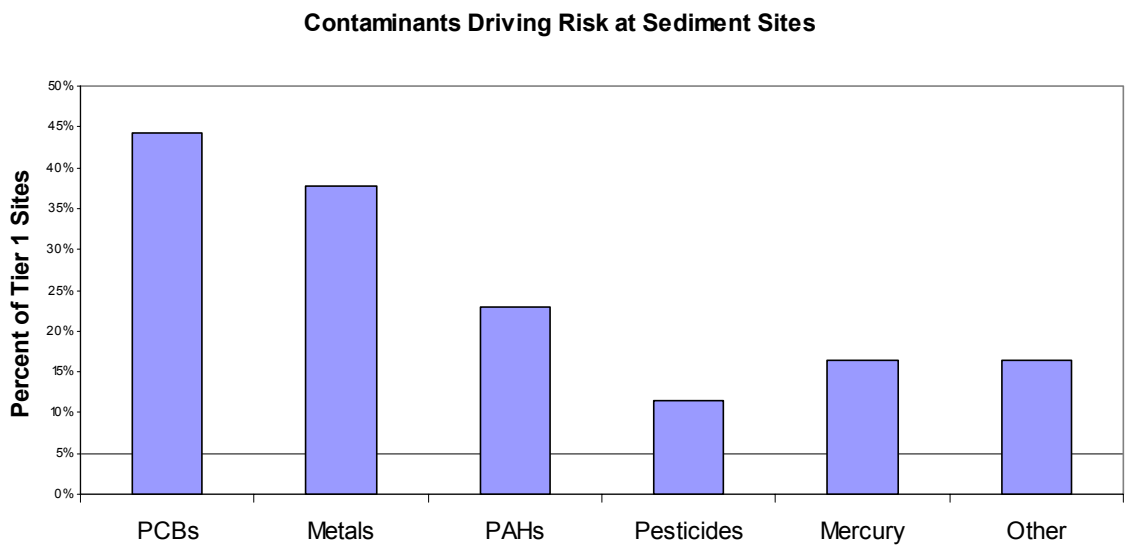
Many contaminants persist for years or decades because the contaminant does not degrade or degrades very slowly in the aquatic environment. Contaminants sorbed to sediment normally develop an equilibrium with the dissolved fraction in the pore water and in the overlying surface water to be taken up by fish and other aquatic organisms. Some bottom-dwelling organisms ingest contaminated sediment, and in shallow water environments, humans may also come into direct contact with contaminated sediment. Some contaminants, such as most metals, are hazardous primarily because of direct toxicity. Although some metals do accumulate in biota (i.e., bioaccumulate), generally they do not significantly increase in concentration as they are passed up the food chain (i.e., biomagnify). Others, called persistent bioaccumulative toxics (PBTs) [e.g., polychlorinated biphenyls (PCBs), pesticides, and methyl mercury] are of concern primarily because they may both bioaccumulate and biomagnify. Concentrations of PBTs in fish may endanger humans and wildlife that eat fish. Women of childbearing age, young children, people who derive much of their diet from fish and shellfish, and people with impaired immune systems may be especially at risk.

In 2004, the EPA released *The Updated Report on the Incidence and Severity of Sediment Contamination in Surface Waters of the United States* (U.S. EPA 2004a). This report identifies locations in all regions of the country where sediment contamination could be associated with probable or possible adverse effects to aquatic life and/or human health. In 2004, state and local authorities issued 3,221 advisories limiting fish consumption, which cover 35 percent of the nation s total lake acreage (excluding the Great Lakes), 24 percent of the nation s total river miles, and 100 percent of the Great Lakes and connecting waters, in part due to sediment contamination (U.S. EPA 2005a). In addition, contaminated sediment can significantly impair the navigational and recreational uses of rivers and harbors in the U.S. Navigational dredging is not currently being performed in many harbors and waterways because of the concern for impacts of dredging on water quality, liability to those performing the dredging, and disposal options for the contaminated dredged material [National Research Council (NRC 1997 and 2001)].

As of 2004, the Superfund program had decided to take an action to address sediment at approximately 140 sites, including federal facilities. The remedies for more than 60 sites, called Tier 1 sites, are large enough that they are being tracked at the national level [for more information view the Office of Superfund Remediation and Technology Innovation s (OSRTI s) Contaminated Sediments in Superfund Web site at http://www.epa.gov/superfund/resources/sediment/sites.htm]. These sites include a wide variety of contaminants, as presented in Highlight 1-2.

Many aspects of the cleanup process may be more complex at sediment sites versus sites with soil or ground water contamination alone. Some potentially complicating factors for addressing contaminated sediment sites are listed in Highlight 1-3. Based on these factors and other reasons as presented in this guidance, a team of experts is frequently needed to advise the project manager (see Section 1.4.2 Technical Team Approach).

Highlight 1-2: Major Contaminants at Superfund Sediment Sites (Sites with Remedies Selected through 2004)



Highlight 1-3: Why Sediment Sites Are a Unique Challenge

- Sediment sites may have a large number of sources, some of which can be ongoing and difficult to control
- The sediment environment is usually dynamic, and understanding the effect of natural forces and man-made (anthropogenic) events on sediment movement and stability as well as contaminant transport can be difficult
- Cleanup work in an aquatic environment is frequently difficult from an engineering perspective and may be more costly than other media
- Contamination is often diffuse and the sites are often large and diverse (e.g., mixed use, numerous property owners)
- Many sediment sites contain ecologically valuable resources or legislatively protected species or habitats
- For large sites, a number of communities with differing views and opinions may be affected
- There may be significant injuries to trustee resources at sediment sites

1.3 RISK MANAGEMENT PRINCIPLES AND REMEDIAL APPROACHES

Office of Solid Waste and Emergency Response (OSWER) Directive 9285.6-08, *Principles for Managing Contaminated Sediment Risks at Hazardous Waste Sites* (U.S. EPA 2002a; attached as Appendix A to this document), presents eleven risk management principles that help project managers make scientifically sound and nationally consistent risk management decisions at contaminated sediment sites. Project managers should carefully consider these principles when planning and conducting site investigations, involving the affected parties, and selecting and implementing a response.

The eleven risk management principles should be applied within the framework of the EPA s existing statutory and regulatory requirements, such as the National Oil and Hazardous Substances Pollution Contingency Plan s (NCP s) nine remedy selection criteria (Title 40 Code of Federal Regulations (40 CFR) 300.430(c)). The eleven principles are listed in Highlight 1-4 and are incorporated throughout this guidance. The project manager should refer to OSWER Directive 9285.6-11, *OSRTI Sediment Team and the NRRB [National Remedy Review Board] Coordination at Large Sediment Sites* (U.S. EPA 2004b) to help ensure that the eleven principles are appropriately considered before making site-specific risk management decisions. Copies of both directives can be found on EPA s Superfund Web site at http://www.epa.gov/superfund/resources/sediment/documents.htm.

Highlight 1-4: Risk Management Principles Recommended for Contaminated Sediment Sites

1.	Control sources early
2.	Involve the community early and often
3.	Coordinate with states, local governments, Indian tribes, and natural resource trustees
4.	Develop and refine a conceptual site model that considers sediment stability
5.	Use an iterative approach in a risk-based framework
6.	Carefully evaluate the assumptions and uncertainties associated with site characterization data and site models
7.	Select site-specific, project-specific, and sediment-specific risk management approaches that will achieve risk-based goals
8.	Ensure that sediment cleanup levels are clearly tied to risk management goals
9.	Maximize the effectiveness of institutional controls and recognize their limitations
10.	Design remedies to minimize short-term risks while achieving long-term protection
11.	Monitor during and after sediment remediation to assess and document remedy effectiveness

Source: U.S. EPA 2002a; see Appendix A

1.3.1 Remedial Approaches

Highlight 1-5 lists the major remedial approaches or alternatives available for managing risks from contaminated sediment. Frequently, a final sediment remedy combines more than one type of approach.

Highlight 1-5: Remedial Approaches for Contaminated Sediment

In-situ Approaches	Ex-situ Approaches
In-situ Capping: • Single-layer granular caps • Multi-layer granular caps • Combination granular/geotextile caps Monitored Natural Recovery: • Physical isolation or other processes • Chemical transformation/sequestration • Biological transformation/sequestration Hybrid Approaches: • Thin layer placement of sand or other material to enhance recovery via natural deposition Institutional Controls: • Fish consumption advisories • Commercial fishing bans • Waterway or land use restrictions (e.g., no anchor or no wake zones, limitations on navigational dredging) • Dam or other structure maintenance agreements In-situ Treatment: • Reactive caps • Additives/enhanced biodegradation	Dredging: • Hydraulic, mechanical, or combination/hybrid dredging and transport to shore • Treatment of dredged sediment and/or removed water • Disposal of dredged sediment or treatment residuals in upland landfill, confined disposal facility, or other placement • Backfill of dredged area, as needed or appropriate Excavation: • Water diversion or dewatering • Excavation of sediment and transport to staging or processing • Treatment of excavated sediment • Disposal of excavated sediment or treatment residuals in upland landfill, confined disposal facility, or other placement • Backfill of excavated area, as needed or appropriate

1.3.2 Urban Revitalization and Reuse

Revitalizing urban areas and returning land and water bodies to productive uses have become increasingly important to the EPA s hazardous waste programs in recent years. Sediment sites may present opportunities to incorporate these concepts into remedy selection, remedial design, and into other phases of the risk management process. At sediment sites in urban areas, project managers should consider the goals of local governments and other entities to revitalize the use of waterfront property, harbors, and water bodies. This may involve reviewing local land use plans and identifying potential partners such as land owners, elected officials, and local land and water planning and development agencies. It may lead to opportunities to consider remedies that take into account the views of local stakeholders, land owners, and land use planners. For example, it may be possible to locate disposal structures or rail lines in areas that maximize future reuse. Beneficial reuse of dredged material may also present an opportunity for urban revitalization. Project managers are encouraged to make use of a collaborative Web site on beneficial reuse co-sponsored by the U.S. Army Corps of Engineers (USACE) Engineer Research and Development Center and EPA s Office of Wetlands, Oceans, & Watersheds, available at http://el.erdc.usace.army.mil/dots/budm/budm.html.

1.4 DECISION-MAKING PROCESS

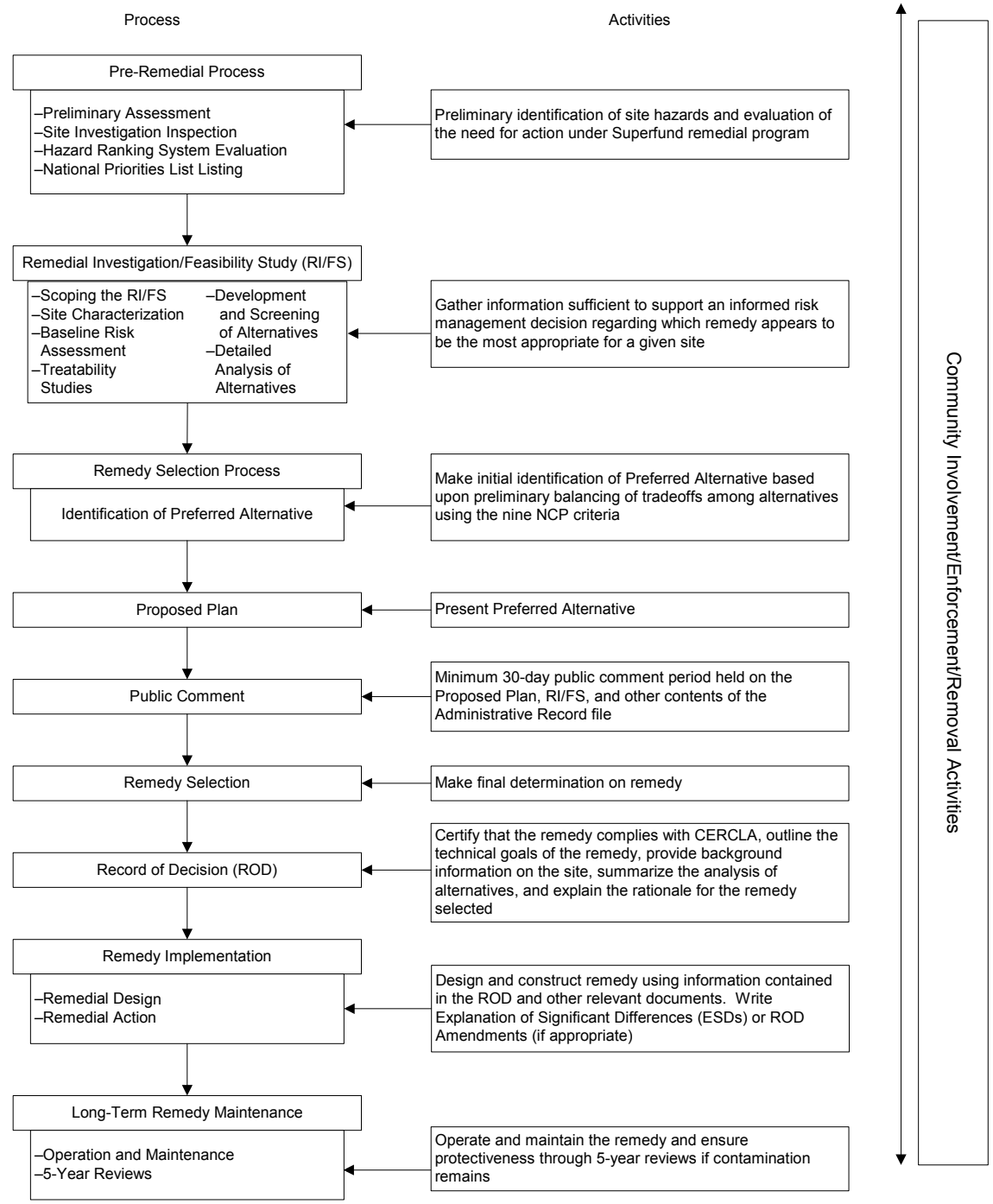
Decision making at sediment sites can follow somewhat different processes depending on the legal authority under which the sediment cleanup is conducted, the entity conducting the cleanup, and the scope of the problem. While meeting all legal and regulatory requirements, it is the intent of the Agency to allow project managers the flexibility needed to make the most appropriate recommendation for their site.

1.4.1 Decision Process Framework

Remedial actions taken under CERCLA generally follow the Superfund remedial response process shown in Highlight 1-6, taken from *A Guide to Preparing Superfund Proposed Plans, Records of Decision, and Other Remedy Selection Decision Documents* (U.S. EPA 1999a, also referred to as the ROD Guidance). Project managers should refer to the ROD Guidance for descriptions of each stage of the remedial process. Corrective actions under RCRA generally follow the RCRA remedial process laid out in the May 1, 1996 Advanced Notice of Proposed Rulemaking [(ANPR), 61 *Federal Register* (*FR*) 19447].

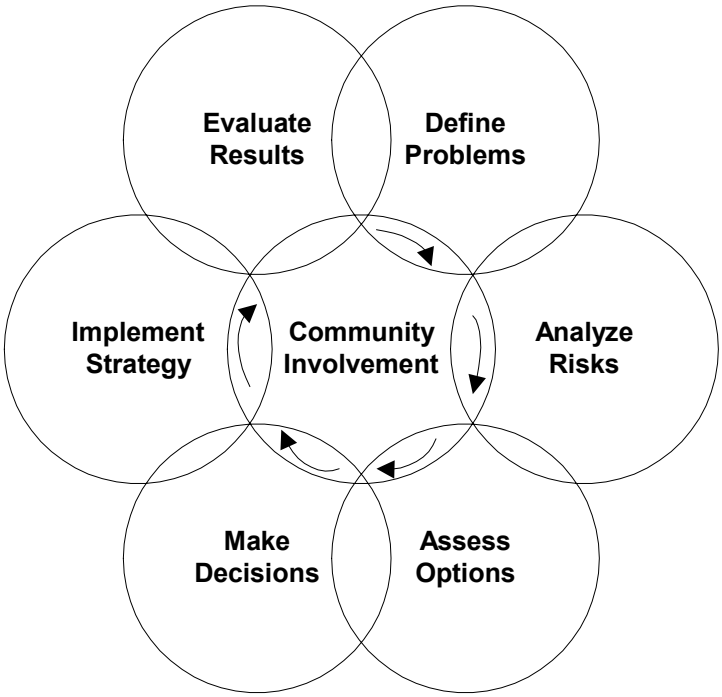
In the report, *A Risk-Management Strategy for PCB-Contaminated Sediments* (NRC 2001), the NRC recommended the use of the iterative decision-making approach, adapted from the 1997 Presidential/Congressional Commission on Risk Assessment and Risk Management (PCCRARM) risk management framework (Highlight 1-7). EPA project managers should consider using this approach within the context of EPA s existing remedial process. The NRC approach emphasizes the unique importance of community involvement throughout the decision-making process and the usefulness of iteration and adaptation if new information becomes available that changes the nature or understanding of the problem.

Highlight 1-6: General Overview of the Superfund Remedial Response Process



Adapted from: U.S. EPA 1999a

Highlight 1-7: National Research Council - Recommended Framework for Risk Management



Source: NRC 2001

1.4.2 Technical Team Approach

At many sediment sites, like other complex sites, a technical team approach frequently works best for effective site management. This team may be made up of lead and support regulatory agency technical personnel and experts from within and outside of the agencies, including those representing responsible parties. Typically, it is most effective to form this group early in the site investigation process and maintain it with as much continuity as possible throughout the decision making and implementation of the project. Ongoing dialogue managed by the project manager among the technical team on all of the technical issues should help to ensure a productive, efficient site investigation and evaluation of remedial alternatives in which the tendency toward an adversarial environment is minimized. This approach may require a strong project manager who facilitates the meetings and makes tough and fair decisions at points of disagreement.

Technical teams, which include experts representing both government and responsible parties, can be especially effective when the following principles are considered:

- Use sound, high quality science as the basis for site-specific decisions to
  - jointly identify information needs and project objectives;
  - call upon appropriate expertise;
  - recognize and understand uncertainty; and
  - operate in an atmosphere of respect.

- Communicate openly and frequently to
  - foster partnerships with all stakeholders and listen to all viewpoints;
  - jointly identify areas of disagreement and means to resolve them; and
  - openly discuss site goals and capabilities of available alternatives.

- Think outside the box to
  - look for common ground and shared goals;
  - solicit help of an outside neutral party when needed;
  - experiment with a change in structure when needed; and
  - look for opportunities to make progress.

1.4.3 Technical Support

In 2004, EPA established the Superfund Sediment Resource Center (SSRC) to make expert technical assistance available to EPA project managers of any Superfund sediment site. The SSRC has the capability of accessing expertise from the EPA s Office of Research and Development, the USACE, as well as private consultants and academic researchers. Information on how to access the SSRC is available through OSRTI s Contaminated Sediments in Superfund Web site at http://www.epa.gov/superfund/resources/sediment/ssrc.htm.

In 2002, EPA established the Contaminated Sediments Technical Advisory Group (CSTAG) to monitor the progress of, and provide advice regarding, a number of large, complex, or controversial contaminated sediment Superfund sites. For most sites, the group meets with the site team several times throughout the site investigation, response selection, and action implementation processes. Involving CSTAG at each major phase of a project provides additional technical support to the project team and ensures consistency with EPA s national sediment policies. General information about CSTAG and site-specific recommendations and responses are available through OSRTI s Contaminated Sediments in Superfund Web site at http://www.epa.gov/superfund/resources/sediment/cstag.htm.

1.5 STATE, TRIBAL, AND TRUSTEE INVOLVEMENT

State cleanup agencies and affected Indian tribes or nations at sediment sites or impacted downstream areas have an important role as co-regulators and/or affected parties and as sources of essential information at sediment sites. States are the lead agency at some sediment sites, or lead the cleanup of land-based source areas or particular operable units within a site. States and Indian tribes are frequently an indispensable source of historic and current information about water body uses, fish consumption patterns, ecological habitat, other sources of contamination within a watershed, and other information useful in characterizing the site and selecting an appropriate remedy. At some sediment sites, states are also owners of aquatic lands, dams, or floodplains. Where this is the case, states have multiple roles at the site. At sediment sites, as for all sites, states (and local and tribal governments where applicable) should be involved early and often in the remedial investigation/feasibility study (RI/FS). Coordination with the state may be especially helpful in the development of the conceptual site model, risk assessment, and remediation goals. Additional coordination during remedial design/remedial action phases is also very important (e.g., an opportunity to consult during the engineering design following remedy selection and on other technical matters related to implementation or monitoring of the remedy). Additional information on coordinating with states and Indian tribes can be found in OSWER Directive

9375.3-03P, *The Plan to Enhance the Role of States and Tribes in the Superfund Program* (U.S. EPA 1998b), and OSWER Directive 9375.3-06P, *Enhancing State and Tribal Role Directive* (U.S. EPA 2001a).

Where there is a potential for natural resource injuries and damages associated with sediment sites, coordination between the remedial and trusteeship roles at the federal, tribal, and state levels is especially important. Several different federal, state, or tribal natural resource trustees may have an interest in decisions concerning contaminated sediment sites and should have an opportunity to be involved throughout the investigation and remedy selection process at sites where they have jurisdiction and interest. The EPA is required to notify natural resource trustees promptly whenever a release of hazardous materials, contaminants, or pollutants may injure natural resources (CERCLA §104 (b)(2)). Trustees may include federal natural resource trustee agencies, such as the U.S. Department of the Interior (DOI), National Oceanic and Atmospheric Administration (NOAA), U.S. Department of Agriculture (USDA) Forest Service, U.S. Department of Defense (DoD), or U.S. Department of Energy (DOE). State agencies and federally recognized tribes may also be natural resource trustees. Where NOAA is the natural resource trustee, project managers should contact the Coastal Resource Coordinators (CRCs) who are assigned to each EPA region (except Regions 7 and 8, where there are no NOAA trust resources). These CRCs are also designated natural resource trustee representatives for marine resources, including migratory fish.

Interests and data needs of the trustees and the EPA may be similar. When trustees are involved, project managers should consult them early in the RI/FS process regarding potential contaminant migration pathways, ecological receptors, and characteristics of the water body and watershed. Sharing information early with federal, tribal, and state trustees (rather than bringing them in later in the process) often leads to more efficient data collection and better coordination of protection of human health and the environment. Information on coordinating with trustees is found in EPA s *ECO Update: The Role of Natural Resource Trustees in the Superfund Process* (U.S. EPA 1992a), in OSWER Directive 9200.4-22A, *CERCLA Coordination with Natural Resource Trustees* (U.S. EPA 1997a), and in OSWER Directive 9285.7-28P, *Ecological Risk Assessment and Risk Management Principles for Superfund Sites* (U.S. EPA 1999b).

1.6 COMMUNITY AND OTHER STAKEHOLDER INVOLVEMENT

Communication and outreach with the community and other stakeholders can pose unique challenges at sediment sites, especially at large sites on publicly used water bodies. Community involvement coordinators often have a critical role as part of the project team at these sites. Sediment sites that span large areas may present barriers to communicating effectively with different communities, local governments, and the private sector along the water body. People who live, work, and play adjacent to water bodies that contain contaminated sediment should receive accurate information about the safety of their activities, and be provided opportunities for involvement in the EPA s decision-making process for sediment cleanup. Community members may have a wide variety of needs and wishes for current and future uses of the water body. Highlights 1-8 and 1-9 list some of the common community concerns about contaminated sediment and risk reduction methods for sediment. These lists are compiled from information provided by Superfund project managers and by the NRC (2001). Project managers should be aware of these potential concerns and others specific to their sites.

Highlight 1-8: Common Community Concerns about Contaminated Sediment

- Human health impacts from eating fish/shellfish, wading, and swimming
- Ecological impacts on wildlife and aquatic species
- Loss of recreational and subsistence fishing opportunities
- Loss of recreational swimming and boating opportunities
- Loss of traditional cultural practices by Indian tribes and others
- Economic effects of loss of fisheries
- Economic effects on development, reduction in property values, or property transferability
- Economic effects on tourism
- Concern whether all contamination sources have been identified
- Increased costs of drinking water treatment, other effects on drinking water, and other water uses
- Loss or increased cost of commercial navigation

Highlight 1-9: Common Community Concerns about Sediment Cleanup

Concerns about MNR	Concerns about In-Situ Capping	Concerns about Dredging and Excavation
• Long time-frame for recovery	• Increased truck or rail traffic	• Increased truck or rail traffic
• Ongoing human and ecological exposure during recovery period	• Loss of resource/harvesting opportunities	• Noise, emissions, and lights at treatment and disposal facilities
• Doubts about effectiveness/spreading of contamination due to flooding/other disturbance	• Increased flooding	• Siting of new disposal facilities
• Extended loss of resources and uses	• Disturbance of aquatic habitat	• Loss of capacity at existing disposal facilities
• Perception of □do nothing□remedy	• Cap material source issues	• Loss of privacy during construction
• Property value/ transferability concerns with leaving significant contamination in place	• Loss of boat anchoring access	• Infrastructure needs on adjacent land
	• Doubts about effectiveness due to cap erosion, disruption, or contaminant migration through cap	• Recreation and tourism impacts
	• Loss of privacy during construction	• Access to private property
	• Recreation and tourism impacts during construction	• Property values near dredging, treatment and disposal facilities
	• Property value/transferability concerns with leaving significant contamination in place	• Disturbance of aquatic habitat
		• Resuspension/spreading contamination during dredging

Existing community involvement and sediment guidance from EPA and the NRC offer some guidelines for involving the community in meeting these and other concerns, as identified in Highlight 1-10.

Highlight 1-10: Community Involvement Guidance and Advice

EPA Office of Solid Waste and Emergency Response on Community Involvement (most available at http://www.epa.gov/superfund/action/community/index.htm):

- *Contaminated Sediments: Impacts and Solutions Video and Presenters Manual* (U.S. EPA 2005b)
- *Early and Meaningful Community Involvement* (U.S. EPA 2001b)
- *Superfund Community Involvement Toolkit* (U.S. EPA 2003a)
- *Community Advisory Group Toolkit for EPA Staff* (U.S. EPA 1997b)
- *The Model Plan for Public Participation*, National Environmental Justice Advisory Council (U.S. EPA 1996b)
- *Incorporating Citizen Concerns into Superfund Decision Making* (U.S. EPA 2001c)

RCRA Community Involvement Guidance (available at http://www.epa.gov/epaoswer/hazwaste/ca/guidance.htm; see list under "Public Involvement/Communication"):

- *RCRA Public Participation Manual*
- *RCRA Expanded Public Participation Rule* (60 *FR* 63417-34)
- *RCRA Corrective Action Workshop Communication Tools*

Office of Water on Communication of Fish Consumption Risks and Surveys (available at http://www.epa.gov/ost/fish):

- *Guidance for Conducting Fish and Wildlife Consumption Surveys* (U.S. EPA 1998c)
- *National Risk Communication Conference Held in Conjunction with the Annual National Forum on Contaminants in Fish* (May 6-8, 2001, conference proceedings available at http://www.epa.gov/waterscience/fish/proceedings.html)

National Research Council:

- *A Risk-Management Strategy for PCB-Contaminated Sediments, Chapter 4, Community Involvement* (NRC 2001)

Considering existing EPA guidance, and advice from the NRC and others, the three points below highlight some of the most critical aspects of community involvement at sediment sites.

Point 1. Involve the Community and Other Stakeholders Early and Often

In addition to the provisions addressing stakeholder involvement in CERCLA §117 and the NCP, one of EPA's eleven principles for managing risk of contaminated sediment is to involve the community early and often. This is an important principle in relation to other stakeholders as well, including local

governments, port authorities, and PRPs. The mission of the Superfund and RCRA community involvement programs is to advocate and strengthen early and meaningful community participation during Superfund cleanups. Planning for community involvement at contaminated sediment sites should begin as early as the site discovery and site assessment phase and continue throughout the entire Superfund process. As noted by the NRC (2001), community involvement will be more effective and more satisfactory to the community if the community is able to participate in or directly contribute to the decision-making process. Passive feedback about decisions already made by others is not what is referred to as community or stakeholder involvement. Early involvement allows necessary input from communities and other stakeholders and facilitates more comprehensive identification of issues and concerns early in the site management process.

Early community involvement enables EPA to learn what stakeholders, especially community members, think are important exposure pathways of the contamination and of potential response options. Available materials about community involvement in the risk assessment process include *A Community Guide to Superfund Risk Assessment – What's it All about and How Can You Help?* (U.S. EPA 1999c). Although the regulators have the responsibility to make the final cleanup decision at CERCLA and RCRA sites, early and frequent community involvement helps the regulators understand differing views and allows the regulators to factor these views into their decisions.

Point 2. Build an Effective Working Relationship with the Community and Other Stakeholders

In addition to the provisions addressing public outreach in CERCLA §117 and the NCP, building partnerships with key community groups, the private sector, and other interested parties is critical to implementing a successful outreach program. Involving communities by fostering and maintaining relationships can lead to better site decisions and faster cleanups. Referring specifically to PCB-contaminated sites, but with application to all sediment sites, the NRC (2001) report recommended that community involvement at PCB-contaminated sediment sites should include representatives of all those who are potentially at risk due to contamination, although special attention should be given to those most at risk.

Participants at EPA's 2001 *Forum on Managing Contaminated Sediments at Hazardous Waste Sites* (U.S. EPA 2001d) offered the following ideas, among others, for building effective working relationships with communities and other stakeholders at sediment sites:

- Create realistic expectations up front for both public involvement and sediment cleanup;
- Where possible, instead of asking for extra meetings, ask for time at existing community meetings;
- Use store-front on-site offices for public information when possible;
- Be aware of tribal cultural and historic sites, not all of which are registered or are on tribal land;
- Minimize jargon when speaking and writing for the public;
- Use independent facilitators for public meetings when needed;

- Include broad representation of the community;
- Look for areas where you can act on input from the community; and
- Encourage continuity of membership as much as possible.

A complete list of forum presentation materials is available through EPA s Superfund Web site at http://www.epa.gov/superfund/resources/sediment/meetings.htm.

Point 3. Provide the Community with the Resources They Need to Participate Effectively in the Decision-Making Process

In addition to the provisions addressing public outreach in CERCLA 117 and the NCP, project managers should ensure that community members have access to the tools and information they need to participate throughout the cleanup process. Educational materials should be accessible, culturally sensitive, relevant, timely, and translated when necessary. One potential resource is a video prepared by EPA s Superfund office, which explains to communities the general remedial options for sediment (U.S. EPA 2005b).

Contaminated sediment sites often involve difficult technical issues. It is especially important to give community members opportunities to gain the technical knowledge necessary to become informed participants. Project managers should provide technical information to communities in formats that are accessible and understandable. The EPA has a number of resources available to help make large volumes of complex data more easily understandable. These resources are often valuable communication tools not only with the community, but also within the EPA and between cooperating agencies. An example includes the graphics and scenario analysis capabilities of Region 5 Fully Integrated Environmental Location Decision Support (FIELDS). FIELDS began as an effort to solve contaminated sediment problems more effectively in and around the Great Lakes and is applied in other regions as well. Information about FIELDS is available at http://www.epa.gov/region5fields.

Information about Superfund community services is available through EPA s Superfund Web site at http://www.epa.gov/superfund/action/community/index.htm. This Web site provides information on community advisory groups (CAGs), EPA s Technical Assistance Grant (TAG) program, and the Technical Outreach Services for Communities (TOSC) program. The TOSC program uses university educational and technical resources to help community groups understand the technical issues involving hazardous waste sites in their communities. The Superfund statute provides for only one TAG per site. At very large sites with diverse community interests, communities may choose to form a coalition and apply for grant funding as one entity. The coalition would need to function as a nonprofit corporation for the purpose of participating in decision making at the site. Individual organizations may choose to appoint representatives to a steering committee that decides how TAG funds should be allocated, and defines the statement of work for the grant. The coalition group may hire a grant administrator to process reimbursement requests to the EPA and to ensure consistent management of the grant. In some cases, EPA regional office award officials may waive a group s $50,000 limit if site characteristics indicate additional funds are necessary due to the nature or volume of site-related information.

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2.0 REMEDIAL INVESTIGATION CONSIDERATIONS

The main purpose of investigating contaminated sediment, as with other media, is generally to determine the nature and extent of contamination to determine if there are unacceptable risks that warrant a response and, if so, to evaluate potential remedies. Investigations may be conducted by a number of different parties under a number of different legal authorities. Most of this chapter presents general information of potential use to any investigator. However, the language and program-specific references are drawn from the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) program, and at times, from the Resource Conservation and Recovery Act (RCRA) program. This chapter is not a comprehensive guide to site characterization and risk assessment of sediment sites, but it does attempt to summarize many of the most important considerations.

Under CERCLA, the investigation process is known as a remedial investigation (RI). Under RCRA, the investigation process is known as a RCRA facility investigation. The RI process is described in the U.S. Environmental Protection Agency s (EPA s) *Guidance for Conducting Remedial Investigations and Feasibility Studies under CERCLA* (U.S. EPA 1988a, also referred to as the RI/FS Guidance). The investigative process in a RCRA corrective action is best described in Office of Solid Waste and Emergency Response (OSWER) Directive 9902.3-2A, *RCRA Corrective Action Plan* (U.S. EPA 1994a), and the May 1, 1996 Advanced Notice of Proposed Rulemaking [(ANPR) 61 *Federal Register* (*FR*) 19447]. This chapter supplements these existing guidances by offering brief sediment-specific guidance about site characterization, risk assessment, and other investigation issues unique to sediment. More detailed guidance concerning site characterization is beyond the scope of this document, but may be developed as needed in the future.

2.1 SITE CHARACTERIZATION

The site characterization process for a contaminated sediment site should allow the project manager to accomplish the following general goals, at a scale and complexity appropriate to the site:

- Identify and quantify the contaminants present in sediment, surface water, biota, flood plain soils, and in some cases, ground water;
- Understand the vertical and horizontal distribution of the contaminants within the sediment and flood plains;
- Identify the sources of historical contamination and quantify any continuing sources;
- Understand the geomorphological setting and processes (e.g., resuspension, transport, deposition, weathering) affecting the stability of sediment;
- Understand the key chemical, and biological processes affecting the fate, transport, and bioavailability of contaminants;
- Identify the complete or potentially complete human and ecological exposure pathways for the contaminants;

- Identify current and potential future human and ecological risks posed by the contaminants;

- Collect data necessary to evaluate the potential effectiveness of natural recovery, in-situ capping, sediment removal, and promising innovative technologies; and

- Provide a baseline of data that can be used to monitor remedy effectiveness in all appropriate media (generally sediment, water, and biota).

The project manager, in consultation with technical experts and stakeholders, should develop site-specific investigation goals that are of an appropriate scope and complexity for the site. Systematic planning, dynamic work strategies, and, where appropriate, real-time measurement technologies may be useful at sediment sites. Combined, these three strategies are known as the triad approach, described on EPA s Innovative Technologies Web site at http://www.cluin.org/triad (although the term triad is the same, this approach should not be confused with the approach to ecological risk assessment known by the same name). This approach attempts to summarize the best current practices in site characterization to collect the correct data, improve confidence in results, and save cost. The triad approach resources also include EPA (2003b), Crumbling (2001), and Lesnick and Crumbling (2001).

Data collection during the remedial investigation frequently has multiple uses, including human health and ecological risk assessment, identification of potential early actions, and remedy decision-making. It is important to consult as many data users as possible (e.g., risk assessors, modelers, as well as quality assurance/quality control (QA/QC) experts) early in the scoping process and throughout data collection.

Data should be of a type, quantity, and quality to meet the objectives of the project. The EPA s data quality objective (DQO) process is one method to achieve this, as described below. Where other agencies (e.g., natural resource trustee agencies, state remediation agencies, and health departments) have an interest at the site, they should be consulted concerning decisions about DQOs so that collected data can serve multiple purposes, if possible. In addition, the community and other stakeholders [e.g., local governments and potentially responsible parties (PRPs)] should be consulted in these decision as appropriate.

2.1.1 Data Quality Objectives

The EPA s DQO process is intended to help project managers collect data of the right type, quality, and quantity to support site decisions. As described in *Guidance for the Data Quality Objective Process* (U.S. EPA 2000a), seven steps generally guide the process. The initial steps help assure that only data important to the decisions that need to be made are collected. The seven DQO process steps include the following, with an example provided in the context of a risk assessment:

1. *State the problem*. Example: There is current exposure of humans to site-related contaminants through eating fish.

2. *Identify the decision*. Example: Is the exposure causing an unacceptable risk?

3. *Identify inputs to the decision*. Examples: What are the appropriate fish species, receptor groups, and consumption rates to evaluate? What existing data are available and what must be collected? What is the toxicity of the contaminants to all receptor groups?

4. *Define boundaries of study.* Example: For purposes of the human health risk assessment, should the water body and the human population each be considered as a whole or in subparts?

5. *Develop a decision rule.* Example: If exposure at the upper 95 percent confidence limit for fish consumption of the recreational fisher population to the mean contaminant concentration of any one of the three most popular fish species exceeds a cancer risk range of 10^{-6} to 10^{-4} or a Hazard Index of 1, risk will be considered unacceptable.

6. *Specify limits on decision errors*. Example: What levels of uncertainty are acceptable for this decision, considering both false positive and false negative errors?

7. *Optimize the design for obtaining data.* Example: What is the most resource-effective fish sampling and analysis design for generating data that will meet the data quality objectives?

Similar hypotheses could be established for evaluating each remedial alternative being considered for the site, and for evaluating the effectiveness of the selected alternative. The way in which the process is followed may vary depending on the decision to be made, from a thought process to a rigorous statistical analysis. Additional guidance provided in *EPA Requirements for Quality Assurance Project Plans* [(QAPPs), U.S. EPA 2001e) describes how DQOs are incorporated into QAPPs.

2.1.2 Types of Data

The types of data the project manager should collect are determined mostly by the following information needed to:

- Develop the conceptual site model;
- Evaluate sediment and contaminant fate and transport;
- Conduct the human health and ecological risk assessments;
- Evaluate the effectiveness of source control;
- Evaluate potential remedies;
- Document baseline conditions prior to implementation of the remedy; and
- Design and implement the selected remedy.

Highlight 2-1 lists some general types of physical, chemical, and biological data that a project manager should consider collecting when characterizing a sediment site. The project manager should

understand the importance of historical changes in some of these characteristics (e.g., water body bathymetry or contaminant distributions in surface and subsurface sediment, water, and biota). It may also be important to understand how characteristics change seasonally, and under various flow and temperature conditions. The relative importance of these types of data variabilities is dependent on the site. It is frequently important to understand the properties affecting the mixing zone or biologically active zone of sediment. Contaminants in the biologically active layer of the surface sediment at a site often drive exposure, and reduction of surface sediment concentrations may be necessary to achieve risk reduction. While sediment sites typically demand more types of data for effective characterization than other types of sites, the type and quantity of data required should be geared to the complexity of the site and the weight of the decision. In addition, the data acquisition process should not prevent early action to reduce risk when appropriate.

Site characterization should include collection of sufficient baseline data to be used to compare to monitoring data collected during and following implementation of the remedy in a statistically defensible manner. Additional sampling could be needed during remedial design, however, to establish reliable baseline data for the monitoring program. Chapter 8, Remedial Action and Long-Term Monitoring, provides a discussion of effective monitoring programs, much of which is also useful during the remedial investigation.

At this time, polychlorinated biphenyls (PCBs) are among the most common contaminants of concern at contaminated sediment sites. The term PCB refers to a group of 209 different chemicals, called PCB congeners, sharing a similar structure. Aroclors are commercial mixtures of PCB congeners and weathering of an Aroclor after release into the environment results in a change in its congener composition (National Research Council, (NRC 2001). EPA s Office of Water *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, Volume 1, Fish Sampling and Analysis, Third Edition* (U.S. EPA 2000b), notes that individual PCB congeners may be preferentially enhanced in environmental media and in biota.

Characterizing PCB risk on a congener-specific basis allows for an accounting of the differences in physiochemical, biochemical, and toxicological behavior of the different congeners in type and magnitude of effects and, therefore, in risk calculations. Although Aroclor analysis can be useful for initial assessment of PCB concentrations, for risk assessment purposes, NRC recommends that PCB sites be characterized on the basis of specific PCB congeners and the total mixture of congeners found at each site (NRC 2001). EPA currently provides congener-specific analyses through its Non-Routine Program under the Contract Laboratory Program (CLP), but it may, in the future, be available through its CLP routine analytical services. However, to the extent that PCB congener-specific data are determined useful at a site, the project manager should not assume this necessarily needs to be done for all samples collected. At times, only a subset of samples or sampling events may need congener analysis. Deciding how best to characterize a PCB site is a complex issue due in part to issues related to dioxin-like PCBs, the lack of congener-specific toxicological data, the need for comparing present and previously collected data, and the cost of congener-specific analyses. The decision about what method or methods to use for PCB analysis should be made on a site-specific basis.

Highlight 2-1: Example Site Characterization Data for Sediment Sites

Physical	Chemical	Biological
• Sediment particle size/distribution and mineralogy in cores	• Near-surface contaminant concentrations in sediment	• Sediment toxicity
• In-situ porosity/bulk density	• Contaminant profiles in sediment cores	• Extent of recreational/commercial harvesting of fish/shellfish for human consumption
• Bearing strength	• Contaminant concentrations (especially metals) in biota tissue, ground water, and pore water	• Extent of predators dependent on aquatic food chain (e.g., mink, otter, kingfisher, heron)
• Specific gravity	• Total organic carbon (TOC) in sediment	• Abundance/diversity of bottom-dwelling species and fishes
• Salinity profile of sediment cores	• Dissolved, suspended, and colloidal contaminant concentrations in surface water	• Abundance/diversity of emergent and submerged vegetation
• Geometry/bathymetry of water body	• Simultaneously extracted metals (SEM) and acid volatile sulfide (AVS) in sediment	• Habitat stressor analyses
• Turbidity	• Radiometric dating profiles in sediment cores	• Contaminant bioavailability
• Temperature	• Non-contaminant chemical species that may affect contaminant mobility	• Pathological condition, such as presence of tumors in fish
• Sediment resuspension and deposition rates	• Oxidation-reduction profile of sediment cores	• Presence of indicator species
• Depth of mixing layer/ degree and depth of bioturbation	• pH profile in sediment cores	
• Geophysical survey results	• Carbon/nitrogen/ phosphorus ratio	
• Flood frequencies, annual and event-driven hydrographs and current velocities	• Non-ionized ammonia concentration in sediment	
• Tidal regime		
• Ground water flow regime and surface water/ground water interaction		
• Ice cover and break-up patterns		
• Water uses causing physical disturbance of sediment		

Currently, metals are also among the most common contaminants of concern at Superfund sediment sites. Concentrations of bulk (total dry weight basis) metals in sediment alone are typically not good measures of metal toxicity. However, in addition to direct measurement of toxicity, EPA has developed a recommended approach for estimating metal toxicity based on the bioavailable metal fraction, which can be measured in pore water and/or predicted based on the relative sediment concentrations of acid volatile sulfide (AVS), simultaneously extracted metals (SEM), and total organic carbon (TOC) (U.S. EPA 2005c). Both AVS and TOC are capable of sequestering and immobilizing a range of metals in sediment.

2.1.3 Background Data

Where site contaminants may also have natural or anthropogenic (man-made) non-site-related sources, it may be important to establish background or reference data for a site. When doing so, project managers should consult EPA s *Role of Background in the CERCLA Cleanup Program* (U.S. EPA 2002b), the *EPA ECO Update - The Role of Screening-Level Risk Assessments and Refining Contaminants of Concern in Baseline Ecological Risk Assessments* (U.S. EPA 2001f), and *Guidance for Comparing Background and Chemical Concentrations in Soil for CERCLA Sites* (U.S. EPA 2002c). Although the latter is written specifically for soil, many of the concepts may be applicable to contaminant data for sediment and biota. It should be noted that a comprehensive investigation of all background substances found in the environment usually will not be necessary at CERCLA sites. For example, radon background samples would not be normally collected at a chemically contaminated site unless radon, or its precursor was part of the CERCLA release.

Where applicable, project managers should consider continuing atmospheric and other background contributions to sites to adequately understand contaminant sources and establish realistic risk reduction goals (U.S. EPA 2002b). For baseline risk assessments, EPA recommends an approach that generally includes the evaluation of the contaminants that exceed protective risk-based screening concentrations, including contaminants that may have natural or anthropogenic sources on and around the Superfund site under evaluation. When site-specific information demonstrates that a substance with elevated concentrations above screening levels originated solely from natural causes (i.e., is a naturally occurring substance and not release-related), these contaminant normally does not need to be carried through the quantitative analysis. However, these contaminants should be generally discussed in the risk characterization summary so that the public is aware of its existence. The presence of naturally occurring substances above screening levels may indicate a potential environmental or health risk, and that information should be discussed at least qualitatively in the document. If data are available, the contribution of background to site conditions should be distinguished (U.S. EPA 2002b). This approach is designed to ensure a thorough characterization of risks associated with hazardous substances, pollutants, and contaminants at sites (U.S. EPA 2002b).

For risk management purposes, understanding whether background concentrations are high relative to the concentrations of released hazardous substances, pollutants, and contaminants may help risk managers make decisions concerning appropriate remedial actions (U.S. EPA 2002b). Generally, under CERCLA, cleanup levels are not set at concentrations below natural or anthropogenic background levels (U.S. EPA 1996a, 1997c, 2000c). If a risk-based remediation goal is below background concentrations, the cleanup level for that chemical may be established based on background concentrations.

In cases where area-wide contamination may pose risks, but these risks are not appropriate to address under CERCLA, EPA may be able to help identify other programs or regulatory authorities that are able to address the sources of area-wide contamination, particularly anthropogenic sources (U.S. EPA 1996a, 1997c, 2000c). In some cases, as part of a response to address CERCLA releases of hazardous substances, pollutants, and contaminants, EPA may also address some of the background contamination that is present on a site due to area-wide contamination.

2.2 CONCEPTUAL SITE MODELS

A conceptual site model (CSM) generally is a representation of the environmental system and the physical, chemical, and biological processes that determine the transport of contaminants from sources to receptors. For sediment sites, perhaps even more so than for other types of sites, the CSM can be an important element for evaluating risk and risk reduction approaches. The initial CSM typically is a set of hypotheses derived from existing site data and knowledge gained from other sites. Natural resource trustee agencies and other stakeholders may have information about the ecosystem that is important in developing the conceptual site model and it is recommended that they have input at this stage of the site investigation. This initial model can provide the project team with a simple understanding of the site based on available data. Information gaps may be discovered in development of the CSM that support collection of new data.

Essential elements of a CSM generally include information about contaminant sources, transport pathways, exposure pathways, and receptors. Summarizing this information in one place usually helps in testing assumptions and identifying data gaps and areas of critical uncertainty for additional investigation. The site investigation is, in essence, a group of studies conducted to test the hypotheses forming the conceptual site model and turning qualitative descriptions into quantitative descriptions. The initial conceptual model should be modified to document additional source, pathway, and contaminant information that is collected throughout the site investigation. Project managers should also be aware of the spatial and temporal dimensions to the processes depicted in a CSM. Although these are difficult to represent in static graphical form, it is important to consider the relevance and role of these dimensions when using the CSM and developing hypotheses or inferences from them.

A good CSM can be a valuable tool in evaluating the potential effectiveness of remedial alternatives. As noted in the following section on risk assessment, the CSM should capture in one place the pathways remedial actions are designed to interdict to reduce exposure of human and ecological receptors to contaminants. Typical elements of a CSM for a sediment site are listed in Highlight 2-2.

Project managers may find it useful to develop several conceptual site models that highlight different aspects of the site. At complex sediment sites, often three conceptual site models are developed: 1) sources, release and media, 2)human health, and 3) ecological receptors. For sites with more than one contaminant that are driving the risks, especially if they behave differently in the environment (e.g., PCBs vs. metals), it is often useful to develop a separate CSM for different contaminants or groups of contaminants. Highlight 2-3, Highlight 2-4, and Highlight 2-5 present examples that focus on ecological and human health threats.

Highlight 2-2: Typical Elements of a Conceptual Site Model for Sediment

Sources of Contaminants of Concern:

- Upland soils
- Floodplain soils
- Surface water
- Ground water
- Non-aqueous phase liquids (NAPL) and other source materials
- Sediment "hot spots"
- Outfalls, including combined sewer outfalls and storm water runoff outfalls
- Atmospheric contaminants

Exposure Pathways for Humans:

- Fish/shellfish ingestion
- Dermal uptake from wading, swimming
- Water ingestion
- Inhalation of volatiles

Exposure Pathways for Biota:

- Fish/shellfish/benthic invertebrate ingestion
- Incidental ingestion of sediment
- Direct uptake from water

Contaminant Transport Pathways:

- Sediment resuspension
- Surface water transport
- Runoff
- Bank erosion
- Ground water advection
- Bioturbation
- Food chain

Human Receptors:

- Recreational fishers
- Subsistence fishers
- Waders/swimmers/birdwatchers
- Workers and transients

Ecological Receptors:

- Benthic/epibenthic invertebrates
- Bottom-dwelling/pelagic fish
- Mammals and birds (e.g., mink, otter, heron, bald eagle)

2.3 RISK ASSESSMENT

Consistent with the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), a human health risk assessment and an ecological risk assessment should be performed at all contaminated sediment sites. In addition to assessing risks due to contaminated sediment, in many cases, risks from soil, surface water, ground water and air pathways may need to be evaluated as well. One of the outputs from the risk assessment should be an understanding of the relative importance or contribution of the pathways depicted in the conceptual site model to actual risk. This understanding is generally key to making informed decisions about which remedial alternative to implement at a site.

Generally, the human health risk assessment should consider the cancer risks and non-cancer health hazards associated with ingestion of fish and other biota inherent to the site (e.g., shellfish, ducks); dermal contact with and incidental ingestion of contaminated sediment; inhalation of volatilized contaminants; swimming; and possible ingestion of river water if it is used as a drinking water supply. Separate analyses should also consider risks from exposure to floodplain soils and may include direct contact, ingestion, and exposures to homegrown crops, beef, and dairy products where appropriate. The relevance and importance of each pathway to actual risks will vary with different contaminants or contaminant classes at a site. In addition, the risk assessment should include an analysis of the risks that may be introduced due to implementation of remedial alternatives (see Section 2.3.3, Risks from Remedial Alternatives). As with all remedial investigation (RI) and feasibility study (FS) data collection efforts, the scope of the assessments should be tailored to the complexity of the site and how much information is needed to reach and support a risk management decision. It is important to involve the risk

assessors early in the process to ensure that the information collected is appropriate for use in the risk assessment.

Screening and baseline risk assessments are designed to evaluate the potential threat to human health and the environment in the absence of any remedial action. Generally, they provide the basis for determining whether remedial action is necessary as well as the framework for developing risk-based remediation goals. Risk assessments should also provide information to evaluate risks associated with implementing various remedial alternatives that may be considered for the site. Detailed guidance on performing human health risk assessments is provided in a number of documents, available through EPA s Superfund Risk Assessment Web site at http://www.epa.gov/oswer/riskassessment/risk_superfund.htm. The *Risk Assessment Guidance for Superfund* (U.S. EPA 1989, also referred to as RAGS), provides a basic plan for developing human health risk assessments. Specific guidance on the standardized planning, reporting, and review of risk assessments is available at http://www.epa.gov/oswer/riskassessment/ragsd/index.htm.

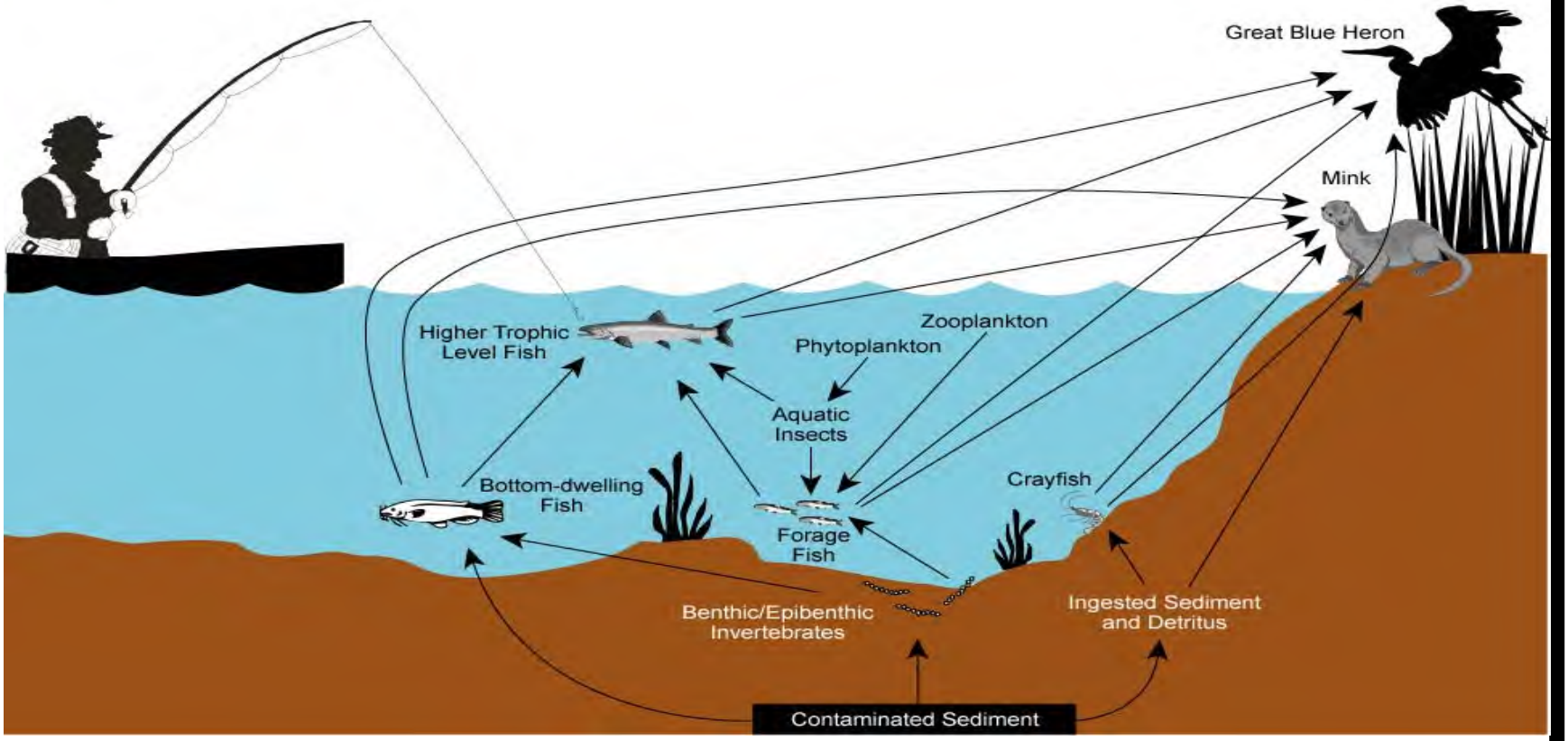
Detailed guidance on performing ecological risk assessments is provided in *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessment* (U.S. EPA 1997d, also referred to as ERAGS). In addition, OSWER Directive 9285.7-28P, *Ecological Risk Assessment and Risk Management Principles for Superfund Sites* (U.S. EPA 1999b), provides risk managers with several principles to consider when making ecological risk management decisions. As stated in the *Role of the Ecological Risk Assessment in the Baseline Risk Assessment* (U.S. EPA 1994b), the purpose of the ecological risk assessment is to 1) identify and characterize the current and potential threats to the environment from a hazardous substance release, 2) evaluate the ecological impacts of alternative remediation strategies, and 3) establish cleanup levels in the selected remedy that will protect those natural resources at risk.

Although not EPA guidance, project managers may find useful the Navy guidance *Implementation Guide for Assessing and Managing Contaminated Sediment at Navy Facilities*, which provides information on performing human health and ecological risk assessments at contaminated sediment sites [U.S. Naval Facilities Engineering Command (FEC) 2003].

2.3.1 Screening Risk Assessment

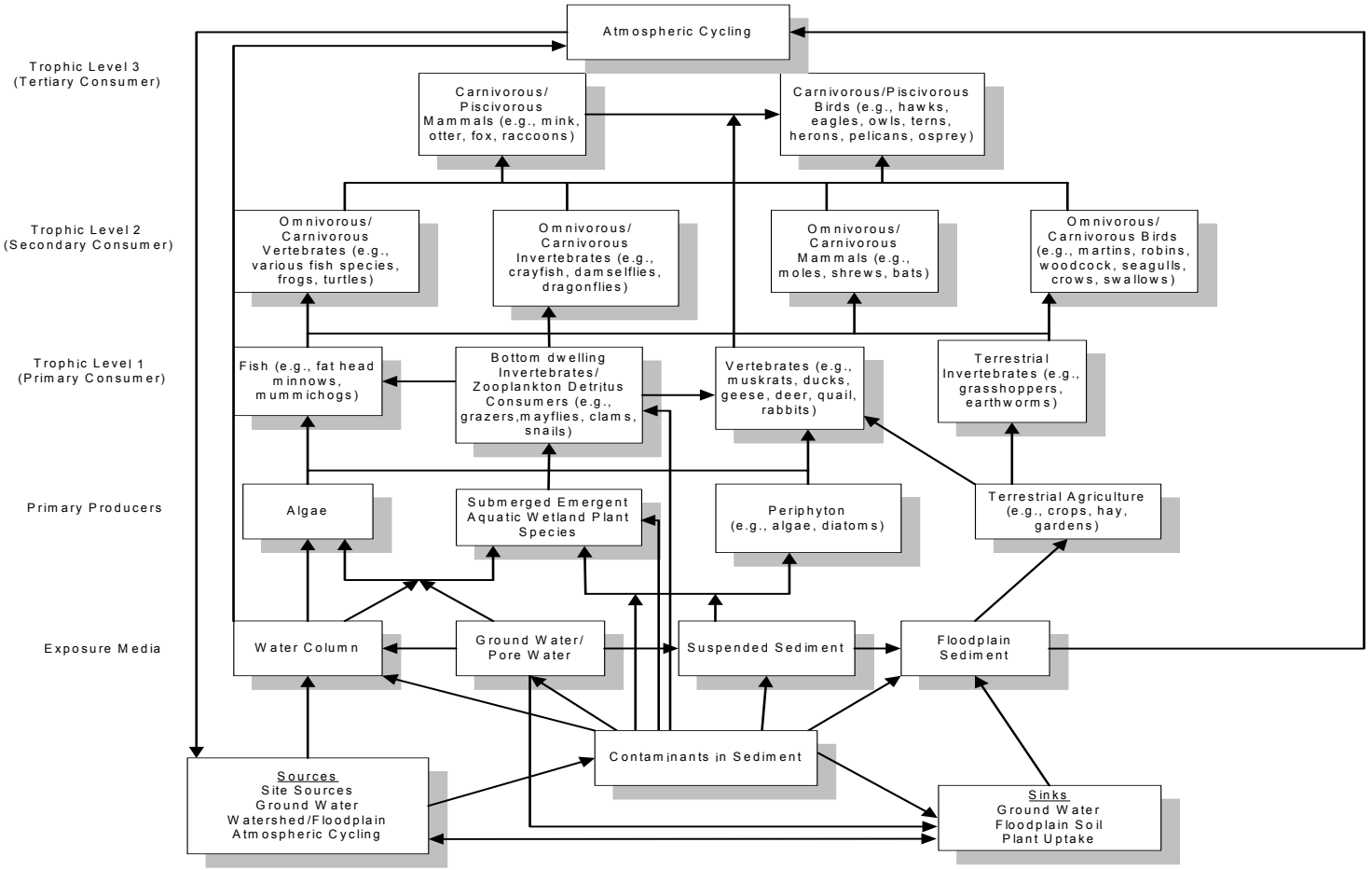
A screening risk assessment typically is performed to identify the contaminants of potential concern (COPCs) and the portions of a site that may present an unacceptable risk to human health or the environment. Currently, there are no widely accepted sediment screening values for human health risk from either direct contact with sediment or from eating fish or shellfish, although research is ongoing. For floodplain and beach soils, human health soil screening levels may be used. Widely accepted screening values do exist for ecological risk from direct toxicity, although, similar to the situation for human health risk, screening values for risk to wildlife and fish from bioaccumulative contaminants have not yet been fully developed. Each of these issues is discussed further below. In cases where screening levels do exist, or may be developed in the future, it is very important for project managers to keep in mind that screening values are not designed to be used as default cleanup levels and generally should not be used for that purpose. In evaluating whether specific screening values are appropriate for a particular site, project managers should consider whether the source of the data used to develop the screening values are relevant to site conditions, and understand the methods by which the screening values were derived. Project managers may also find ecological screening values or human health screening level exposure assumptions useful for evaluating whether detection levels for sediment analytical work are sufficiently low to be useful for risk assessment.

Highlight 2-3: Sample Pictorial-Style Conceptual Site Model Focusing on Human and Ecological Threats

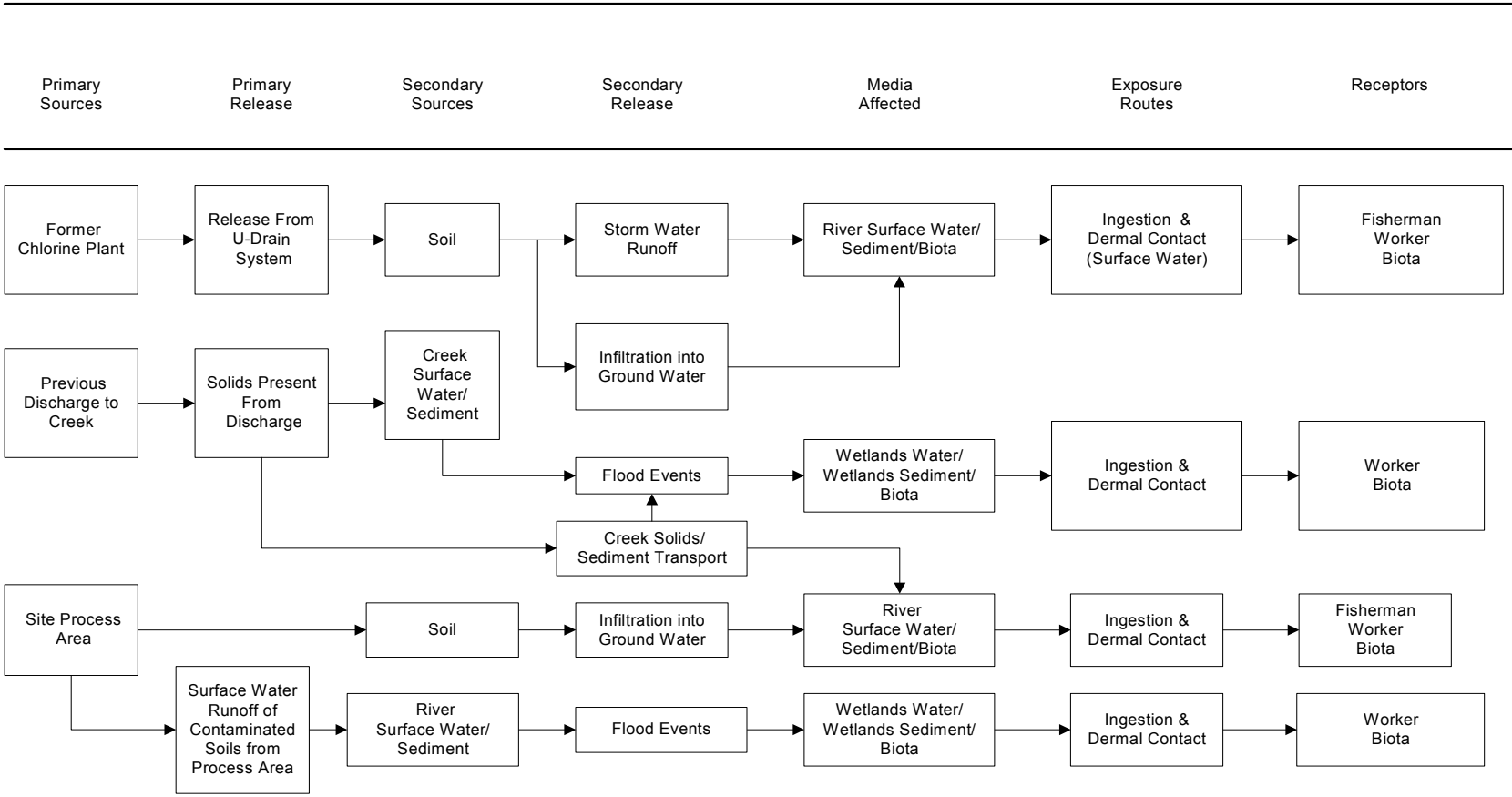


Source: Adapted from EPA Region 5, Sheboygan Harbor and River Site

Highlight 2-4: Sample Conceptual Site Model Focusing on Ecological Threats



Highlight 2-5: Sample Conceptual Site Model Focusing on Human Health Threats



When evaluating human health risks from direct contact with sediments and from bioaccumulative contaminants in fish and shellfish, RAGS (U.S. EPA 1989), and other risk guidance discussed above, should be followed to identify the COPCs that may present an unacceptable risk. In general, if bioaccumulative contaminants are found in biota at levels above site background, they should not be screened out and should be carried into the baseline risk assessment.

When evaluating human health risks from direct contact with floodplain or beach soils, OSWER and several regions have soil screening values that may be useful. Human health soil screening levels (SSLs) for residential and industrial properties are available through EPA s Superfund Web site at http://www.epa.gov/superfund/resources/soil, which provide a generic approach and exposure assumptions for evaluation of risks from direct contact with soil.

When screening ecological risk to benthic biota from direct toxicity, project managers should consult EPA s Eco-Updates *EcoTox Thresholds* (U.S. EPA 1996c) and *The Role of Screening-Level Risk Assessment and Refining Contaminants of Concern in Baseline Ecological Risk Assessments* (U.S. EPA 2001f), which describes the process of screening COPCs. The EPA s equilibrium-partitioning sediment benchmarks are available at http://www.epa.gov/nheerl/publications/, and the Superfund program s Ecotox Thresholds (ETs) are available at http://www.epa.gov/oswer/riskassessment/pdf/eco_updt.pdf can be used as screening values for risk to benthic biota from direct toxicity. Other published sediment guidelines [e.g., National Oceanic and Atmospheric Administration (NOAA) Screening Quick Reference Tables (SQuiRTs), http://response.restoration.noaa.gov/cpr/sediment/squirt/squirt.html] can also be used as screening values. Table 3-1 in the Navy guidance (U.S. Navy FEC 2003) also provides a list of citations for ecological screening values for sediment.

When screening ecological risks to terrestrial receptors from contaminated floodplain soils, the OSWER Directive 9285.7-55, *Guidance for Developing Ecological Soil Screening Levels* [(Eco-SSLs), U.S. EPA 2003c, http://www.epa.gov/oswer/riskassessment/ecorisk/ecossl.htm] should be used. Eco-SSLs for some receptors have been developed for aluminum, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, copper, dieldrin, iron, lead, manganese, nickel, pentachlorophenol, selenium, trinitrotoluene (TNT), and zinc. Screening values for dichloro diphenyl trichlorethane (DDT), polycyclic aromatic hydrocarbons (PAHs), silver, and vanadium are currently under development.

For ecological risk to wildlife or fish from food chain effects, widely accepted screening values have not yet been fully developed. As for the human health risk assessment, if bioaccumulative contaminants are found in biota at levels above site background, they generally should not be screened out and should be carried into the baseline risk assessment for ecological risk as well.

2.3.2 Baseline Risk Assessment

At contaminated sediment sites with bioaccumulative contaminants, the human health exposure pathway driving the risk is usually ingestion of biota, most commonly the ingestion of fish by recreational anglers and sometimes by subsistence anglers. However, depending on the contaminant and the use of the site there can also be significant risks from direct contact with the sediment, water, or floodplain soils, through incidental ingestion and dermal contact.

Generally, the ecological risk assessment should consider the risks to invertebrates, plants, fish and wildlife from direct exposure and from food chain expsoures. The selection of appropriate site-

specific assessment endpoints is a critical component of the ecological risk assessment. Once assessment endpoints have been selected, testable hypotheses and measurement endpoints can be developed to evaluate the potential threat of the contaminants of potential concern to the assessment endpoints. PCBs, for example, bioaccumulate in food chains and can diminish reproductive success in upper trophic level species (e.g., mink, kingfishers) exposed to contaminants through their diet. Therefore, reduced reproductive success in fish-eating birds and mammals may be an appropriate assessment endpoint. An appropriate measurement endpoint in this case might be contaminant concentrations in fish or in the sediment where the concentrations in these media can be related to reproductive effects in the top predator that eats the fish. The sediment concentration range associated with an acceptable level of reproductive success usually would constitute the remediation goal.

2.3.3 Risks from Remedial Alternatives

Although significant attention has been paid to evaluating baseline risks, traditionally less emphasis has been placed on evaluating risks from remedial alternatives, in part because these risks may be difficult to quantify. In 1991, the EPA issued a supplement to the RAGS Guidance, *Risk Assessment Guidance for Superfund: Volume 1 - Human Health Evaluation Manual, Part C, Risk Evaluation of Remedial Alternatives* (U.S. EPA 1991a). Although the 1991 guidance addresses only human health risks, it does note that remedial actions, by their nature, can alter or destroy aquatic and terrestrial habitat, and advises that this potential for destruction or alteration of habitat and subsequent consequences be evaluated and considered during the selection and implementation of a remedial alternative.

The short-term and long-term risks to human health and the environment that may be introduced by implementing each of the remedial alternatives should be estimated and considered in the remedy selection process. Generally, the types, magnitude, and time frames of risk associated with each alternative is extremely site specific. Increases to current risks and the creation of new exposure pathways and risk should be considered.

Implementing a MNR remedy should cause no increase in baseline risks and no creation of new risks, although existing risks may change due to disturbance or significant watershed changes. Implementing in-situ capping might result in increased risk of exposure to contaminants released to the surface water during capping; other community impacts (e.g., accidents, noise, residential or commercial disruption; worker exposure during transport of cap materials and cap placement; and disruption of the benthic community. Existing risks of exposure to contaminants may also occur if contaminants are released through the cap. Implementing dredging or excavation might result in increased risk of exposure to contaminants released during sediment removal, transport, or disposal; other community impacts (e.g., accidents, noise, residential or commercial disruption); worker exposure during sediment removal and handling; and disruption of the benthic community. Risks of exposure to contaminants in residual contamination may also occur. Each of these risks or potential exposure pathways may exist for different periods of time; some are relatively short-lived, while others may exist for a longer period of time. The analysis of risk from implementation of various alternatives is important for remedy selection, and is discussed in more detail in the remedy-specific chapters of this guidance and in Chapter 7, Section 7.4, Comparing Net Risk Reduction.

2.4 CLEANUP GOALS

In selecting the most appropriate remedy for a site, usually it is important to develop clearly defined remedial action objectives (RAOs) and contaminant-specific remediation goals (RGs). RAOs are generally used in developing and comparing alternatives for a site and in providing the basis for developing more specific RGs, which in turn are used by project managers to select final sediment cleanup levels based on the other NCP remedy selection criteria. RAOs, RGs, and cleanup levels are normally dependent on each other and represent three steps along a continuum leading from RI/FS scoping to the selection of a remedial action that will be protective of human health and the environment, meet applicable or relevant and appropriate requirements (ARARs), and provide the best balance among the remaining NCP criteria. Under CERCLA, RAOs and cleanup levels generally are final when the record of decision (ROD) is signed. Where the site is not available for unlimited access and unrestricted use, their protectiveness is reviewed every five years.

2.4.1 Remedial Action Objectives and Remediation Goals

RAOs are intended to provide a general description of what the cleanup is expected to accomplish, and help focus the development of the remedial alternatives in the feasibility study. RAOs are typically derived from the conceptual site model (Section 2.2), and address the significant exposure pathways. RAOs may vary widely for different parts of the site based on the exposure pathways and receptors, regardless of whether these parts of the site are managed separately as operable units under CERCLA. For example, a sediment site may include a recreational area used by fishermen and children, as well as a wetland that provides critical habitat for fish and wildlife. Though both areas may contain similarly contaminated sediment, the different receptors and exposure pathways may lead a project manager to develop different RAOs and RGs for each area that are protective of the different receptors.

The development of RAOs should also include a discussion of how they address all the unacceptable human health and ecological risks identified in the risk assessment. Examples of RAOs specific for sediment sites are included in Highlight 2-6. Sediment sites also may need RAOs for other media (e.g., soils, ground water, or surface water). When developing RAOs, project managers should evaluate whether the RAO is achievable by remediation of the site or if it requires additional actions outside the control of the project manager. For example, complete biota recovery may depend on the cleanup of sources that are regulated under other authorities. The project manager may discuss these other actions in the ROD and explain how the site remediation is expected to contribute to meeting area-wide goals outside the scope of the site, such as goals related to watershed concerns, but RAOs should reflect objectives that are achievable from the site cleanup.

Generally, preliminary remediation goals (PRGs) that are protective of human health and the environment are developed early in the remedial investigation process based on readily available screening levels for both human health and ecological risks (although project managers should be aware that currently available screening levels for sediment may be limited; see Section 2.3.1).

Highlight 2-6: Sample Remedial Action Objectives for Contaminated Sediment Sites

Human Health:

- Reduce to acceptable levels the risks to children and adults from the incidental ingestion of and dermal exposure to contaminated sediment while playing, wading, or swimming at the site
- Reduce to acceptable levels the risks to adults and children from ingestion of contaminated fish and shellfish taken from the site

Ecological Risk:

- Reduce to acceptable levels the toxicity to benthic aquatic organisms at the site
- Reduce to acceptable levels the risks to birds and mammals that feed on fish that have been contaminated from sediment at the site

As more information is generated during the investigation, these PRGs should be replaced with site-specific RGs by incorporating an improved understanding of site conditions (e.g., site-specific information on fish ingestion rates and bioaccumulation of contaminants in sediment into biota; resource use; other human activities), and other site-specific factors, such as the bioavailability of contaminants. The human health and ecological risk assessors should identify appropriate RGs for each contaminant of concern in each medium of significance. RGs for sediment often address direct contact for humans and biota to the sediment as well as bioaccumulation through the food chain. The concentrations of bioaccumulative contaminants in fish typically are a function of both the sediment and water concentrations of the contaminant, and are, to some extent, species-dependent. The development of the sediment RGs may involve a variety of different approaches that range from the simple application of a bioaccumulation factor from sediment to fish or more sophisticated food chain modeling. The method used and the level of complexity in the back calculation from fish to sediment should be consistent with the approaches used in the human health and ecological risk assessments.

RGs should be represented as a range of values within acceptable risk levels so that the project manager may consider the other NCP criteria when selecting the final cleanup levels. For human health, general guidance is available regarding the exposure equations necessary to develop RG concentrations in various media for both cancer risks and non-cancer health hazards (see Section 2.3.) The development of the human health-based RGs should provide a range of risk levels (e.g., 10^{-6}, 10^{-5}, and 10^{-4} and a non-cancer Hazard Index of 1 or less depending on the health end points of the specific contaminants of concern.) The development of the ecologically based RGs should also provide a range of risk levels based on the receptors of concern identified in the ecological risk assessment (see Section 2.3). Human health and ecological RGs should be developed through iterative discussions between the project manager, risk assessor, and modeler or other appropriate members of the team.

2.4.2 Cleanup Levels

At most CERCLA sites, RGs for human health and ecological receptors are developed into final, chemical-specific, sediment cleanup levels by weighing a number of factors, including site-specific uncertainty factors and the criteria for remedy selection found in the NCP at Title 40 Code of Federal Regulations (40 CFR) □300.430. These criteria include long-term effectiveness and permanence;

reduction of toxicity, mobility and volume through treatment; short-term effectiveness; implementability; cost; and state and community acceptance. Chapter 3, Section 3.2, NCP Remedy Selection Criteria discusses these criterion in detail. Regions should note, however, that some states do have chemical and/or biological standards for contaminated sediment (e.g., in development by the State of Washington and others) that may be ARARs at sediment sites.

Uncertainty factors that may be relevant to consider include (among others) the reliability of inputs and outputs of any model used to estimate risks and establish cleanup levels, reliability of the potential approaches to achieve those results, and the likelihood of occurrence for the exposure scenarios being considered. Other technical factors include (among others) limitations of remedial alternatives and detection and quantification limits of contaminants in environmental media. It is especially important to consider both background levels of contamination and what has been achieved at similar sites elsewhere, so that achievable cleanup levels are developed. All of these factors should be considered when establishing final cleanup levels that are within the risk range.

The derivation of ecologically based cleanup levels is a complex and interactive process incorporating contaminant fate and transport processes, toxicological considerations and potential habitat impacts of the remediation alternatives. Before selecting a cleanup level, the project manager, in consultation with the ecological risk assessor, should consider at least the following factors (U.S. EPA 1999b):

- The magnitude of the observed or expected effects of site releases and the level of biological organization affected (e.g., individual, local population, or community);
- The likelihood that these effects will occur or continue;
- The ecological relationship of the affected area to the surrounding habitat;
- Whether the affected area is a highly sensitive or ecologically unique environment; and
- The recovery potential of the affected ecological receptors and expected persistence of the chemicals of concern under present site conditions.

Generally, for CERCLA actions, the ROD should include chemical-specific cleanup levels as provided in the NCP at 40 CFR §300.430(c)(2)(I)(A). The ROD should also indicate the approach that will be used to measure attainment of the cleanup levels and how cleanup levels relate to risk reduction. At many sediment sites, especially but not exclusively those with bioaccumulative contaminants, the attainment of sediment cleanup levels may not coincide with the attainment of RAOs. For example, this may be due to the length of time needed for fish or the benthic community to recover. Where cleanup levels have been achieved but progress towards meeting RAOs is not as expected, the five-year review process, or where appropriate, a similar process conducted before five years, should be used to assess whether additional actions are needed. Consistent with the NCP (40 CFR §300.430(f)(4)(ii)), where contaminants remain present above unlimited use and unrestricted exposure levels, Superfund sites should be reviewed no less than every five years after initiation of the selected remedial action. Chapter 8, Remedial Action and Long-Term Monitoring, provides additional guidance on the information that should be collected for this review to be effective. As explained further in Chapter 8, the need for long-term monitoring is not limited to sites where five-year reviews are required. Most sites where

contaminated sediment has been removed also should be monitored for some period to ensure that cleanup levels and RAOs are met and will continue to be met.

2.5 WATERSHED CONSIDERATIONS

A unique aspect of contaminated sediment sites is their relationship within the overall watershed, or drainage area, in which they are located. Within the watershed there often is a spectrum of issues that the project manager may need to consider. Foremost among them at many sites is to work with the state to ensure that fish consumption advisories are in place and well publicized. In addition, project managers should understand the role of the contaminated water body in the watershed, including the habitat or flood control functions it may serve, the presence of non-site-related contaminant sources in the watershed, and current and reasonably anticipated or desired future uses of the water body and surrounding land.

2.5.1 Role of the Contaminated Water Body

Most water bodies provide important habitat for spawning, migration, or food production for fish, shellfish, birds, and other aquatic and land-based animals. One significant issue is the protection of migratory fish. These are fish such as salmon, shad, and herring that migrate as adults from marine waters up estuaries and rivers to streams and lakes where they spawn. The juveniles spend varying lengths of time in freshwater before migrating to estuarine/marine waters. It can be difficult to evaluate the impact of a particular contaminated sediment site on wide-ranging species that may encounter several sources of contamination along their migratory route. This can be an important consideration when evaluating alternatives and establishing remediation goals for a site, as these fish populations may not show improvement if any link in their migratory route is missing, blocked, or toxic. For migratory species, it may be more appropriate to measure risk and remedy effectiveness in terms of risk to juveniles, or whatever part of the life cycle is spent at the site.

The size, topography, climate, and land use of a watershed, among other factors, may affect characteristics of a water body, such as water quality, sedimentation rate, sediment characteristics, seasonal water flows and current velocities, and the potential for ice formation. For example, watersheds with large wetland areas tend to store flood waters and enable ground water recharge, thereby protecting downstream areas from increased flooding, whereas an agricultural or urbanized watershed may have increased erosion and greater flow during storm events. Watershed changes can result from natural events, such as wildfires, or from human activities such as road and dam construction/removal, impoundment releases, and urban/suburban development. When considering watershed characteristics, it is generally important to consider both current and future watershed conditions.

Some sediment sites are located in watersheds with a large number of historical and ongoing point and non-point sources, from many potentially responsible parties. Where this is the case, it can be especially important to attain expert assistance to plan site characterization strategies that are well suited to the complexity of the issues and designed to answer specific questions. In urban watersheds and others with a large number of ongoing sources, it may be beneficial for a broader group of stakeholders to participate in setting priorities for site characterization and remediation efforts. In these areas, it can be especially important to consider background concentrations when developing remedial objectives and to evaluate the incremental improvement to the environment if an action is taken at a specific site in the watershed. Approaching management of a site within the watershed context may provide an opportunity

to better determine the needs and coordinate the sequence and schedule of cleanup activities in the watershed.

2.5.2 Water Body and Land Uses

Water body uses at sediment sites may include commercial navigation; commercial fisheries, shellfisheries, or aquaculture; boating, swimming, and other forms of recreation; other commercial or industrial uses; recreational or subsistence fishing or shellfishing; and other, less easily categorized uses. Most water bodies used for commercial navigation, such as for shipping channels, turning basins, and port areas, are periodically dredged to conform to the minimum depth for the area prescribed by Congress; such dredging is typically performed or permitted by the U.S. Army Corps of Engineers (USACE). Other commercial or industrial uses of a site may include the presence of gravel pits, drinking water use, and industrial uses of water including cooling, washing, or waste water disposal.

The NCP preamble (55 *FR* 8710) states that both current and future land uses should be evaluated in assessing risks posed by contaminants at a Superfund site and discusses how Superfund remedies should be protective in light of reasonably anticipated future uses. EPA has provided further guidance on how to evaluate future land use in the OSWER Directive 9355.7-04, *Land Use in the CERCLA Remedy Selection Process* (U.S. EPA 1995a, also referred to as the Land Use Guidance). This guidance encourages early discussions with state and local land use planning authorities and the public, regarding reasonably anticipated future uses of properties associated with a National Priorities List (NPL) site. This coordination should begin during the scoping phase of the RI/FS, and ongoing coordination is recommended to ensure that any changes in expectations are incorporated into the remedial process.

There are additional factors the project manager should include in considering anticipated future uses for aquatic sites not specifically addressed in the Land Use Guidance. For example, future use of the site by ecological receptors may be a more important consideration for an aquatic sediment Superfund or RCRA site as compared to an upland terrestrial site. A remediated sediment site may attract more recreational, subsistence, and cultural uses, including fishing, swimming, and boating. Where applicable, the project manager should consider tribal treaty rights to collect fish or other aquatic resources. The project manager should also consider [generally as TBCs (or to be considered), see Chapter 3, Section 3.3 on ARARs] designated uses in the state s water quality standards, priorities established as a result of total maximum daily loads (TMDLs), or pollution reduction efforts under various Clean Water Act (CWA) programs in projecting future waterway uses. In ports and harbors, the project manager should consult master plans developed by port and harbor authorities for projections of future use. The USACE should also be contacted regarding future navigational dredging of federally maintained channels.

There may be more parties to consult about anticipated future use at large sediment sites as opposed to typical upland sites. These parties include the community, environmental groups, natural resource trustees, Indian tribes, the local department of health, as well as local government, port and harbor authorities, and land use planning authorities. As with upland sites, consultation should start at the RI/FS scoping phase and continue throughout the life of the project. Different stakeholders often have divergent and conflicting ideas about future use at the site. Local residents and environmental groups may anticipate future habitat restoration and increased recreational and ecological use while local industrial landowners may project increased shipping and industrial use. The NCP preamble (55 *FR* 8710) states that, in the baseline risk assessment, more than one future use assumption should be considered when decision makers wish to understand the implications of different exposure scenarios.

Especially where there is some uncertainty regarding the anticipated future uses, the project manager should compare the potential risks associated with several use scenarios.

The identification of appropriate future use assumptions during the baseline risk assessment and the feasibility study should allow the project manager to focus on developing protective, practicable, and cost-effective remedial alternatives. In addition, coordination with stakeholders on land and water body uses leads to opportunities to coordinate Superfund or RCRA remediation in conjunction with local development or habitat restoration projects. For example, at some sites the EPA has worked with port authorities to combine Superfund or RCRA remedial dredging with dredging needed for navigation. Others have combined capping needed for Superfund or RCRA remediation with habitat restoration, allowing PRPs to settle natural resource damage claims in conjunction with the cleanup. However, as noted in Chapter 1, Section 1.5, State, Tribal, and Trustee Involvement, whether remediation and restoration are addressed concurrently is a site-specific decision that involves input from a number of different parties.

2.6 SOURCE CONTROL

Identifying and controlling contaminant sources typically is critical to the effectiveness of any Superfund sediment cleanup. Source control generally is defined for the purposes of this guidance as those efforts are taken to eliminate or reduce, to the extent practicable, the release of contaminants from direct and indirect continuing sources to the water body under investigation. At some sediment sites, the original sources of the contamination have already been controlled, but subsequent sources such as contaminated floodplain soils, storm water discharges, and seeps of ground water or non-aqueous phase liquids (NAPLs) may continue to introduce contamination to a site. At sites with significant sediment mobility, areas of higher contaminant concentration may act as continuing sources for less-contaminated areas.

Some sources, especially those outside the boundaries of the Superfund or RCRA site, may best be handled under another authority, such as the CWA or a state program. These types of sites can present an opportunity for partnering with private industry and other governmental entities to identify and control sources on a watershed basis. Water bodies with sources outside the Superfund site can also present a need to balance the desire for watershed-wide solutions with practical considerations affecting a subset of responsible parties. It can be difficult to determine the proper party to investigate sources outside the Superfund site, but the site RI/FS must be sufficient to determine the extent of contamination coming onto the site and its likely effect on any actions at the site. A critical question often is whether an action in one part of the watershed is likely to result in significant and lasting risk reduction, given the probable timetable for other actions in the watershed.

Source control activities are often broad-ranging in scope. Source control may include application of regulatory mechanisms and remedial technologies to be implemented according to ARARs, including the application of technology-based and water quality-based National Pollutant Discharge Elimination System (NPDES) permitting to achieve and maintain sediment cleanup levels. Source control actions may include, among others, the following:

- Elimination or treatment of contaminated waste water or ground water discharges (e.g., installing additional treatment systems prior to discharge);

- Isolation or containment of sources (e.g., capping of contaminated soil) with attendant engineering controls;
- Pollutant load reductions of point and nonpoint sources based on a TMDL;
- Implementation of best management practices (e.g., reducing chemical releases to a storm drain line); and
- Removal or containment of potentially mobile sediment hot spots.

EPA's Contaminated Sediment Management Strategy (U.S. EPA 1998a) includes some discussion of EPA s strategy for abating and controlling sources of sediment contamination. Source control activities may be implemented by state or local governments using combinations of voluntary and mandatory actions.

The identification of continuing sources and an evaluation of their potential to re-contaminate site sediment are often essential parts of site characterization and the development of an accurate conceptual site model, regardless of source areas within the site. When there are multiple sources, it is often important to prioritize sources to determine the relative significance of continuing sources versus on-site sediment in terms of site risks to determine where to focus resources. Where sources are a part of the site, project managers should develop a source control strategy or approach for the site as early as possible during site characterization. Where sources are outside the site, project managers should encourage the development of source control strategies by other authorities, and understand those strategies. Generally, a source control strategy should include plans for identifying, characterizing, prioritizing, and tracking source control actions, and for evaluating the effectiveness of those actions. It is also useful to establish milestones for source control that can be linked with sediment remedial design and cleanup actions. If sources can be substantially controlled, it is normally very important to reevaluate risk pathways to see if sediment actions are still needed. If sources cannot be substantially controlled, it is typically very important to include these ongoing sources in the evaluation of what sediment actions may or may not be appropriate and what RAOs are achievable for the site.

Generally, significant continuing upland sources (including ground water, NAPL, or upgradient water releases) should be controlled to the greatest extent possible before sediment cleanup. Once these sources are controlled, project managers should evaluate the effectiveness of the actions, and should refine and adjust levels of source control, as warranted. In most cases, before any sediment action is taken, project managers should consider the potential for recontamination and factor that potential into the remedy selection process. If a site includes a source that could result in significant recontamination, source control measures will be likely necessary as part of that response action. However, where sediment remediation is likely to yield significant benefits to human health and/or the environment after considering the risks caused by an unaddressed or ongoing source, it may be appropriate to conduct an action for sediment prior to completing all land-based source control actions.

2.7 PHASED APPROACHES, ADAPTIVE MANAGEMENT, AND EARLY ACTIONS

At some sediment sites, a phased approach to site characterization, remedy selection, or remedy implementation may be the best or only practical option. Phasing site characterization can be especially useful when risks are high, yet some important site-specific factors are unknown. Phasing in remedy

selection and implementation may be especially useful at sites where contaminant fate and transport processes are not well understood or the remedy has significant implementation uncertainties. Phasing may also be useful where the effectiveness of source control is in doubt. By knowing the effectiveness of source control prior to implementing sediment cleanups, the risk of having to revisit recontaminated areas is greatly reduced. High remedy costs, the lack of available services and/or equipment, and uncertainties about the potential effectiveness or the risks of implementing the preferred sediment management approach, can also lead to a decision to phase the cleanup. At some sites, it may be advantageous to pilot less invasive or less costly remedial alternatives early enough in the process that performance could be tracked. If performance does not approach desired levels, then more invasive or more costly approaches could be pursued.

Phasing can also be used at large, multi-source, multi-PRP sites with primarily historic contamination where contaminated sediment is still near the sources. At these types of sites, working with a single responsible party to address sediment with higher contaminant concentrations near a specific source may be an effective risk reduction measure, while the more complex decision making for the rest of the site is ongoing.

Project managers are encouraged to use an adaptive management approach, especially at complex sediment sites to provide additional certainty of information to support decisions. In general, this means testing of hypotheses and conclusions and reevaluating site assumptions as new information is gathered. This is an important component of updating the conceptual site model. For example, an adaptive management approach might include gathering and evaluating multiple data sets or pilot testing to determine the effectiveness of various remedial technologies at a site. The extent to which adaptation is cost-effective is, of course, a site-specific decision. Resources on adaptive management at sediment sites include the NRC s report *Environmental Cleanup at Navy Facilities* (NRC 2003) and Connolly and Logan (2004).

Even before the sediment at a site is well characterized, if risk is obvious, it may be very important to begin to control significant ongoing land-based sources. It also may be appropriate to take other early or interim actions, followed by a period of monitoring, before deciding on a final remedy. Highlight 2-7 provides examples of early actions taken to control sources, minimize human exposure, control sediment migration, or reduce risk from sediment hot spots at contaminated sediment sites. Early or interim actions are frequently used to prevent human exposure to contaminants or to control sources of sediment contamination. However, such actions for sediment are less frequent. Factors for determining which response components may be suitable for early or interim actions include the time frame needed to attain specific objectives, the relative urgency posed by potential or actual exposure, the degree to which an action may reduce site risks, and compatibility with likely long-term actions (U.S. EPA 1992b).

An early action taken under Superfund removal authority may be appropriate at a sediment site when, for example, it is necessary to respond quickly to a release or a threatened release of a hazardous substance that would present an immediate threat. At contaminated sediment sites, removal authority or state authorities have been used to implement many of the actions listed in Highlight 2-7. The NCP at 40 CFR □300.415 outlines criteria for using removal authority, as further explained in the EPA guidance and directives (U.S. EPA 1993a, U.S. EPA 1996d, U.S. EPA 2000d). Project managers may also consider separating the management of source areas from other, less concentrated areas by establishing separate operable units (OUs) for the site.

2.8 SEDIMENT AND CONTAMINANT FATE AND TRANSPORT

An important part of the remedial investigation at many sediment sites is an assessment of the extent of sediment and contaminant transport and the effect of that transport on exposure and risk. This usually includes gaining an understanding of the processes and events in the past and predicting future transport and exposure.

Highlight 2-7: Potential Examples of Early Actions at Contaminated Sediment Sites

Actions to prevent releases of contaminants from sources:

- Excavation or containment of floodplain soils or other source materials in the floodplain
- Engineering controls (e.g., sheet pilings, slurry walls, grout curtains, and extraction) to prevent highly contaminated ground water, NAPL, or leachate from reaching surface water and sediment
- Engineering controls to prevent contaminated runoff from reaching surface water and sediment

Actions to minimize human exposure to contaminants (coordinated with other appropriate agencies):

- Access restrictions
- Fish consumption advisories
- Use restrictions and advisories for water bodies
- Actions to protect downstream drinking water supplies

Actions to minimize further migration of contaminated sediment:

- Boating controls (e.g., vessel draft or wake restrictions to prevent propeller wash, anchoring restrictions)
- Excavating, dredging, capping, or otherwise isolating contaminated sediment hot spots

Actions taken to reduce risk from highly contaminated sediment hot spots:

- Capping, excavation, or dredging of localized areas of contaminated sediment that pose a very high risk

In most aquatic environments, surface sediment and any associated contaminants move over time. The more important and more complex issue is whether movement of contaminated sediment (surface and subsurface), or of contaminants alone, is occurring or may occur at scales and rates that will significantly change their current contribution to human health and ecological risk. Addressing that issue requires an understanding of the role of natural processes that counteract sediment and contaminant movement and fate, such as natural sedimentation and armoring, and contaminant transformations to less toxic or less bioavailable compounds. For this reason, it is important for project managers to use technical experts to help in the analysis, especially where large amounts of resources are at stake.

Sediment movement also is a complex topic because it has both positive and negative effects on risk. For example, floods frequently transport both clean and contaminated sediment, which are subsequently deposited within the water body and on floodplains. This may spread contamination,

isolate (through burial) other existing contamination, and lower concentrations of contaminants (through dilution) within the immediate site boundaries.

Both natural and man-made (i.e., anthropogenic) forces may cause sediment and contaminants to move. Highlight 2-8 lists examples of each.

Highlight 2-8: Potential Causes of Sediment and or Contaminant Movement

Natural causes of sediment movement include:

- Routine currents in rivers, streams, and harbors
- Tides in marine waters and estuaries
- Floods generated by rainfall or snow-melt induced runoff from land surfaces
- Ice thaw and ice dam-induced scour
- Seiches (oscillation of lake elevation caused by sustained winds), especially in the Great Lakes
- Storm-generated waves and currents (e.g., hurricanes, Pacific cyclones, nor□easters)
- Seismic-generated waves (e.g., tsunamis)
- Earthquakes, landslides, and dam failures
- Bioturbation from micro- and macrofauna

Anthropogenic causes of sediment movement include:

- Navigational dredging and channel maintenance
- Placer mining as well as sand and gravel mining
- Intentional removal or breaching of hydraulic structures such as dams, dikes, weirs, groins, and breakwaters
- In-water construction
- Boat propeller wash, ships□wakes, ship grounding or anchor dragging

Causes of dissolved contaminant movement without sediment movement include:

- Flow of ground water through sediment
- Molecular diffusion
- Gas-assisted transport

Many contaminated sediment sites are located in areas that are primarily depositional, or in areas where only a limited surface layer of sediment is routinely mobilized. In these fairly stable areas, other processes may contribute to sediment and contaminant movement and resulting exposure and risk. These include, for sediment, bioturbation, and for dissolved contaminants, ground water flow, molecular diffusion, and, potentially, gas-assisted transport. Like erosion and deposition, these processes continue

to operate after remedies are in place, so an understanding of whether or not they are likely to be significant ongoing contaminant transport pathways at a particular site is especially important for evaluating in-situ capping and MNR alternatives.

Various empirical and modeling methods exist for evaluating sediment and contaminant movement and their consequences. The models normally rely upon site-specific empirical data for input parameters. Both empirical methods and models have limitations, so it is usually important to consider a variety of methods in evaluating a site and to compare the results. For large or complex sediment sites, project managers should approach an assessment of sediment and contaminant movement from the following aspects:

- A site-specific assessment of empirical site characterization data (see Section 2.8.1);
- A site-specific assessment of the frequencies and intensities of expected routine and extreme events that mobilize sediment (see Section 2.8.2);
- A site-specific assessment of ongoing processes that mobilize contaminants in otherwise stable sediment, such as bioturbation, diffusion, and advection (see Section 2.8.3); and
- A site-specific assessment of the expected consequences or results of sediment and contaminant movement in terms of exposure and risk, cost, or other consequences (see Section 2.8.4).

As noted above, this assessment will frequently require the use of models. A wide variety of models is available, ranging from simple models with small numbers of input criteria to complex, multi-dimensional models that are data intensive. A discussion of model uses and selection is presented in Section 2.9.

Especially for larger sites, a lines of evidence approach should be used to evaluate the extent of sediment and contaminant movement and resultant exposure for various areas of the water body. Where multiple lines of evidence point to similar conclusions, project managers may have more confidence in their predictions. Where the lines of evidence do not concur, project managers should bring their technical experts together to determine the source of the discrepancies and understand their significance. This approach is described in more detail in Chapter 4, Section 4.4, Evaluation of Natural Recovery.

2.8.1 Data Collection

An assessment of sediment and contaminant movement begins with the collection of a variety of empirical data (i.e., data derived from field or laboratory observation). Although literature values may be available for some parameters, project managers are encouraged to collect site-specific information for the most important processes at the site (as identified in the conceptual site model), especially where large resources are at stake in decision making.

The vertical and horizontal sediment and contaminant distributions present at a site are a result of all of the routine and extreme, natural and anthropogenic processes that contribute to the physical, chemical, and biological attributes of a water body. Site conditions at the time of investigation generally reflect a combination of influences. Project managers should not assume that current conditions represent

stable conditions when, in fact, sediment may be actively responding to recent or current forces and events. Conversely, project managers should not assume a site or all areas of a site are unstable or contaminants are mobile at a scale or rate which significantly impacts risk. At many sites, the same areas of contamination persist over many years, despite some level of surface sediment and contaminant redistribution.

Processes that are important in terms of exposure and risk on a watershed scale may be less important in smaller, more isolated areas of a water body. Both scales of investigation may be needed. For example, in some situations, the large scale rainstorms associated with hurricanes may greatly impact sediment loading to the water body through erosion of watershed soils, but have little effect on stability of the in-water sediment bed itself. When considering the potential impacts of disruptive forces on sediment movement, it is important to assess these forces as they relate to the overall watershed and in terms of current and future site characteristics.

Many site characteristics affect sediment movement, but primary among them are the flow-induced shear stress at the bottom of the water body during various conditions, and the cohesiveness of the upper sediment layers. In most environments, bottom shear stress is controlled by currents, waves, and bottom roughness (e.g., sand ripples, biologically formed mounds in fines). A preliminary evaluation of the significance of sediment movement should include at least site-specific measurements of surface water flow velocities and discharges, water body bathymetry, and surface sediment types (e.g., by use of surface grab samples).

In some cases, empirically measured erosion rates are lower than anticipated from simple models, due to natural armoring. Winnowing (suspension and transport) of fines from the surface layers of sediment is one common form of armoring. Others are listed in Highlight 2-9, including the effect known as dynamic armoring, which describes the effect caused by suspended sediment or a fluff, floc, or low density mud layer (present in some estuaries and lakes) that decreases the expected erosion rate of underlying sediment.

Highlight 2-9: Principal Types of Armoring

Physical:
- Winnowing of fine grained materials, leaving larger-grained materials on surface
- Compaction of fine-grained sediment

Chemical:
- Chemical reactions and weathering of surface sediment

Dynamic:
- Suspended sediment dampening turbulence during high flow events

Biological:
- Physical protection and sequestration by rooted aquatic vegetation
- Mucous excretions of polychaetes
- Erosion-resistant fecal pellets or digested sediment

Sediment properties that affect cohesion and erosion in many sediment environments include bulk density, particle size (average and distribution), clay mineralogy, the presence of methane gas, and the organic content. It is not unusual for erosion rates to vary by 2 to 3 orders of magnitude spatially at a site, depending on currents, bathymetry, bioturbation, and other factors (e.g., pore water salinity). In a fairly uniform cohesive sediment core, erosion rates may drop several orders of magnitude with depth into the sediment bed, but in more variable cores this may not be the case.

Biological processes by macro- and microorganisms also affect sediment in multiple ways, both to increase erosion (e.g., gas generation and bioturbation by lowering bulk density) and to decrease erosion (e.g., aquatic vegetation, biochemical reactions which increase shear strength of sediment). The process of sediment mixing caused by bioturbation is discussed further in Section 2.8.3.

A wide variety of empirical methods is available to assess the extent of past sediment and contaminant movement. Highlight 2-10 lists some key examples. Each of these methods has advantages and limitations, and generally none should be used in isolation. The help of technical experts is likely to be needed to determine which methods are most likely to be useful at a particular site.

2.8.2 Routine and Extreme Events

Naturally occurring hydrodynamic forces such as those generated by wind, waves, currents, and tides, occur with great predictability and significantly influence sediment characteristics and movement (Hall 1994). While these routine forces seldom cause changes that are dramatically visible, they may be the events causing highest shear stress and, therefore, the most important factors in controlling the physical structure of a given water body. In northern climates, formation of ice dams and ice scour are also routine events that may have significant effects on sediment. It is important to note that seasonal changes in water flow may also affect where erosion and deposition occur. Depending on the location of the site, (e.g., riverine areas, coastal/marine area, inland water bodies), different water body factors will play important roles in determining sediment movement. To determine the frequency of particular routine forces acting upon sediment, project managers should obtain historical records on flows and stages from nearby gauging stations and on other hydrodynamic forces. However, project managers should keep in mind that residential or commercial development in a watershed may significantly increase the impervious area and subsequently increase the frequency and intensity of routine flood events. While the intensity of most routine forces may be low, their high frequency may cause them to be an important influence on sediment movement within some water bodies.

Highlight 2-10: Key Empirical Methods to Evaluate Sediment and Contaminant Movement

Bathymetry (evaluates net change in sediment surface elevations)

- Single point/local area devices
- Transects/cross-sections (with known vertical and horizontal accuracy)
- Longitudinal river profiles along the thalweg (i.e., location of deepest depth)
- Acoustic surveys (with known vertical and horizontal accuracy)
- Comparison to dredging records, aerial photos, overall geomorphology

Contaminant data (from continuous cores, surface sediment, and water column):

- Time-series observations (event scale and long-term seasonal, annual, decade-scale)
- Comparison of core pattern or changing pattern in surface sediment, with pollutant loading history
- Comparison of concentration patterns during and after high energy events

Sediment data (e.g., from continuous cores or surface samples):

- Patterns of grain-size distribution (McLaren and Bowles 1985, McLaren et al. 1993, Pascoe et al. 2002)
- In-situ or ex-situ erosion measurement devices [e.g., SEDFLUME (Jepsen et al. 1997, McNeil et al. 1996), PES (Tsai and Lick 1986), Sea Carousel (Maa et al. 1993), or Inverted Flume (Ravens and Gschwend 1999)]
- Sediment water interface camera

Geochronology (evaluates continuity of sedimentation and age of sediment with depth in cores):

- ^{137}Cs, lignin, stable Pb (longer-lived species to evaluate burial rate and age progression with depth)
- ^{210}Pb, ^{7}Be, ^{234}Th (shorter-lived species to evaluate depth of mixing zone)
- X-radiography, color density analysis

Geomorphological studies:

- Land and water body geometry and bathymetry; physical processes
- Human modifications

Sediment-contaminant mass balance studies, especially during high energy events:

- Upstream and tributary loadings (grain size distributions and rating curves)
- Tidal cycle sampling (in marine estuaries and coastal seas)
- Sampling during the rising limb of a rain-event generated runoff hydrograph (frequently greatest erosion)

Dissolved contaminant movement:

- Seepage meters at sediment surface
- Gradients near water body

In contrast, some water bodies are significantly affected by short-term extreme forces that are much less common. In many cases, these extreme forces originate by the same mechanisms as routine forces (e.g., wind) but are significantly stronger than routine conditions and capable of moving large amounts of sediment. Some extreme events, however, have no routine event counterparts (e.g., earthquakes). Meteorological events, such as hurricanes, may move large amounts of sediment in coastal areas due to storm surges and unusually high tides that cause flooding. Flooding may occur from snow-melt and other unusually heavy precipitation events resulting in the movement of large amounts of upland soil and erosion of sediment, which are then deposited in other areas of the water body or on floodplains when the flow slows during the falling limb of the runoff hydrograph. Scour of the sediment bed may also result from the movement of ice and/or natural or man-made debris during extreme flood events. To obtain a preliminary understanding of extreme event frequency at a site, it is important to examine both historical records (e.g., meteorological and flow records) and site characterization data (e.g., core data and bathymetry).

Floods are frequently classified by their probability of occurrence; for example 50-year, 100-year, 200-year, and probable maximum flood. Although the term 100-year flood suggests a time frame, it is in fact a probability expression that a flood has a one percent probability of occurring (or being exceeded) in any year. Similarly, 200-year flood refers to a flood with a 0.5 percent probability of occurring in any year. Probable maximum flood refers to the most extreme flood that could theoretically occur based on maximum rainfall and maximum runoff in a watershed. It is not uncommon for multiple low probability events to happen more frequently than expected, especially when the hydrograph record used to determine these probabilities is not very long or where land use or climate is changing.

It is important to consider the intensity of extreme hydrodynamic forces as well as their frequency. Intensity is a measure of the strength, power or energy of a force. The intensity of a force will be a significant determinant of its possible impact on the proposed remedy. Tropical storms (including hurricanes) are often classified according to their intensity, that is, the effects at a particular place and time, which is a function of both the magnitude of and distance from the event. Tropical storms such as hurricanes are commonly classified by intensity using the Saffir-Simpson Scale of Category 1 to Category 5. Other physical forces and events, such as earthquakes, may be classified according to magnitude, that is, a measure of the strength of the force or the energy released by the event. Earthquakes are most commonly classified in this way (e.g., the Richter scale) although they may also be classified by intensity at a certain surface location (e.g., the Modified Mercalli scale).

For sites in areas that may be affected by extreme events, project managers should assess the record of occurrence near the site and determine the appropriate category or categories for analysis. The recurrence interval that is considered in a project generally relates to the magnitude of the resultant impacts. The choice of design event gives consideration to the impact of the event and the cost of designing against the event. For evaluation of contaminated sediment sites, project managers should evaluate the impacts on sediment and contaminant movement of a 100-year flood and other events or forces with a similar probability of occurrence (i.e., 0.01 in a year). A similar probability of occurrence may be appropriate for analysis of other extreme events such as hurricanes and earthquakes. At some sites, it may be appropriate to analyze the effects of events with lower and higher probabilities to understand the cost-effectiveness of various design decisions. Recorded characteristics of physical events, such as current velocities or wave heights, may provide project managers with parameters needed to calculate or model sediment movement. If information from historical records is insufficient or the historical record is too short to be useful, project managers should consider obtaining technical assistance

to model a range of potential events to estimate effects on sediment movement and transport. Section 2.9 of this chapter discusses modeling in more detail.

2.8.3 Bioturbation

In some depositional environments, the most important natural process bringing contaminants to the sediment surface is bioturbation. Broadly speaking, bioturbation is the movement of sediment by the activities of aquatic organisms. Although this movement may be in many directions, it is the vertical mixing that is mainly of concern for project managers because it brings contaminants to the bed surface, where most exposures occur. While many discussions of bioturbation are focused on sediment dwelling animals, such as worms and clams, bioturbation may also include the activity of larger organisms such as fish and aquatic mammals. The effects of bioturbation can include the mixing of sediment layers, alteration of chemical forms of contaminants, bioaccumulation, and transport of contaminants from the sediment to interstitial/pore water or the water column. Many bottom-dwelling organisms physically move sediment particles during activities such as locomotion, feeding, and shelter building. These activities may alter sediment structure, biology, and chemistry, but the extent and magnitude of the alteration depends on site location, sediment type, and the types of organisms and contaminants present.

One factor of concern for understanding exposure is the depth to which significant physical mixing of sediment takes place, sometimes known as the mixing zone. The depth of the mixing zone can be determined by examination of sediment cores (especially radioisotope analysis of core sections), or other site characterization data that displays the cumulative results of bioturbation through time, but useful information may also be gained from a sediment profile camera and other results. It is also useful to be aware of the typical burrowing depths of aquatic organisms in uncontaminated environments similar to the site. Project managers should keep in mind, however, that population density has a tremendous effect on whether organisms present at the site may have a significant effect on the mixing zone. It is important to understand the depth of the mixing zone in the various environments at a site because, where sediment is not subject to significant erosion and contaminants are not significantly mobilized by ground water advection, contaminants below this zone are unlikely to contribute to current or future risk at a site.

Typically, the population of benthic organisms is greatest in the top few centimeters of sediment. In fresh waters, the decline in population density with depth is such that the mixed layer is commonly five to 10 cm deep (NRC 2001), although it may be deeper, especially in marine waters with high populations of deep burrowing organisms. Highlight 2-11 provides examples of organisms that cause bioturbation, their activity type, and the general depth of the activity. However, project managers should also consider the activity type, the intensity of the activity, and organism population density, when determining the extent bioturbation should be considered in site evaluation. For example, the depth and effectiveness of bioturbation may be very different in a highly productive estuary and in a heavily used commercial boat slip.

A project manager should be aware of at least the following parameters when assessing the depth of the mixing zone and the potential role bioturbation will play on a given sediment bed:

- *Site location* - Salinity, water temperatures, depths, seasonal variation);
- *Sediment type* - Size distribution, organic and carbonate content, bulk density); and

- *Organism type* - Organisms either present and/or likely to recruit to and recolonize the area).

This analysis may be done for naturally deposited sediment as well as potential in-situ capping material or dredging backfill material. Where bioturbation is likely to be a significant process, it is important to evaluate the depth over which it causes significant mixing, using site-specific data and assistance by technical experts, to assess alternative approaches for the site.

Highlight 2-11: Sample Depths of Bioturbation Activity

Organism	Activity Type	Depth	Reference
Freshwater			
Tubificid worm (oligochaete)	Burrowing/Feeding	0 - 3 cm	Matisoff, Wang, and McCall 1999 Pennak 1978
Midge and Mayfly (insects)	Burrowing/Feeding	0 - 15 cm	Matisoff and Wang 2000 Pennak 1978
Burbot (fish)	Burrowing	0 cm - 30 cm	Boyer et al. 1990
Marine/Estuarine (Atlantic Coast)			
Bristleworm (polychaete)	Burrowing	0 cm -15 cm	Hylleberg 1975
Bamboo worm (polychaete)	Burrowing/Feeding	0 cm - 20 cm	Rhoads 1967
Fiddler crab (crustacean)	Burrowing	0 cm - 30.5 cm	Warner 1977
Clam (bivalve)	Burrowing	0 cm - 3 cm	Risk and Moffat 1977
Marine/Estuarine (Pacific Coast)			
Bristleworm (polychaete)	Burrowing	0 cm - 15 cm	Hylleberg 1975
Fiddler crab (crustacean)	Burrowing	0 cm - 30.5 cm	Warner 1977
Clam (bivalve)	Burrowing	0 cm - 3 cm	Risk and Moffat 1977

2.8.4 Predicting the Consequences of Sediment and Contaminant Movement

Depending on its extent, movement of sediment or contaminants may or may not have significant consequences for risk, cost, or other important factors at a specific site. A number of differing factors may be important in determining whether expected or predicted movements are acceptable. Historical records or monitoring data for contaminant concentrations in sediment and water during events such as floods may be valuable in analyzing the increase in exposure and risk. Where this information is not available or has significant uncertainty, models may also be very useful to help understand and predict changes. This analysis should include increased risk from not only contaminant releases to the immediate water body, but wherever those contaminants are likely to be deposited. Increased cost may include remedy costs such as cap repair or costs related to contaminant dispersal, such as increased disposal cost

of downstream navigational dredging. There may also be societal or cultural impacts of contaminant releases the project manager should consider, such as lost use of resources.

Project managers should assess the impacts of contaminant release on potential receptors on a site-specific basis, using information generated during the baseline human health and ecological risk assessments. Where natural recovery is being evaluated, project managers should recognize that not only the rate of net sedimentation, but also the frequency of erosive episodes, can help determine the rate of recovery for surface sediment and biota. Where in-situ capping is being evaluated, project managers should recognize that some amount of erosion and sediment transport may be acceptable and can be incorporated into plans for remedial design and cap maintenance. Increased risk to human or ecological receptors due to contaminant releases during dredging may be a related analysis when considering dredging. Comparing the increased risks, costs, or other consequences of sediment disruption due to natural causes or the remedy itself also may be an important part of the remedy selection process.

When evaluating remedy alternatives, the significance of potential harm due to reexposure of contaminated sediment or contaminated sediment redistribution is an important consideration. Factors to be considered include the nature of the contaminants, the nature of the potential receiving environment and biological receptors, and the potential for repair or recovery from the disturbance. These factors can be used to evaluate risks, costs, and/or other effects of different events on existing contaminated sediment or sediment remedies.

2.9 MODELING

Models are tools that are used at many sediment sites when characterizing site conditions, assessing risks, and/or evaluating remedial alternatives. A complex computer model (e.g., multi-dimensional numerical model) may not be needed if there is widespread agreement about the best remedial strategy based on an adequate understanding of site conditions, however, this is not often the case. At some sites, significant uncertainties exist about site characterization data and the processes that contribute to relative effectiveness of available remedial alternatives. Models can help fill gaps in knowledge and allow investigation of relationships and processes at a site that are not fully understood. For this reason, simple or complex modeling can play a role at most sediment sites.

There is a wide range of simpler empirical models and more robust computer models that can be applied to contaminated sediment sites. Simple models that aggregate processes or consider only some portion of a problem can provide significant insights and should be applied routinely at sediment sites, even complex sites. For example, simple steady-state mass balance models applied during a time period where there are no disruptive events can be used to determine whether external contaminant sources have been identified and properly quantified. Hydrodynamic model predictions of currents and associated bottom shear stresses can provide information about the potential for erosion and the degree of interaction between backwater and main channel areas. Even if a complex fate and transport model is never developed, simple modeling can be used to develop a better understanding of current and future site conditions and lead to selection of the most appropriate remedial alternative.

More complex fate and transport models are frequently applied to the most complex sites. These sites typically have a long history of data collection, have documented contaminant concentrations in sediment and biota, and often have fish consumption advisories already in place. Fate and transport models can be useful tools, even though they can be time consuming and expensive to apply at complex

sediment sites. Most of these modeling efforts require large quantities of site-specific data, and typically a team of experienced modelers is needed. Nevertheless, these models are helpful in that they give, when properly applied, a more complete understanding of the transport and fate of contaminants than typically can be provided by empirical data (from field or laboratory) alone.

Whether and when to use a model, and what models to use, are site-specific decisions and modeling experts should be consulted. Modeling of contaminated sediment, just as with other modeling, should follow a systematic planning and implementation process. Technical assistance is available to project managers from EPA s Superfund Sediment Resource Center (SSRC), where experts from inside and outside the Agency may be accessed. Additional research about contaminated sediment transport and food web modeling is underway at the Office of Research and Development (ORD) (e.g., U.S. EPA in preparation 1 and 2). Project managers should monitor the Superfund sediment Web site at http://www.epa.gov/superfund/resources/sediment or contact their region s ORD Hazardous Substance Technical Liaison for more information.

In most cases, simple or complex models are expected to complement environmental measurements and address gaps that exist in empirical information. Examples of the uses of models include the following:

- Identifying data gaps during the initial phases of a site investigation;
- Illustrating how contaminant concentrations vary spatially at a site. Empirical information can provide useful benchmarks that can be interpolated or modeled to get a better understanding of the distribution of contaminants;
- Predicting contaminant fate and transport over long periods of time (e.g., decades) or during episodic, high-energy events (e.g., tropical storm or low-frequency flood event);
- Predicting future contaminant concentrations in sediment, water and biota to evaluate relative differences among the proposed remedial alternatives, ranging from monitored natural recovery to extensive removal; and
- Comparing modeled results to observed measurements to show convergence of information. Both modeling results and empirical data usually will have a measure of uncertainty, and modeling can help to examine the uncertainties (e.g., through sensitivity analysis) and refine estimates, which may include indications for where to sample next.

The use of models at sediment sites is not limited to the remedy selection phase. Most sites that use models for evaluation of proposed remedies have previously developed a mass balance or other type of model during the development of the baseline risk assessment. These models are often used to quantify the relationships among contaminant sources, exposure pathways, and receptors. At these sites, the same model is often used to predict the response of the system to various cleanup options. Where this is done, it is important to continue to test the model predictions by monitoring during the remedy implementation and post-remedy phases to assess whether cleanup is progressing as predicted by the model. Where it is not, information should be relayed to the modeling team so the model can be modified or recalibrated and then used to develop more accurate future predictions.

2.9.1 Sediment/Contaminant Transport and Fate Model Characteristics

A sediment/contaminant transport and fate model typically is a mathematical or conceptual representation of the movement of sediment and associated contaminants, and the chemical fate of those contaminants, as governed by physical, chemical and biological factors, in water bodies. Currently, there are two basic types of sediment transport models: conceptual and mathematical models. In addition, there are several different types of mathematical models. General types of models are described in Highlight 2-12, and an example of a conceptual site model is presented in Highlight 2-13.

Highlight 2-12: Key Characteristics of the Major Types of Sediment Contaminant Transport and Fate Models

Conceptual Model:

Identifies the following: 1) contaminants of potential concern; 2) sources of the contaminants; 3) physical and biogeochemical processes and interactions that control the transport and fate of sediment and associated contaminants; 4) exposure pathways; and 5) ecological and human receptors.

Mathematical Model:

A set of equations that quantitatively represent the processes and interactions identified by the conceptual model that govern the transport and fate of sediment and associated contaminants. Mathematical models include analytical, regression, and numerical models.

Analytical Model:

An analytical model is one or more equations (e.g., simplified - a linearized, one-dimensional form of the advection-diffusion equation) for which a closed-form solution exists. This type of model may not be applicable at most sites due to the complexities associated with the forcing hydrodynamics and spatial and temporal heterogeneities in sediment and contaminant properties/characteristics.

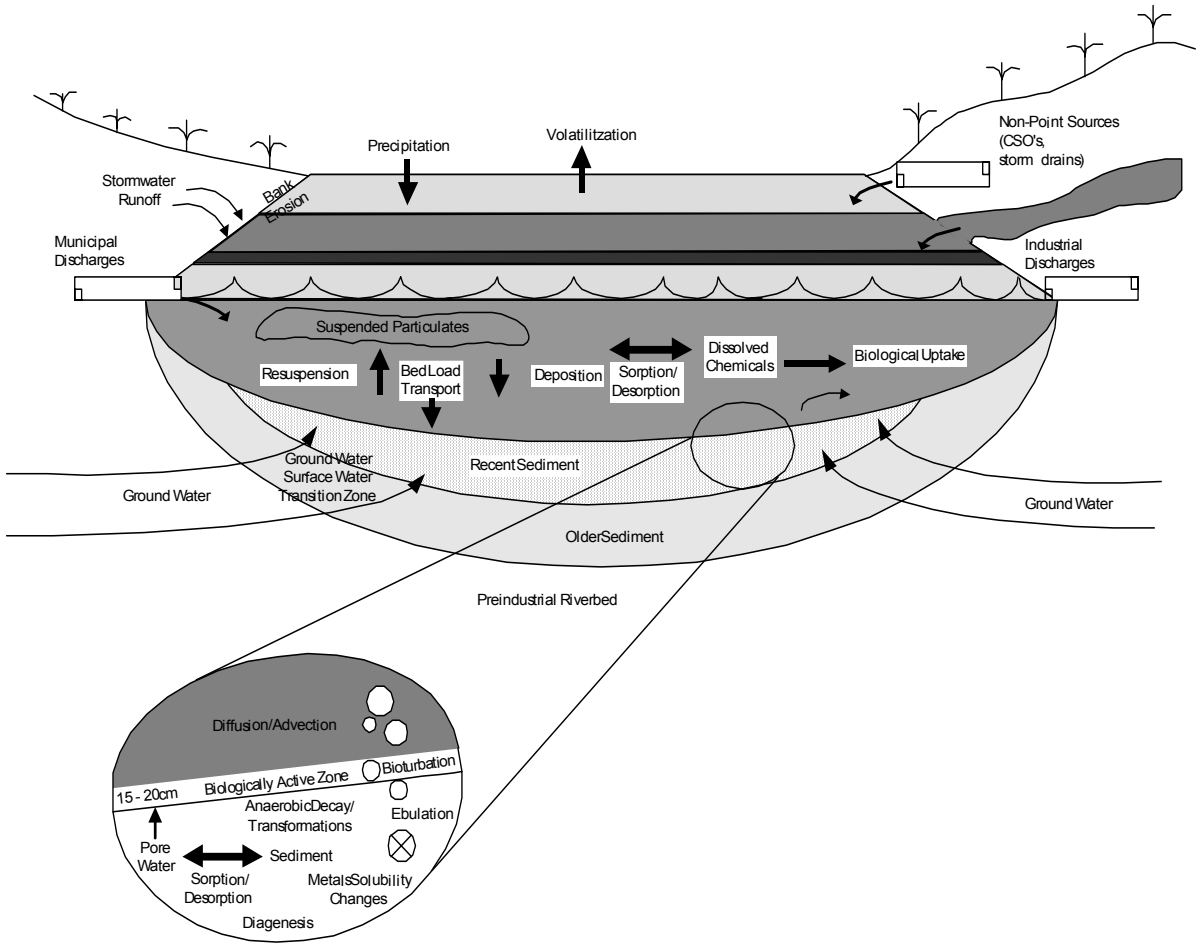
Regression Model:

A regression model is a statistically determined equation that relates a dependent variable to one or more independent variables. A stage-discharge rating curve is an example of a regression model in which stage (e.g., water level) and discharge (e.g., amount of water flow) are the independent and dependent variables, respectively.

Numerical Model:

In a numerical model, an approximate solution of the set of governing differential equations is obtained using a numerical technique. Examples of numerical techniques include finite difference and finite element methods. A numerical model is used when the processes being modeled are represented by nonlinear equations for which closed-form solutions do not exist.

Highlight 2-13: Sample Conceptual Site Model Focusing on Sediment-Water Interaction



Source: Modified from Sediment Management Workgroup (SMWG)

Typically, transport and fate models are inherently limited by our current understanding of the factors governing these processes and our ability to quantify them (i.e., represent mathematically their interactions and effects on the transport and fate of sediment and contaminants). Even the most complex sediment model may be a relatively simplistic representation of the movement of sediment through natural and engineered water bodies. It may be simplistic due to the following:

- Limitations in our understanding of natural systems, as reflected in the current state-of-the-science;
- Empiricism inherent in predicting flow-induced sediment transport, bank erosion, and nonpoint source loads;

- The relatively large space and time blocks used for modeling the water body; and
- The inability to realistically simulate geomorphological processes such as river meandering, bank erosion, and localized effects (e.g., due to natural debris or beaver dams).

Nevertheless, sediment/contaminant transport and fate models generally are useful tools when properly applied, although they are data intensive and require specialized expertise to apply and interpret the results.

2.9.2 Determining Whether A Mathematical Model is Appropriate

Since mathematical transport and fate models can be time-intensive and expensive to apply, their use and interpretation generally require specialized expertise. Because of this, mathematical modeling is not recommended for every sediment site. In some cases, existing empirical data and new monitoring data may be sufficient to support a decision. A mathematical modeling study is usually not warranted for very small (i.e., localized) sites, where cleanup may be relatively easy and inexpensive. Mathematical modeling generally is recommended for large or complex sites, especially where it is necessary to predict contaminant transport and fate over extended periods of time to evaluate relative differences among possible remedial approaches.

Project managers should use the following series of questions to help guide the process for determining the appropriate use of site-specific mathematical models:

- Have the questions or hypotheses the model is intended to answer been determined?
- Are historical data and/or simple quantitative techniques available to answer these questions with the desired accuracy?
- Have the spatial extent, heterogeneity, and levels of contamination at the site been defined?
- Have all significant ongoing sources of contamination been defined?
- Do sufficient data exist to support the use of a mathematical model, and if not, are time and resources available to collect the required data to achieve the desired level of confidence in model results? and
- Are time and resources available to perform the modeling study itself?

If the decision is made that some level of mathematical modeling is appropriate, the following section should assist project managers in deciding what type of model should be used.

2.9.3 Determining the Appropriate Level of Model

When the decision is made that a mathematical model is appropriate at a site, project managers should generally consider three steps in determining what level of modeling to use. It is important to consider all three steps in order. In some cases, these three steps may be more useful when performed in

an iterative fashion (for example, based on additional data analysis or from results obtained during Step 3, it may become apparent that the conceptual site model (CSM) should be modified).

Step 1: Develop Conceptual Site Model

Development of a CSM is recommended as the key first step in this process in determining the level of modeling. As described in Section 2.2, a CSM identifies the processes and interactions that typically control the transport and fate of contaminants, including sediment associated contaminants. If this step is not performed, then the decision of what level of modeling is appropriate may be made with less than the requisite information that might be needed to make a scientifically defensible decision.

The development of a CSM usually requires examination of existing site data to assist in determining the significant physical and biogeochemical processes and interactions. Relatively simple quantitative expressions of key transport and fate processes using existing site data, such as presented by Reible and Thibodeaux (1999) or Cowen et al. (1999), may help in identifying those processes most significant at the site.

Step 2: Determine Processes that Can and Cannot be Currently Modeled

This step concerns determining if the most significant processes and interactions that control the transport and/or fate of sediment contaminants, as identified in the CSM, can be simulated with one or more existing sediment transport and fate models. Mathematical models (in particular numerical models) that have been developed can simulate most of the processes controlling the transport and fate of sediment and contaminants in water bodies (including a wide variety of physical, chemical, and biological processes). Highlight 2-14 depicts the inter-relationship of some major processes and the type of model with which they are associated. If it is determined that there are existing models capable of simulating at a minimum the most significant (i.e., first-order) processes and interactions, then the project manager should (using the appropriate technical experts) identify the types of models (e.g., analytical, regression, numerical) having this capability and eliminate from further consideration those types of models not having this capability.

Depending on the needs at the site, models or model components (modules) may link many of these processes presented in Highlight 2-14 into one model. Examples of the processes that can be modeled include the following:

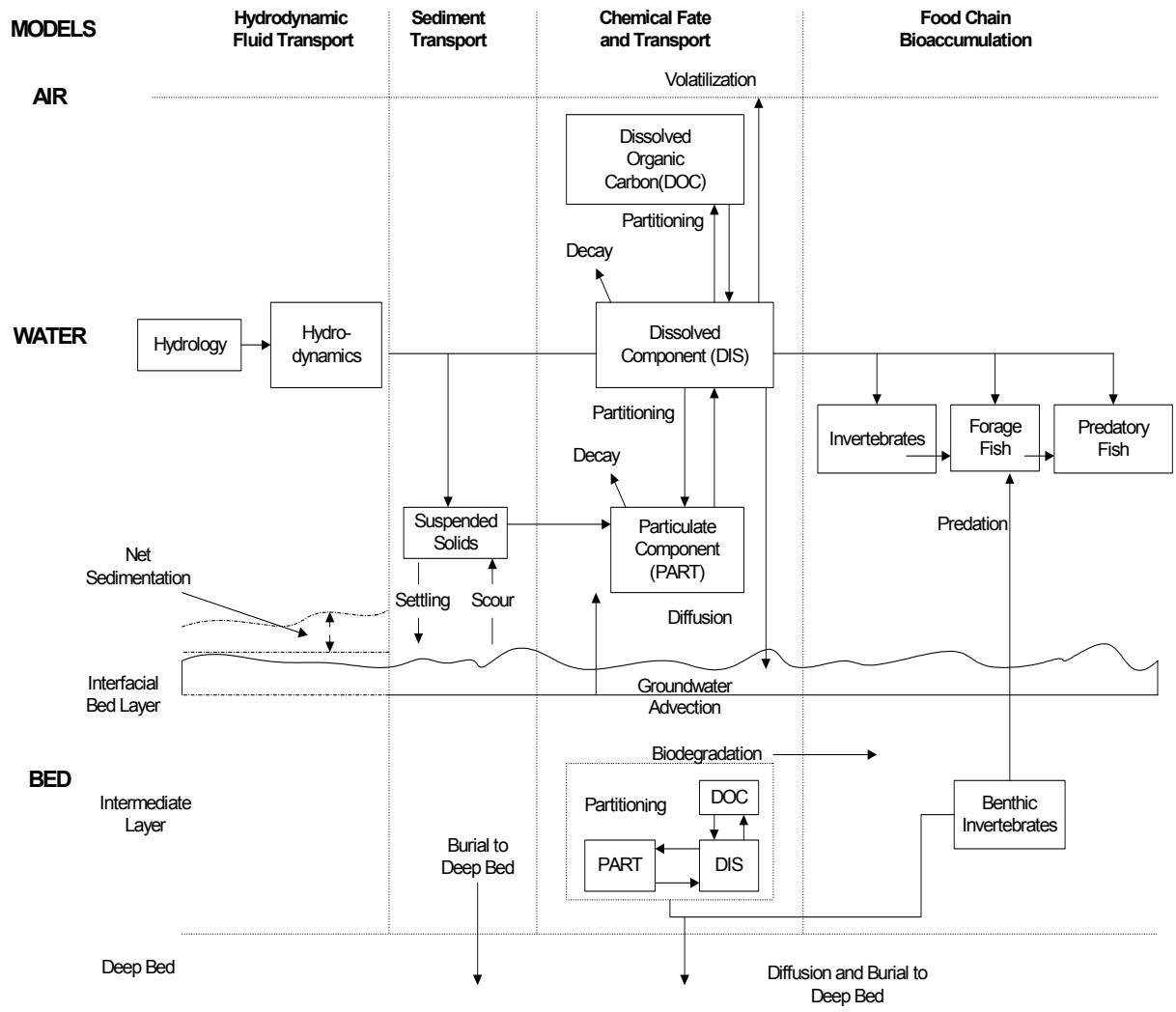
- *Land and air:* Physical processes that result in loading of contaminants to water bodies may include point discharges, overland flow (i.e., runoff), discharge of ground water, NAPL seeps, and air deposition;

- *Water column:* Physical processes that may result in movement of dissolved or sediment-sorbed contaminants include transport via the water s ambient flow (advection), diffusion, and settling of sediment particles containing sorbed contaminants;

- *Sediment bed:* Important physical processes include the movement of pore water and dissolved contaminants, seepage into and out of the sediment bed and banks, and the mixing of dissolved and sediment-sorbed contaminants by bioturbation. In addition, both sorbed and dissolved material may be exchanged between the water column and sediment bed due to sediment deposition and resuspension or erosion; and

- *Water column and sediment bed:* Physiochemical processes influencing the fate and transport of contaminants include two-phase and three-phase chemical partitioning as described below. Biogeochemical reaction processes influencing the fate of contaminants include speciation, volatilization, anaerobic gas formation, hydrolysis, oxidation, photolysis, biotransformation, and biological uptake.

Highlight 2-14: Sample Contaminant Exposure Modeling Framework



Source: NRC 2001

In Highlight 2-14 and in other modeling discussions, generally, two-phase partitioning refers to modeling the contaminant in two parts or phases: a bioavailable dissolved fraction and a generally non-

bioavailable particulate fraction. In three-phase partitioning, contaminant concentrations are normally considered in three phases: the bioavailable dissolved phase, a generally non-bioavailable dissolved organic carbon (DOC) phase, and a generally non-bioavailable particulate organic carbon phase.

If it is determined that there are no existing models capable of simulating, at a minimum, the most significant (i.e., first-order) processes and interactions, then project managers may need to rely on other tools or methods for evaluating proposed approaches, or develop and test new models or modules.

Examples of processes that cannot be dynamically simulated, even using state-of-the-art sediment transport models, may include geomorphological processes such as the development of meanders in streams and rivers, bank cutting/erosion, nepheloid layer sediment transport, and mud wave phenomena. However, there are empirical methods for simulating some of these processes, including estimating the total quantity of sediment introduced to a water body due to the failure of a river/stream bank. Likewise, there are empirical tools to estimate the importance of nepheloid layer transport (i.e., relatively high sediment flux occurring immediately above the sediment-water interface). Empirical tools are also being developed to simulate mud wave transport processes resulting from sediment disturbances such as dredging and resultant dispersal of contaminated sediment residuals.

Step 3: Select an Appropriate Model

If one or more models or types of mathematical models capable of simulating the controlling transport and fate processes and interactions exist, then project managers should use the process described above to choose the appropriate type of model (i.e., level of analysis). If the decision is made to apply a numerical model at a sediment site, selection of the most appropriate contaminated sediment transport and fate model to use at a specific site is one of the critical steps in a modeling program. During this process, familiarity with existing sediment transport models is essential. Comprehensive technical reviews of available models have been conducted by the EPA s ORD National Exposure Research Laboratory (see U.S. EPA in preparation 1 and 2).

2.9.4 Model Verification, Calibration, and Validation

Where numerical models are used, verification, calibration, and validation typically should be performed to yield a scientifically defensible modeling study. The project manager should be aware that the terms verification and validation are frequently used interchangeably in modeling literature. These terms, for purposes of this guidance, mean:

Model verification: Evaluating the model theory, consistency of the computer code with model theory, and evaluation of the computer code for integrity in the calculations. This should be an ongoing process, especially for newer models. Model verification should be documented, or the model or model component should be peer-reviewed by an independent party if it is new.

Model calibration: Using site-specific information from a historical period of time to adjust model parameters in the governing equations (e.g., bottom friction coefficient in hydrodynamic models) to obtain an optimal agreement between a measured data set and model calculations for the simulated state variables.

Model validation: Demonstrating that the calibrated model accurately reproduces known conditions over a different period of time with the physical parameters and forcing functions

changed to reflect the conditions during the new simulation period, which is different from that used for calibration. The parameters adjusted during the calibration process should NOT be adjusted during validation. Model simulations during validation should be compared to the measured data set. If an acceptable level of agreement is achieved between the data and model simulations, then the model can be considered validated as an effective tool, at least for the range of conditions defined by the calibration and validation data sets. If an acceptable level of agreement is not achieved, then further analysis should be carried out to determine possible reasons for the differences between the model simulations and measured data during the validation period. The latter sometimes leads to refinement of the model (e.g., using a finer model grid) or to the addition of one or more physical/chemical processes that are represented in the model.

It is important that both calibration and validation be conducted at the space and time scales associated with the questions the model must answer. For example, if the model will be used to make decade-scale predictions, when possible, it should be compared to decade-scale trend data. Even when data exist for a much shorter time period than will be used for prediction, the long-term behavior of the model should be examined as a part of the calibration process. It is not unusual for a model to perform well for a short-term period, but produce unreasonable results when run for a much longer duration. The extent to which components of a modeling study are performed using verified models can determine to a large degree the defensibility of the modeling project. If a verified model has not been sufficiently calibrated or validated for a specific site, then the modeling study may lack defensibility and be of little value. Where possible, project managers should use verified models in the public domain, calibrated and validated to site-specific conditions. Proprietary models may also be useful, but project managers should be aware they contain code that has not been shared publicly and may not have been verified. The interpretation of modeling results and the reliance placed on those results should heavily consider the extent of documented model verification, calibration, and validation performed.

2.9.5 Sensitivity and Uncertainty of Models

Another important tool for understanding model results may be a sensitivity analysis. This process typically consists of varying each of the input parameters by a fixed percent (while holding the other parameters constant) to determine how the predictions vary. The resulting variations in the state variables are a measure of the sensitivity of the model predictions to the parameter whose value was varied. This can be very informative, especially in understanding how the various processes being modeled affect contaminant fate and transport and which are dominant. This analysis is frequently used to identify the model parameters having the most impact on model results, so that the project team can ensure these parameters are well constrained by site data.

Uncertainty in models usually results from the following three principal sources:

- The necessity for models to use equations that are simplifications and approximations of complex processes, which can result in uncertainty in just how well the equations represent the actual processes;

- The uncertain accuracy of the values used to parameterize the equations (i.e., uncertainty about how well the input data represent actual conditions); and

- The uncertain accuracy of model assumptions about future conditions, when using the model for prediction, (e.g., assumptions about future rainfall, land use, or upstream contaminant sources).

Typically, uncertainty analyses focus on only the second source, the accuracy of the input values for the model. While quantitative uncertainty analyses are possible and practical to perform with watershed loading models and food chain/web models, they are generally not so (at the current time) for fate and transport models. If a quantitative assessment of the uncertainty of fate and transport model predictions could be provided, the value of that prediction would be greatly increased. Lacking a quantitative uncertainty analysis, one method modeling teams might consider to assess uncertainty is to use bounding calculations to produce a conservative model outcome to compare to the model s best estimate outcome. This conservative model outcome may be developed by using parameter values that result in a conservative outcome but do not result in significantly degraded model performance, as measured by comparison to the calibration and validation data sets. A second method to assess uncertainty involves quantification of model error by comparison of results to the calibration and validation data and application of that error to model predictions, as described in Connolly and Tonelli (1985).

2.9.6 Peer Review

It is EPA policy that a peer review of numerical models is often appropriate to ensure that a model provides decision makers with useful and relevant information. Project managers should use EPA s *Guidance for Conducting External Peer Review of Environmental Regulatory Models* (U.S. EPA 1994c) and the *Peer Review Handbook* (U.S. EPA 2000e) to determine whether a peer review of a model is appropriate and, if so, what type of peer review should be used. As a rule of thumb, when a model is being used outside the niche for which it was developed, is being applied for the first time, or is a critical component of a decision that is very costly, a peer review should be performed. In addition, project managers should refer to OSWER Directive 9285.6-08, *Principles for Managing Contaminated Sediments at Hazardous Waste Sites*, Principle 6 (U.S. EPA 2002a; see Appendix A).

EPA peer review guidance for models (U.S. EPA 1994c) also notes that environmental models that may form part of the scientific basis for regulatory decision making at EPA are subject to the peer review policy. However, it cannot be more strongly stressed that peer review should be considered only for judging the scientific credibility of the model including applicability, uncertainty, and utility (including the potential for misuse) of results and not for directly advising the Agency on specific regulatory decisions stemming in part from consideration of model output. Peer reviewers advise the Agency regarding proper use and interpretation of a model; it is then the Agency s task to apply that advice properly to regulatory decisions.

Highlight 2-15 summarizes some important points to remember about modeling at sediment sites.

Highlight 2-15: Important Principles to Consider in Developing and Using Models at Sediment Sites

1. **Consider site complexity before deciding whether and how to apply a mathematical model.** Site complexity and controversy, available resources, project schedule, and the level of uncertainty in model predictions that is acceptable, are generally the critical factors in determining the applicability and complexity of a mathematical model. Potential remedy cost and magnitude of risk are generally less important, but they can significantly affect the level of uncertainty that is acceptable.

2. **Develop and refine a conceptual site model that identifies the key areas of uncertainty where modeling information may be needed.** When evaluating if a model is needed and in deciding which models might be appropriate, a conceptual site model should be developed that identifies the key exposure pathways, the key sediment and water-body characteristics, and the major sources of uncertainty that may affect the effectiveness of potential remedial alternatives (e.g., capping, dredging, and/or MNR).

3. **Determine what model output data are needed to facilitate decision making.** As part of problem formulation, the project manager should consider the following: 1) what site-specific information is needed to make the most appropriate remedy decision (e.g., degree of risk reduction that can be achieved, correlation between sediment cleanup levels and protective fish tissue levels, time to achieve risk reduction levels, degree of short-term risk); 2) what model(s) are capable of generating this information; and 3) how the model results can be used to help make these decisions. Site-specific data collection should concentrate on input parameters that will have the most influence on model outcome.

4. **Understand and explain model uncertainty.** The model assumptions, limitations, and the results of the sensitivity and uncertainty analyses should be clearly presented to decision makers and should be clearly explained in decision documents such as proposed plans and RODs.

5. **Conduct a complete modeling study.** If an intermediate or advanced level model is used in decision making, the following components should be included in every modeling effort:
   - Model verification (or peer-review if a new model is used)
   - Model calibration
   - Model validation

6. **Consider modeling results in conjunction with empirical data to inform site decision making.** Mathematical models are useful tools that, in conjunction with site environmental measurements, can be used to characterize current site conditions, predict future conditions and risks, and evaluate the effectiveness of remedial alternatives in reducing risk. Modeling results should generally not be relied upon exclusively as the basis for cleanup decisions.

7. **Learn from modeling efforts.** If post-remedy monitoring data demonstrate that the remedy is not performing as expected (e.g., fish tissue levels are much higher than predicted), consider sharing these data with the modeling team to allow them to perform a post-remedy validation of the model. This could provide a basis for model enhancements that would improve future model performance at other sites. If needed, this information could also be used to re-estimate the time frame when RAOs are expected to be met at the site.

3.0 FEASIBILITY STUDY CONSIDERATIONS

Generally, the purpose of a feasibility study for a contaminated sediment site is to develop and evaluate a number of alternative methods for achieving the remedial action objectives (RAOs) for the site. This process lays the groundwork for proposing and selecting a remedy for the site that best eliminates, reduces, or controls risks to human health and the environment. The feasibility study process is described in the U.S. Environmental Protection Agency s (EPA s) *Guidance for Conducting Remedial Investigations and Feasibility Studies under CERCLA* (U.S. EPA 1988a, also referred to as the RI/FS Guidance). The proposed plan and record of decision (ROD) process is described in the EPA s *Guide to Preparing Superfund Proposed Plans, Records of Decision, and other Remedy Selection Decision Documents* (U.S. EPA 1999a, also referred to as the ROD Guidance). This chapter is intended to supplement existing guidance by offering sediment-specific guidance about developing alternatives, considering the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) criteria, identifying applicable or relevant and appropriate requirements (ARARs), estimating cost, and implementing institutional controls. Chapters 4, 5, and 6 present more detailed guidance on evaluating alternatives based on the three major approaches for sediment: monitored natural recovery (MNR), in-situ capping, and dredging (or excavation) with treatment or disposal.

Although this chapter focuses on remedial alternatives for managing contaminated sediment, project managers beginning this stage of site management should keep in mind the first step at almost every sediment site should be to implement measures to control any significant ongoing sources and to evaluate the effectiveness of those controls. Until this is done, appropriately evaluating alternatives for sediment may be difficult. However, it may be appropriate to evaluate implementation of interim sediment cleanup measures prior to completing source control to control further dispersal of sediment hot spots or reduce risks to human health and the environment due to sediment contamination.

In addition, project managers should keep in mind that flexibility is frequently important in the feasibility study process at sediment sites. Iterative or adaptive approaches to site management are likely to be appropriate at these sites. Also, project managers should consider pilot testing various approaches as part of the feasibility study process. Phasing, adaptive management, and early actions are described further in Chapter 2, Section 2.7, Phased Approaches, Adaptive Management, and Early Actions.

3.1 DEVELOPING REMEDIAL ALTERNATIVES FOR SEDIMENT

As described in Chapter 1, Section 1.3.1, Remedial Approaches, there are typically three major approaches that can be taken to reduce risk from contaminated sediment when source control measures are insufficient to reduce risks: MNR, in-situ capping, and sediment removal by dredging or excavation. Hybrid approaches may combine these three. A fourth approach, in-situ treatment, is currently under development and may become a viable alternative in the future, especially in combination with in-situ caps. Highlight 1-5 in Chapter 1 briefly summarizes these major approaches for sediment sites.

Project managers should consider the following steps, which build on EPA s RI/FS Guidance by adding details specific to sediment, when developing alternatives at sediment sites:

1. Develop remedial action objectives specifying the contaminants and media of interest, exposure pathways, and remediation goals that permit a range of alternatives to be

developed including each of the three major approaches (MNR, capping, and removal), and that consider state and local objectives for the site;

2. Identify estimated volumes or areas of sediment to which the approaches may be applied, taking into account the need for protectiveness as identified in the RAOs and the biological, chemical and physical characteristics of the site;

3. Develop additional detail concerning the equipment, methods, and locations to be evaluated for each alternative, including the three major approaches (e.g., potential natural recovery processes, potential cap materials and placement methods, number and types of dredges or excavators, transport methods, treatment methods, type of disposal units, general disposal location, need for monitoring and/or institutional controls);

4. Develop additional detail concerning known major constraints on each alternative, including the three major approaches at the site (e.g., need to maintain flow capacity for flood control, need to accommodate navigational dredging);

5. To the extent possible with information available at this stage of the FS, identify the time frame(s) in which the alternatives are expected to achieve cleanup levels and RAOs; and

6. Assemble the more detailed methods into a set of alternatives representing a range of options, including MNR, in-situ capping, and removal options or combination of options, as appropriate.

This process often is best done in an iterative fashion, especially at complex sites. For example, investigation into equipment and disposal options for sediment removal may lead to evaluation of a variety of time frames for achieving risk reduction goals. Typically, the number and type of remedial alternatives that a project manager develops for any site is a site-specific decision. The project manager should take into account the size, characteristics, and complexity of the site. However, due to the limited number of approaches that may be available for contaminated sediment, generally project managers should evaluate each approach carefully, including the three major approaches (MNR, in-situ capping, and removal through dredging or excavation) at every sediment site at which they might be appropriate.

3.1.1 Alternatives that Combine Approaches

At sites with multiple water bodies or sections of water bodies with differing characteristics or uses, or differing levels of contamination, project managers have found that alternatives that combine a variety of approaches are frequently the most promising. In many cases, institutional controls are also part of many alternatives (see Section 3.6, Institutional Controls). The following examples illustrate how different approaches might be combined into alternatives:

- An alternative might combine a variety of dredging, transport, and disposal methods that remove differing volumes of higher-risk contaminated sediment with MNR for more widespread areas of lesser risk;

- An alternative might combine armored in-situ capping of contaminated sediment in more erodible areas, with MNR in highly depositional areas;

- An alternative might combine dredging in federal navigation channels or for areas where there is insufficient water depth to maintain navigation or flood capacity with a cap, with in-situ capping of floodplain, intertidal or under-pier areas where a more technically practicable and less costly approach is desired; and

- An alternative might combine thin-layer placement (see Chapter 4, Monitored Natural Recovery) with MNR where the natural rate of sedimentation is insufficient to bury contaminants in a reasonable time frame.

3.1.2 No-Action Alternative

The NCP at Title 40 Code of Federal Regulations (40 CFR) §300.430(e)(6) provides that the no-action alternative should be considered at every site. The no action alternative should reflect the site conditions described in the baseline risk assessment and remedial investigation. This alternative may be a no-further-action alternative if some removal or remedial action has already occurred at the site, such as under another ROD.

No-action or no-further-action alternatives normally do not include any treatment, engineering controls, or institutional controls but may include monitoring. For example, at a site where risk is acceptable (e.g., because contaminant levels in surface sediment and biota are low and the site is stable), but the site contains higher levels of contamination at depth, it may be advisable to evaluate periodically the continued stability of buried contaminants. A no action alternative may include monitoring of these buried contaminants. Project managers and others should not confuse this however with MNR, where natural processes are relied upon to reduce an unacceptable risk to acceptable levels. The difference is often the increased level and frequency of monitoring included in the MNR alternative and the fact that the MNR alternative includes a cleanup level and expected time frame for achieving that level. Project managers should normally evaluate both a no action alternative and a MNR alternative at sediment sites.

If a no-action or no-further-action alternative does not meet the NCP s threshold criteria addressed in 40 CFR §300.430 (i.e., protection of human health and the environment and meeting applicable or relevant and appropriate requirements), it is not necessary to carry it though to the detailed analysis of alternatives. However, the ROD should explain why the no action alternative was dropped from the analysis. Chapter 7, Remedy Selection Considerations, includes guidance on when it may be appropriate to select a no-action alternative.

3.1.3 In-Situ Treatment and Other Innovative Alternatives

Generally, in-situ treatment is an approach that involves the biological, chemical, or physical treatment of contaminated sediment in place. This approach is currently under development by researchers and several pilot- and full-scale applications of the more promising technologies are underway. Although significant technical limitations currently exist for many of the treatment technologies, the results of the ongoing testing may demonstrate the viability of some of these approaches in certain situations. Project managers are encouraged to track the development of in-situ treatment methods. Potential in-situ treatment methods include the following:

- *Biological Treatment:* Enhancement of microbial degradation of contaminants by the addition of materials such as oxygen, nitrate, sufate, hydrogen, nutrients, substrate (e.g., organic carbon), or microorganisms into the sediment or into a reactive cap;

- *Chemical Treatment:* The destruction of contaminants through oxidation and dechlorination processes by providing chemical reagents, such as permanganate, hydrogen peroxide, or potassium hydroxide, into the sediment or into a reactive cap; and

- *Immobilization Treatment:* Solidification, stabilization, or sequestering of contaminants by adding coal, coke breeze, Portland cement, fly ash, limestone, or other additives to the sediment for encapsulating the contaminants in a solid matrix and/or chemically altering the contaminants by converting them into a less bioavailable, less mobile, or less toxic form.

Most techniques for in-situ treatment of sediment are in the early stages of development, and few methods are currently commercially available. Experiences gained to date in experimental or small-scale applications of in-situ remedies have indicated that technical limitations to the effectiveness of available in-situ treatments continue to exist. For example, in-situ remedies relying on the addition of required substrates and nutrients, reagents, or catalysts have been developed for some contaminants, such as polychlorinated biphenyls (PCBs), but developing an effective in-situ delivery system to add and mix the needed levels of reagents to contaminated sediment is more problematic. The lack of an effective delivery system has also hindered the application of in-situ stabilization systems [National Research Council (NRC) 2001]. However, new developments may make this a more promising approach in the future.

Several EPA-funded bench and field studies in this area are underway. These include studies conducted by EPA s Superfund Innovative Technology Evaluation (SITE) program, which encouraged the development and routine use of innovative treatment, monitoring, and measurement technologies. The SITE program is in the process of completing demonstration of several in-situ treatment technologies (Highlight 3-1). More information on the SITE program is available at http://www.epa.gov/ORD/SITE/. Also, the Hazardous Substance Research Center (HSRC) - South and Southwest, is performing research about in-situ treatment and other innovative capping alternatives for contaminated sediment in the Anacostia River in Washington, DC. More information on this program is available from the HSRC Web site at http://www.hsrc.org.

Highlight 3-1: SITE Program In-situ Treatment Technology Demonstrations

Site	Technology Type	Contaminant
Jones Island CDF (Confined Disposal Facility)	Phytoremediation	Polycyclic aromatic hydrocarbons (PAHs) and PCBs
Milwaukee Harbor	Phytoremediation	PAHs and PCBs
Whatcom Waterway, Puget Sound	Electrochemical Oxidation	Mercury and PAHs
Anacostia River	Multiple Reactive Caps	PAHs and PCBs

Other sources of information about innovative approaches to contaminated sediment management include the U.S. Army Corps of Engineers (USACE) Dredging Operations Environmental Research Program (DOER), which has contributed substantially to work in the area of risk assessment methods, fate and transport models, and dredging and capping technologies. Information on this program and on the Dredging Operations Technical Support (DOTS) program is available at http://el.erdc.usace.army.mil/dots. In addition, the Strategic Environmental Research and Development Program (SERDP) has made recent investments in contaminated sediment research. Information about these projects can be accessed from the SERDP Web site at http://www.serdp.org.

3.2 NCP REMEDY SELECTION CRITERIA

The NCP at 40 CFR §300.430(e)(9) establishes a framework of nine criteria for evaluating remedies. These criteria address the requirements of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), and additional technical and policy considerations that are important for selecting remedial actions. Many of these criteria are also important for actions under the Resource Conservation and Recovery Act (RCRA).

The NCP at 40 CFR §300.430(e)(7) describes a method for screening potential alternatives prior to developing detailed alternatives when a number of alternatives are being considered at a site. Only the alternatives judged as the best or most promising following this screening should be retained for further development and detailed analysis. The three broad criteria for screening preliminary remedial alternatives are: 1) effectiveness; 2) implementability; and 3) cost. Although a screening level analysis may be necessary in some cases, due to the relatively limited number of approaches available for sediment, project managers generally should not screen out any of the three major approaches early in the FS.

More detailed discussions of what should be addressed under each of the nine criteria can be found in the ROD Guidance (U.S. EPA 1999a) and the RI/FS Guidance (U.S. EPA 1988a). The following provides a summary of the nine criteria (U.S. EPA 1988a). More detailed explanations related to sediment sites are cited after each criterion, as appropriate.

Threshold Criteria

- *Overall Protection of Human Health and the Environment*: This criterion is used to evaluate how the alternative as a whole achieves and maintains protection of human health and the environment; and

- *Compliance with Applicable or Relevant and Appropriate Requirements (ARARs)*: This criterion is used to evaluate whether the alternative complies with chemical-specific, action-specific, and location-specific ARARs or if a waiver is justified. In addition to ARARs, this criterion also commonly includes whether the alternative considers other criteria, advisories, and guidance that are to be considered at the site. This criterion is discussed further with respect to contaminated sediment in Section 3.3.

Balancing Criteria

- *Long-Term Effectiveness and Permanence*: This criterion includes an evaluation of the magnitude of human health and ecological risk from untreated contaminated materials or treatment residuals remaining after remedial action has been concluded (known as residual risk), and the adequacy and reliability of controls to manage that residual risk. It also includes an assessment of the potential need to replace technical components of the alternative, such as a cap or a treatment system, and the potential risk posed by that replacement. This criterion is discussed further with respect to contaminated sediment in Section 3.4;

- *Reduction of Toxicity, Mobility, and Volume Through Treatment*: This criterion refers to the evaluation of whether treatment processes can be used, the amount of hazardous material treated, including the principal threat that can be addressed, the degree of expected reductions, the degree to which the treatment is irreversible, and the type and quantity of treatment residuals. This criterion is discussed further with respect to contaminated sediment in Chapters 4, 5, and 6 related to the individual remedies;

- *Short-Term Effectiveness*: This criterion includes an evaluation of the effects of the alternative during the construction and implementation phase until remedial objectives are met. This criterion includes an evaluation of protection of the community and workers during the remedial action, the environmental impacts of implementing the remedial action, and the expected length of time until remedial objectives are achieved. This criterion is discussed further with respect to contaminated sediment in Section 3.4;

- *Implementability*: This criterion is used to evaluate the technical feasibility of the alternative, including construction and operation, reliability, monitoring, and the ease of undertaking an additional remedial action if the remedy fails. It also considers the administrative feasibility of activities needed to coordinate with other offices and agencies, such as for obtaining permits for off-site actions, rights of way, and institutional controls, and the availability of services and materials necessary to the alternative, such as treatment, storage, and disposal facilities. This criterion is discussed further with respect to contaminated sediment in Chapters 4, 5, and 6 related to the individual remedies; and

- *Cost*: This criterion includes an evaluation of direct and indirect capital costs, including costs of treatment and disposal, annual costs of operation, maintenance, monitoring of the alternative, and the total present worth of these costs. This criterion is discussed further with respect to contaminated sediment in Section 3.5.

Modifying Criteria

- *State (Or Support Agency) Acceptance*: This criterion is used to evaluate the technical and administrative concerns of the state (or the support agency, in the case of state-lead sites) regarding the alternatives, including an assessment of the state or the support agency s position and key concerns regarding the alternative, and comments on ARARs or the proposed use of waivers. Tribal acceptance is also evaluated under this criterion.

This criterion is discussed further with respect to contaminated sediment in Chapter 1, Section 1.5; and

- *Community Acceptance*: This criterion includes an evaluation of the concerns of the public regarding the alternatives. It determines which component of the alternatives interested persons in the community support, have reservations about, or oppose. This criterion is discussed further with respect to contaminated sediment in Chapter 1, Section 1.6.

Additional guidance about how to apply these criteria to sediment alternatives is found throughout the guidance, as indicated above. In addition, Chapter 7, Remedy Selection Considerations, summarizes general considerations of each of the nine criteria with respect to the three major approaches.

3.3 APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS

Pursuant to CERCLA §121(d)(4), all remedial actions at CERCLA sites must be protective of human health and the environment. In addition, on-site actions need to comply with the substantive portions of ARARs unless the ARAR is waived. ARARs may be waived only under limited circumstances. Compliance with administrative procedures, such as permits, is not required for on-site response actions. Off-site actions must comply with both substantive and administrative requirements of legally applicable laws and regulations.

Sediment cleanup levels for response actions under CERCLA are generally based on site-specific risk assessments, but are occasionally based on ARARs. Project managers may also consider non-promulgated advisories or guidance issued by federal, state, or tribal governments, frequently called TBC (to be considered). While TBCs may not be legally binding on their own, and, therefore, do not have the same status as ARARs, TBCs can be used as a basis for making cleanup decisions. The project manager should refer to *CERCLA Compliance with Other Laws Manual* (U.S. EPA 1988b). Also, the preamble to the final NCP (55 *Federal Register* (*FR*) 8741) states that, as a matter of policy, it is appropriate to treat Indian tribes as states for the purpose of identifying ARARs (see NCP at 40 CFR §300.515(b) for provisions dealing with tribal governments).

The process of identifying ARARs typically begins in the scoping phase of the RI/FS, continues until the ROD is finalized, and may be reexamined during the five-year review process. Identification of ARARs should be done on a site-specific basis and usually involves a two-part analysis. First, a determination of whether a given requirement is applicable should be made, and second, if it is not applicable, then a determination should be made as to whether it is relevant and appropriate. Highlight 3-2 lists some examples of potential federal, state, and tribal ARARs for sediment sites and actual and hypothetical examples of how remedial strategies have been adapted to comply with ARARs.

For more information about ARARs, the project manager should consult the *Compendium of CERCLA ARARs Fact Sheets and Directives* (U.S. EPA 1991b), and the *Assessment and Remediation of Contaminated Sediments (ARCS) Program Remediation Guidance Document* (U.S. EPA 1994d).

As part of the ARARs analysis, project managers, in consultation with the site attorney, should consider appropriate requirements promulgated under the Clean Water Act (CWA). As described in the examples in Highlight 3-2, federal water quality criteria as well as state-promulgated regulations

including state water quality standards may be potential ARARs for surface water when water is discharged from dewatering or treatment areas or as effluent from confined disposal facilities (CDFs). Furthermore, some states may have their own promulgated sediment quality standards that may be potential ARARs for sediment.

Total maximum daily loads (TMDLs) established or approved by the EPA under the CWA are planning tools designed to reduce contributing point and nonpoint sources of pollutants in water quality limited segments (WQLS). TMDLs calculate the greatest amount of loading of a pollutant that a water body can receive without exceeding CWA water quality standards. TMDLs are usually established by the states, territories, or authorized tribes and approved by the EPA. Effluent limits in point source national pollutant discharge elimination system (NPDES) permits should be consistent with the assumptions and requirements in a wasteload allocation in an approved TMDL.

EPA-established TMDLs are not promulgated as rules, are not enforceable, and, therefore, are not ARARs. TMDLs established by states, territories or authorized Indian tribes may or may not be promulgated as rules. Therefore, TMDLs established by states, territories, or authorized Indian tribes, should be evaluated on a regulation-specific and site-specific basis. Even if a TMDL is not an ARAR, it may aid in setting protective cleanup levels and may be appropriately a TBC. Project managers should work closely with regional EPA Water program and state personnel to coordinate matters relating to TMDLs. The project manager should remember that even when a TMDL or wasteload allocation is not enforceable, the water quality standards on which they are based may be ARARs. TMDLs can also be useful in helping project managers evaluate the impacts of continuing sources, contaminant transport, and fate and effects. Similarly, Superfund s RI/FS may provide useful information and analysis to the federal and state water programs charged with developing TMDLs.

Project managers are also strongly encouraged to follow the consultation requirements of the Endangered Species Act. For on-site actions, the Endangered Species Act, Section 7, requires federal agencies to ensure that the actions they authorize, fund or carry out are not likely to jeopardize the continued existence of endangered or threatened species, or adversely modify or destroy their critical habitat. By policy, EPA consults with the U.S. Fish and Wildlife Service and the National Marine Fisheries Service (NMFS) where a threatened or endangered species or their habitat is or may be present. The Commencement Bay NPL (National Priorities List) site provides an example of how a remedial strategy has been adapted to comply with this act. Chinook salmon are threatened species that are found at this site during part of the year. After following EPA s policy of consulting with the NMFS, EPA decided that to avoid harming the species, some in-water remedial work would be conducted only during a window of time when juvenile salmon were not migrating through the area. Other in-water work would be performed outside of this window, using special conditions recommended by NMFS to minimize impacts to salmon.

Highlight 3-2: Examples of Potential ARARs for Sediment Sites		
Law or Regulation	**Description**	**Examples of How Remedial Strategies have been Adapted to Comply with ARARs**
Potential Federal ARARs		
Clean Water Act §304 40 CFR part 130	EPA publishes national recommended Ambient Water Quality Criteria (AWQC) for the protection of aquatic life and human health. CERCLA §121(d)(2) requires EPA to consider whether nationally recommended AWQC should be relevant and appropriate requirements at a site. CERCLA §121(d)(2)(B) establishes the guidelines to consider in determining when AWQC may be relevant and appropriate requirements, including consideration of the designated or potential uses of surface water, the purposes for which the criteria were developed and the latest information available.	In developing a remedy that included treatment of water following dewatering sediment, EPA determined that a revised AWQC was a relevant and appropriate criteria for discharging to the waterway.
Clean Water Act §404 33 CFR parts 320-330 and 40 CFR part 230	Regulates the discharge of dredged or fill materials into waters of the U.S. Discharges of dredged or fill materials are not permitted unless there is no practicable alternative that would have less adverse impact on the aquatic ecosystem. Any proposed discharge must avoid, to the fullest extent practicable, adverse effects, especially on aquatic ecosystems. Unavoidable impacts must be minimized, and impacts that cannot be minimized must be mitigated.	Work at the ASARCO, Tacoma Washington, National Priorities List (NPL) site included construction of an armored cap in the inter-tidal zone. Work at the Wyckoff/Eagle Harbor, Washington, NPL site included construction of a sheet pile barrier wall to control subsurface non-aqueous phase liquid (NAPL) migration. To compensate for the loss of habitat, intertidal habitat was created in another part of these two sites. Work at the Lavaca Bay, Texas site involved construction of a CDF with effluent discharge to the Bay. CDF effluent discharged to waters of the U.S. is defined as the discharge of dredged material under EPA and USACE regulations implementing Section 404 (40 CFR §232.2).

Law or Regulation	Description	Examples of How Remedial Strategies have been Adapted to Comply with ARARs
Resource Conservation and Recovery Act (RCRA); 40 CFR parts 260 to 268	Dredged material may be subject to RCRA regulations if it contained a listed waste, or if it displays a hazardous waste characteristic, for example, by the Toxicity Characteristic Leaching Procedure (TCLP). Most states have been authorized in lieu of EPA to implement the RCRA program. RCRA regulations may be potentially ARARs for the storage, treatment, and disposal of the dredged material unless an exemption applies. One such exemption is if CWA 404 applies to the cleanup activity (40 CFR part 261).	The material to be dredged contains a listed pesticide formulation waste, and thus RCRA may be a applicable. However, the site is located in a state where EPA implements the RCRA program, and the on-site cleanup action will comply with substantive requirements of a 404 permit. Thus the cleanup action is exempted from RCRA. This situation is explained in the description of the selected remedy in the ROD.
Rivers and Harbors Act, Section 10 33 CFR parts 320 to 323	Activities that could impede navigation and commerce are prohibited. Prohibits authorized obstruction or alteration of any navigable waterway.	A site with contaminated sediment has an authorized navigation depth of 30 ft. The evaluation of alternatives needs to consider the need to maintain this minimum depth when evaluating whether capping is or is not a feasible alternative for the entire site.
Toxic Substances Control Act (TSCA) 40 CFR part 761	Section 6(e) of TSCA regulates PCBs from cradle to grave (i.e., from manufacture to disposal). TSCA and portions of its implementing regulations may be an ARAR for on-site response actions involving contaminated sediment. The regulations provide several factors for determining whether PCB contaminated media is PCB remediation waste (as defined per 40 CFR §761.3), including the date of the spill, PCB concentration of material spilled, and PCB concentration currently at the site (i.e., the □as found□ concentration.) In general, material meeting the definition of PCB remediation waste may be disposed of using one of the three options under 40 CFR §761.61, which includes a self-implementing option (40 CFR §761.61(a)), a performance-based option (40 CFR §761.61(b)), and a risk-based option (40 CFR §761.61(c)). Under the regulations, however, the self-implementing option cannot be used to clean up sediments in marine or freshwater ecosystems (see 40 CFR §761(a)(1)(i)).	Example: A determination was made to identify PCB remediation waste by sampling the sediments. Based on the definition of PCB remediation waste (40 CFR §761.3), as the spill occurred prior to 1978, those sediments with PCB concentrations greater than 50 ppm are considered PCB remediation wastes. The risk-based option (under 40 CFR §761.61(c)) for PCB remediation waste is selected (the self-implementing option at 40 CFR §761.61(a) is not available for sediments). A site-specific disposal plan is prepared that includes a sites specific sampling protocol as well as detailed performance standards for on-site temporary storage and off-site disposal for dredged sediments. After determining that this approach will not pose an unreasonable risk of injury to health or the environment (as specified in 40 CFR §761.61(c)), the Regional Administrator approves the plan.

Law or Regulation	Description	Examples of How Remedial Strategies have been Adapted to Comply with ARARs
	Selection of disposal options under 40 CFR §761.61 for wastes generated at a Superfund site is generally made at the regional level. The risk-based option under 40 CFR §761.61(c) may often be the most appropriate option at Superfund sites. In appropriate circumstances, the risk-based option may allow disposal of PCB remediation wastes with <50 ppm in a municipal landfill. Substantive TSCA requirements also exist for storage and other activities involving PCB contaminated wastes.	
Potential State and Tribal ARARs		
State Water Quality Standards Regulation	Under the CWA, states are required to designate surface water uses, and to develop water quality standards based on those uses and the AWQC. Often an applicable requirement for discharges to surface water. Where an Indian tribe has promulgated water quality standards, these may also be an applicable requirement.	A tribe has an EPA approved water quality standard regulation which designates the uses of a river to include rearing of aquatic life and other uses. Design and construction of the selected remedy, including the confined aquatic disposal facility, needs to achieve or waive the tribe's water quality standards based on that use.
State Hazardous Waste Regulations	Many states have been authorized by EPA to implement the RCRA Subtitle C Hazardous Waste Program in lieu of EPA.	The sediment at a site was contaminated with a listed hazardous waste. The state has been authorized for RCRA, and decided to not adopt the hazardous waste identification rule (HWIR) sediment exemption. Treatment and disposal of the dredged contaminated sediment must meet or waive the state's hazardous waste regulations.
State Solid Waste Regulations	Most states have regulations for the location, design, construction, operation and closure of solid waste management facilities. Potential applicable or relevant and applicable requirement for disposal of non-hazardous waste contaminated sediment.	A remedial alternative includes on-site upland disposal of dredged sediment. The feasibility study examines the state solid waste regulations and determines that a disposal facility at two of the three possible sites can be designed to meet the ARAR. The third site is eliminated from further analysis.

Law or Regulation	Description	Examples of How Remedial Strategies have been Adapted to Comply with ARARs
Total Maximum Daily Load (TMDL) Regulation	Some states have established wasteload allocations in State-promulgated and EPA-approved TMDLs. These allocations may be an applicable or a relevant and appropriate requirement, where promulgated by the state as an enforceable regulation. Non-promulgated TMDLs may be a TBC.	A remedial dredging alternative includes an expected temporary increase in total suspended solids in the water body and residual contamination that provides a small continuing load to the water body. EPA consulted with the state TMDL program to determine whether TMDLs are a potential ARAR or TBC and how they interact with the alternative.
National Pollutant Discharge Elimination System (NPDES) Permit Regulations	Under the CWA, many states have been delegated the authority for the NPDES permit program. These regulations generally regulate discharges, including monitoring requirements and effluent discharge limitations for point sources. Where a remedy has a point discharge that is on-site, the substantive requirements may be an applicable regulation.	A Superfund remedy includes ground water remediation with discharge of the water to surface water. EPA consulted with the state NPDES permit program to determine water treatment standards prior the discharge.

Project managers are also strongly encouraged to follow the consultation requirements of the National Historic Preservation Act, Section 106 (36 CFR part 800). Section 106 requires federal agencies to consider the effects of their actions on historic properties that are on or are eligible for listing on the National Register of Historic Places. Compliance generally includes conducting a preliminary survey to determine the presence of significant resources, including among others, historic, prehistoric, archeological, architectural, engineering or cultural resources. If significant resources are found, generally a documentation package is prepared for review and comment by the State or Tribal Historic Preservation Office and appropriate mitigation is included in site plans. Examples of how remedial strategies have been adapted to comply with this Act include the Pine Street Canal Site in Vermont, where mitigation for damages related to capping sunken barges and other historic features included study and artifact collection by a local maritime museum related to a historic sunken barge of similar type in nearby Lake Champlain. In addition, at the Fox River PCB (polychlorinated biphenyl) site in Wisconsin, historic and prehistoric artifacts will be protected during nearby site activities and a potential shipwreck site will either be avoided during dredging or a diver study employed for further examination.

Project managers should also be aware of Executive Orders such as those covered by the *Statement of Procedures on Floodplain Management and Wetland Protections* (Appendix A of 40 CFR part 6). Although not ARARs, the Agency normally follows Executive Orders as a matter of policy. The Statement of Procedures cited above sets forth EPA policy and guidance for carrying out Executive Orders 11988 and 11990, which were written in furtherance of the National Environmental Policy Act (NEPA) and other environmental statutes. Executive Order 11988 concerns floodplain management and the evaluation by federal agencies of the potential effects of actions they may take in a floodplain to avoid, to the extent possible, adverse effects associated with direct and indirect development of a floodplain. Executive Order 11990 concerns protection of wetlands and the avoidance by federal agencies, to the extent possible, of the adverse impacts associated with the destruction or loss of wetlands if a practical alternative exists. OSWER Directive 9280.0-03, *Considering Wetlands at CERCLA Sites* (U.S. EPA 1994e), contains further guidance on addressing this Executive Order.

Examples of ways in which remedial strategies for sediment have been adapted in light of these Executive Orders as a matter of policy include the following:

- EPA determined that capping above grade would be an inappropriate alternative for remediating contaminated sediment in a small river, as the increased bottom elevation would increase the risk of flooding. Instead, the final EPA remedy called for dredging contaminated sediment and capping back to the existing grade; and

- EPA selected a route that avoided the wetland and would minimize the potential for effects on the floodplain, after evaluating possible alignments for the access road to the contaminated sediment site. During design of the access road, additional features were incorporated to further minimize any indirect impact on the floodplain.

3.4 EFFECTIVENESS AND PERMANENCE OF SEDIMENT ALTERNATIVES

Two NCP balancing criteria for which project managers of sediment sites may find additional guidance helpful are those related to short-term effectiveness, and long-term effectiveness and permanence. Each is described in more detail below, as it relates to evaluation of contaminated sediment

alternatives. The NCP describes the assessment of short-term effectiveness as follows 40 CFR §300.430(e)(9)(iii)(E)):

> The short-term impacts of alternatives shall be assessed considering the following:
>
> (1) Short-term risks that might be posed to the community during implementation of an alternative;
>
> (2) Potential impacts on workers during remedial action and the effectiveness and reliability of protective measures;
>
> (3) Potential environmental impacts of the remedial action and the effectiveness and reliability of mitigative measures during implementation; and
>
> (4) Time until protection is achieved.

For contaminated sediment alternatives, short-term risks to the community and workers may include those that may occur during dredging or capping operations or during the first few years of a MNR remedy. For a sediment remedy involving bioaccumulative contaminants, short-term impacts may include those due to continued human or ecological exposure to contaminants currently in the food chain. For a MNR alternative, these impacts may also be frequently due to continued human and ecological exposure to contaminants in surface sediment. For in-situ capping, short-term impacts may be due to factors such as contaminant releases during capping or accidents during transport or placement of cap material. For dredging or excavation, short-term impacts may include those due to contaminant releases during sediment removal, transport, treatment, or disposal or accidents during construction and operation of facilities. Short-term impacts to the benthic community as a result of capping or dredging should also be considered. Additional possible short-term impacts are presented in Highlight 7-3, Examples of Some Key Differences Between Remedial Approaches for Contaminated Sediment.

The time needed until protection is achieved can be difficult to assess at sediment sites, especially where bioaccumulative contaminants are present. Generally, for sites where risk is due to contaminants in the food chain, time to achieve protection can be estimated using models. These models may have significant uncertainty, but may be useful for predicting whether or not there are significant differences between time to achieve protection using different alternatives. When comparing time to achieve protection from MNR to that for active remedies such as capping and dredging, it is generally important to include the time for design and implementation of the active remedies in the analysis.

The NCP describes the assessment of long-term effectiveness and permanence as follows (40 CFR §300.430(e)(9)(iii)(C)):

> Alternatives shall be assessed for the long-term effectiveness and permanence they afford, along with the degree of certainty that the alternative will prove successful. Factors that shall be considered, as appropriate, include the following:
>
> (1) Magnitude of residual risk remaining from untreated waste or treatment residuals remaining at the conclusion of the remedial activities. The characteristics of the residuals should be

considered to the degree that they remain hazardous, taking into account their volume, toxicity, mobility, and propensity to bioaccumulate; and

(2) Adequacy and reliability of controls such as containment systems and institutional controls that are necessary to manage treatment residuals and untreated waste. This factor addresses in particular the uncertainties associated with land disposal for providing long-term protection from residuals; the assessment of the potential need to repair or replace technical components of the alternative, such as a cap, a slurry wall, or a treatment system; and the potential exposure pathways and risks posed should the remedial action need replacement.

For contaminated sediment alternatives, residual risk generally may be considered to be the risk remaining after completion of dredging, capping, or MNR. In their evaluation of residual risk, project managers should consider the volume, toxicity, mobility, and bioavailability of the remaining contaminants, as well as their propensity to bioaccumulate. The adequacy and reliability of controls used to manage post-remediation sediment residuals or untreated contamination that remains in the sediment should also be considered. Where institutional controls such as fish consumption advisories are one of the controls used to manage residual risk, project managers should assess their expected effectiveness and whether resulting exposures are expected to be within protective levels. Developing answers to the following questions may help the project manager in evaluating the long-term effectiveness and permanence of alternatives:

- What is the likelihood that the planned cap, dredging approach, or MNR will meet the cleanup levels and RAOs?
- What is the level of human health and/or ecological risk remaining after implementation?
- What is the expected pattern of risk reduction over time for the various alternatives and what uncertainties are associated with that pattern?
- How much of the risk is due to the area that was remediated versus unremediated areas of contamination?
- What type and degree of long-term operation and maintenance (O&M) will be required?
- What are the requirements for long-term monitoring?
- What is the potential need for replacing or modifying the technical components of the alternative?
- What is the magnitude of risk should the remedy fail? and
- What is the degree of confidence that there are adequate controls to identify and prevent remedy failure?

It is important to remember that each of the three major approaches may be capable of reaching acceptable levels of both short-term effectiveness and long-term effectiveness and permanence, and that site-specific characteristics should be reviewed during the alternatives evaluation to ensure that the

selected alternative will be effective in that environment. Project managers should evaluate and compare the effectiveness of in-situ (capping and MNR) and ex-situ (dredging) alternatives under the conditions present at the site. There should not be necessarily a presumption that removal of contaminated sediments from a water body will be necessarily more effective or permanent than capping or MNR. Likewise, without sufficient evaluation there should not be a presumption that capping or MNR will be effective or permanent. What constitutes an acceptable level of effectiveness and permanence is a site-specific decision that should also consider each of the other NCP remedy selection criteria. Each of the major approaches for sediment has its own remedy-specific considerations under these criteria, which are summarized below. Some aspects are discussed in more detail in the following remedy-specific chapters.

Monitored Natural Recovery

For a MNR remedy, the risk present at the time of remedy selection should decrease with time as natural processes progress. The level of risk reduction afforded by this remedy generally depends on what cleanup levels the natural processes are expected to be able to achieve in a reasonable time frame and the level of contamination which may continue to enter the system from any uncontrolled sources.

Residual risk following MNR and permanence for a MNR alternative frequently are related to the stability of the sediment bed, or the chance that clean sediment overlying buried contaminants may be eroded to such an extent that unacceptable risk is created. Residual risk for an MNR remedy may also be related to the chance that ground water flow, bioturbation, or other mechanisms may move buried contaminants to the surface where they could cause unacceptable human or ecological exposure, even in otherwise stable, non-erosional sediment. Whether erosion, ground water flow, or other processes cause unacceptable risk depends on the rate of exposure due to those processes. For example, erosion of some portions of a sediment bed, or some movement of contaminants through bioturbation, may not create an unacceptable risk; therefore, it is important to review such factors on a site-specific basis. Evaluating the adequacy of controls for these risks in an MNR remedy may include evaluating the ability of the monitoring plan to detect significant sediment erosion or contaminant movement, and evaluating the adequacy of any institutional controls that are relied upon to control erosion (e.g., dam or breakwater maintenance agreements).

In-Situ Capping

For an in-situ capping remedy, risk due to direct exposure to contaminated sediment in the capped area generally decreases rapidly, although risks may remain from uncapped areas. The level of risk reduction associated with this remedy generally depends on the action level selected for capping (i.e., what level of contamination will remain outside the capped area) and the level of contamination that may continue to enter the system from any uncontrolled sources. Residual risk, after the cap is in place, usually is related to the following: 1) likelihood of cap erosion or disruption exposing contaminants; 2) likelihood of contaminants migrating through the cap; and 3) risks from contaminants remaining in uncapped areas. Like MNR, whether cap erosion or contaminant migration through a cap cause unacceptable risk depends on depends on the rate of exposure due to those processes. An evaluation of long-term effectiveness and permanence for capping also should include an evaluation of the ability to monitor the effectiveness of the cap and to replace or replenish components of the cap through time before any significant contaminant releases occur.

Dredging or Excavation

For a dredging or excavation remedy, risks within the site itself may initially increase due to increased exposure to contaminants released into the surface water during sediment removal, but this increase should be temporary and localized. After this time, risk should decrease. The speed of the decrease and the level of long-term risk reduction associated with this remedy generally depends on the action level and/or cleanup levels selected for sediment removal (i.e., what level of contamination will remain outside of the dredged/excavated area), the level of residual contamination in the area after dredging, and the level of contamination that may continue to enter the system from any uncontrolled sources.

Residual risk, after the dredging or excavation is complete, is usually related to the following: 1) risk from contaminated sediment left behind outside of the dredged or excavated areas and from contaminated sediment resuspended and transported by dredging; 2) residual contamination left in place after dredging (an estimate of the likely post-dredging/post-backfilling surficial contamination levels should be developed); and 3) risk posed by untreated contaminants and treatment residuals at their disposal location. Similar to capping, the long-term effectiveness evaluation should include the need to replace technical components of the remedy after remedial action is completed. For dredging or excavation, this usually focuses on technical components of any on-site disposal units and the need to replenish backfill material in the dredged areas if backfill was used.

Project managers should recognize that all approaches for remediating sediment leave some contaminants in place after remedial actions are completed, whether buried beneath a natural sediment layer or engineered cap, left near the surface or mixed with backfill as residuals following dredging or excavation, or as low levels of contamination outside of areas that were capped or dredged. All of these residual contaminants are affected by a variety of natural processes that can disperse, contain or sequester them. As described above and in the three remedy-specific chapters of this guidance that follow, MNR, in-situ capping, and sediment removal, each may be capable of achieving acceptable levels of effectiveness and permanence. Site-specific site characteristics should be reviewed to ensure that the selected alternative will provide adequate short-term and long-term effectiveness at a particular site.

3.5 COST

Developing accurate cost estimates generally is an essential part of evaluating alternatives. It is also appropriate at many sites, and can be especially useful at large sites, to include the relative cost of achieving different cleanup levels. This typically is an important part of evaluating the cost-effectiveness of a range of protective alternatives which may, for example, be associated with different fish consumption rates or different levels of ecological protection.

Guidance on preparing cost estimates and the general role of cost in remedial alternative selection is discussed in *A Guide to Developing and Documenting Cost Estimates During the Feasibility Study* (U.S. EPA and USACE 2000). The general elements of a cost estimate include capital costs, annual and periodic O&M costs, and net present value (U.S. EPA and USACE 2000). A cost estimate prepared as part of the CERCLA cleanup process should not include potential claims for natural resource damages or potential restoration credits, but may include costs for mitigation of habitat lost or impaired by the remedial action, where appropriate.

3.5.1 Capital Costs

Capital costs generally are those expenditures needed to construct a remedial action (U.S. EPA and USACE 2000). Capital costs include only those expenditures initially incurred to implement a remedial alternative and major capital expenditures in future years. Capital cost elements that may be important at sediment sites include those listed in Highlight 3-3. As indicated in the Highlight, capital costs may include construction monitoring and environmental monitoring before, during and immediately following the remedial action. Monitoring beyond that point should be considered part of O&M.

Highlight 3-3: Examples of Categories of Capital Costs for Sediment Remediation

Categories	Capital Costs
General (may apply to several or all remedial approaches)	• Mobilization/demobilization • Site preparation (e.g., fencing, roads, utilities) • Construction monitoring, sampling, testing, and analysis before, during, and immediately following construction (e.g., bathymetric surveys) • Environmental monitoring before, during, and immediately following construction (e.g., water quality monitoring) • Debris and/or structure (e.g., piers, pilings) removal and disposal • Project management and support throughout construction, including preparation of remedial action documentation and construction submittals • Engineering needs during construction (not pre-construction design) • Post-construction habitat restoration (e.g., plantings) • Pilot studies • General contingency • Indirect costs • Implementation of institutional controls
Monitored Natural Recovery	• Monitoring and reporting prior to attainment of cleanup levels
In-situ Capping	• Cap materials – Material costs – Equipment and labor costs – Cost of mitigation if required under CWA §404 • Transport, storage, and placement of cap materials – Barge/tug lease costs – Stockpiling of cap material – Land use cost

Categories	Capital Costs
Dredging or Excavation	• Dredging or excavation equipment and labor costs • Engineering controls to protect water quality (e.g., silt curtains) • Site decontamination for support facilities (e.g., truck wash, dewatering area) • Sediment isolation for excavation (e.g., sheetpile, earthen dams) • Construction of dewatering area/temporary storage of dredged material • Transporting sediment to treatment or disposal site – Barge/tug lease costs – Pipeline costs • Land acquisition costs for construction easements or relocating utilities
Pretreatment/Treatment	• Land acquisition costs • Construction of pretreatment/treatment/storage buildings • Treatment of sediment • Treatment and discharge of water from dewatering process • Engineering controls to protect water quality (e.g., process water and storm water runoff controls) • Disposal of treatment residuals
In-Water Contained Aquatic Disposal, In-Water or Upland Confined Disposal Facilities	• Land acquisition or use costs • Construction of disposal site and any associated disposal costs – Demolition of existing facilities – Excavation to support berm – Equipment and labor costs • Berm construction – Imported materials for berm – Equipment costs • Capping disposal site – Cap materials – Equipment and labor costs • Engineering controls to protect water quality • Cost of mitigation if required under CWA §404

Categories	Capital Costs
Upland Landfill Disposal	• Land acquisition costs • Construction costs • Transportation costs • Tipping fees for regional landfill

The basis for a cost estimate may include a variety of sources, including cost curves, generic unit costs, vendor information, standard cost estimating guides, and similar estimates, as modified for the specific site. Where site-specific costs are available from pilot studies or removal actions, they are likely to be the best source of realistic cost information. Where this is not available, actual costs from similar projects implemented at other sites is frequently the next best source of costs.

Substantial amounts of historical cost data for some components of sediment remediation (e.g., removal, transport, disposal, and residue management) may be available from other project managers. EPA s Office of Superfund Remediation and Technology Innovation (OSRTI) can help project managers locate sites where a similar approach has been implemented. Additionally, the project manager may find it useful to refer to the ARCS program s remediation guidance document (U.S. EPA 1994d) for a discussion on the general elements of cost estimates for sediment sites. This document provides examples of percentages for general costs and site-specific costs for both in-situ and ex-situ remedies. Also, many of the local district USACE offices have extensive experience with dredging and in-water construction and may be an additional source of good cost information.

3.5.2 Operation and Maintenance (O&M) Costs

O&M costs are generally those post-construction costs necessary to ensure or verify the continued effectiveness of a remedial action (U.S. EPA and USACE 2000). These costs may be annual or periodic (e.g., once only, or once every five years). It is important to note that short-term O&M costs generally are incurred as part of the remedial action phase of a project, while long-term O&M costs or long-term cap maintenance generally are part of the O&M phase of a project (U.S. EPA and USACE 2000). At Fund-lead sites, it can be very important to differentiate these two cost categories because CERCLA has specific requirements addressing payment for long-term O&M [CERCLA 104(c))(3)), see Section 3.5.4, State Cost Share]. Some examples of categories that are generally considered short-term O&M at sediment sites include the following:

- Operation of sediment or water treatment facilities during the remedial action;
- Monitoring, sampling, testing, analysis, and reporting during the remedial action (some may be considered capital costs, see Section 3.5.1 above);
- Maintenance of in-situ cap or on-site disposal site during the shake-down period (e.g., one year);
- Maintenance of engineering site controls during shake-down period (e.g., one year);

- Cost overrun contingency; and
- Project management and support.

Some examples of categories that are generally considered long-term O&M at sediment sites include the following:

- Maintenance and monitoring of institutional controls;
- Long-term monitoring, sampling, testing, analysis, and reporting;
- Long-term maintenance of in-situ cap or on-site disposal unit; and
- Long-term maintenance of engineering site controls.

Additional issues related to long-term monitoring and maintenance of all three remedial approaches (MNR, capping, and dredging or excavation) are discussed in Chapter 8 of this guidance.

3.5.3 Net Present Value

The NCP also provides that an analysis of remedy net present value, or present worth, should be used [NCP §300.430(e)(9)(iii)(G)]. A net present value analysis should be used to compare expenditures occurring over different time periods. This standard methodology allows for a cost comparison of different alternatives having capital, O&M, and monitoring costs that would be incurred in different time periods on the basis of a single cost figure for each alternative. In general, the period of analysis should be equivalent to the project duration, resulting in a complete life cycle cost estimate for implementing the remedial alternative. Past EPA guidance recommended the general use of a 30-year period of analysis for estimating present value costs (U.S. EPA 1988a). Although this may be appropriate in some circumstances, the blanket use of a 30-year period is no longer recommended. Site-specific justification should be provided for the period of analysis selected, especially when the project duration (i.e., time period required for design, construction, O&M, and closeout) exceeds the selected period of analysis (U.S. EPA and USACE 2000).

For sediment approaches that leave significant quantities of contaminated sediment in place, such as in-situ capping or MNR based on natural burial, the actual monitoring period is likely to be longer than 30 years, although project managers are encouraged not to assume that monitoring in perpetuity will be necessary at every site. This is discussed further in Chapter 8, Remedial Action and Long-Term Monitoring.

The discount rate that should be used for this analysis is established by the Office of Management and Budget (OMB). Based on current Agency policy, as reflected in the NCP preamble (55 *FR* 8722) and the OSWER Directive 9355.3-20, *Revisions to OMB Circular A-94 on Guidelines and Discount Rates for Benefit-Cost Analysis* (U.S. EPA 1993b), a seven percent discount rate should be used in estimating the present worth value for potential alternatives. This figure could be revised in the future, and project managers should use the current figure contained in an update of the OMB Circular. Project managers should be aware that this rate may not be the same as rates that various potentially responsible parties (PRPs) or federal facilities use for similar analyses. The project manager should refer to *A Guide to*

Developing and Documenting Cost Estimates for the Feasibility Study (U.S. EPA and USACE 2000) for more information.

3.5.4 State Cost Share

At Fund-lead sites, generally the state is responsible under CERCLA for ten percent of remedial action costs and 100 percent of long-term O&M costs (see also 40 CFR □300.510(b) and (c)). Other requirements may apply if the facility was publicly operated at the time of disposal of hazardous substances and for federal facilities. Where O&M costs are significantly different between alternatives, this may add to differences of opinion about preferred alternatives. For the discussion to be based on the best available information, it is especially important that cost estimates be as accurate as possible, including costs of long-term O&M.

After a joint EPA/state inspection of an implemented Fund-financed remedial action, EPA may share, for a period of up to one year, in the cost of the operation of the remedial action to ensure that the remedy is operational and functional (40 CFR □300.510(c)(2)). For sediment sites, this may arise at sites involving in-situ caps and on-site disposal facilities.

The RAOs at sediment sites typically address sediment and biota, but remedies may also include surface water restoration as a goal of the remedial action. The NCP specifies the following in 40 CFR □300.510(c)(2):

> In the case of the restoration of ground or surface water, EPA shall share in the cost of the state s operation of ground or surface water restoration remedial actions as specified in 40 CFR □300.435(f)(3).

The NCP at 40 CFR □300.435(f)(3) specifies that:

> For Fund-financed remedial actions involving treatment or other measures to restore ground- or surface-water quality to the level that assures protection of human health and the environment, the operation of such treatment or other measures for a period of up to 10 years after the remedy becomes operational and functional will be considered part of the remedial action. Activities required to maintain the effectiveness of such treatment or other measures following the 10-year period, or after remedial action is complete, whichever is earlier, shall be considered O&M.

In 40 CFR □300.435(f)(3) and (4), the NCP also addresses when a restoration activity can be considered administratively complete for purposes of federal funding and discusses several actions that are excluded from consideration under this provision.

Where a sediment site includes surface water restoration as a goal, the project manager should consult with their Office of Regional Counsel to determine how these provisions may apply to their site.

3.6 INSTITUTIONAL CONTROLS

The term institutional control (IC) generally refers to non-engineering measures intended to affect human activities in such a way as to prevent or reduce exposure to hazardous substances, often by

limiting land or resource use. ICs can be used at all stages of the remedial process to reduce exposure to contamination. Chapter 7, Remedy Selection Considerations, offers guidance on when it may be appropriate to select a remedy that includes institutional controls at sediment sites and considerations regarding their effectiveness and enforceability. For more detailed information on ICs in general, refer to OSWER Directive 9355.0-74FS-P, *Institutional Controls: A Site Manager's Guide to Identifying, Evaluating, and Selecting Institutional Controls at Superfund and RCRA Corrective Action Cleanups* (U.S. EPA 2000f) and Federal Facilities Restoration and Reuse Office (FFRRO) guidance, *Institutional Controls and Transfer of Real Property under CERCLA Section 120 (h)(3)(A), (B), or (C)* (U.S. EPA 2000g).

As explained in the site managers guide cited above (U.S. EPA 2000f), the following are the four general categories of ICs:

- Governmental controls;
- Proprietary controls;
- Enforcement and permit tools with IC components; and
- Information devices.

Usually, governmental controls (e.g., bans on harvesting fish or shellfish) are implemented and enforced by the state or local government. Proprietary controls (often referred to as deed restrictions), such as easements or covenants, typically involve legal instruments placed in the chain of title of the site or property. Where enforcement tools are used to implement ICs, they may include provisions of CERCLA Unilateral Administrative Orders (UAOs), Administrative Orders on Consent (AOCs), or Consent Decrees (CD). Information devices are designed to provide information or notification to the public. The three most common types of ICs at sediment sites include fish consumption advisories and commercial fishing bans, waterway use restrictions, and land use restriction/structure maintenance agreements. Each of these ICs is discussed in more detail below.

Fish Consumption Advisories and Fishing Bans

Fish consumption advisories are informational devices that are frequently already in place and incorporated into sediment site remedies. Commercial fishing bans are government controls that ban commercial fishing for specific species or sizes of fish or shellfish. Usually, state departments of health are the governmental entities that establishes these advisories and bans. Frequently, fish consumption advisories and fishing bans are in place before a site is listed on the NPL, but if not, it could be necessary for the state to issue or revise them in conjunction with an early or interim action, or the final remedial action. An advisory usually consists of informing the public that they should not consume fish from an area, or consume no more than a specified number of fish meals over a specific period of time from a particular area. Sensitive sub-populations or subsistence fishers may be subject to more stringent advisories. Advisories can be publicized through signs at popular fishing locations, pamphlets, or other educational outreach materials and programs. Information should be provided in appropriate languages to meet the needs of the impacted communities. However, project managers should be aware that consumption advisories are not enforceable controls and their effectiveness can be extremely variable. This is discussed further in Chapter 7, Remedy Selection Considerations.

Waterway Use Restrictions

For any alternative where subsurface contamination remains in place (e.g., capping, MNR, or an in-water confined disposal site), waterway use restrictions may be necessary to ensure the integrity of the alternative. Examples include restricting boat traffic in an area to establish a no-wake zone, or prohibiting anchoring of vessels. In considering boating restrictions, it is important to determine who can enforce the restrictions, and under what authority and how effective such enforcement has been in the past. In addition, a restriction on easements for installing utilities, such as fiber optic cables, can be an important mechanism to help ensure the overall protectiveness of a remedy. It may also be necessary to evaluate remedial alternatives that involve changing the navigation status of a waterway. For a federally authorized navigation channel, deauthorization or reauthorization of the channel to a different width and/or depth configuration would be required and should be fully investigated before selecting the remedy. The state may also have additional authority to change harbor lines or the navigation status of a waterway.

Federal deauthorization can be a lengthy process that requires a formal request to the USACE, an opportunity for users of the waterway to comment, and, ultimately, deauthorization by Congress. By comparison, for those waterways or portions of waterways the USACE has placed in caretaker status (i.e., not actively maintained), channel reauthorization to widths and depths consistent with local requirements (e.g., to support continued recreational use) can be completed relatively quickly. Proposed channel modifications/reauthorizations are typically processed by congressional conferees and may be incorporated into the Water Resources Development Act (WRDA) or other equivalent legislative vehicles.

In designing caps to be placed within federal navigational channels, horizontal and vertical offsets, developed by the USACE based on considerations of normal dredging accuracy and overdepth allowances, can provide a factor of safety to protect the surface of the cap from potential damage during potential future maintenance dredging activities.

Land Use Restrictions and Structure Maintenance Agreements

Where contamination remains in place, it may be necessary for the project manager to work with private parties, state land management agencies, or local governments to implement use restrictions on nearshore areas and adjacent upland properties. For example, construction of boat ramps, retaining walls, or marina development can expose subsurface contamination and compromise the long-term effectiveness of a remedy. Where contaminated sediment exceeding cleanup levels is identified in proximity to utility crossings or other infrastructure and temporary or permanent relocation of utilities in support of a dredging remedy may not be feasible or practical, capping may be desirable even though temporary cap disruption may be necessary periodically.

Ownership of aquatic lands varies by state and locality. In many cases, nearshore areas can be privately owned out to the end of piers. For private property owners, more traditional ICs, such as proprietary controls or enforcement tools with IC components, can be considered. Potentially, some of these restrictions can be implemented through agencies who permit construction activities in the aquatic environment. Several federal, state, and local laws place restrictions on and may require permits or substantive requirements documents to be obtained for dredging, filling, or other construction activities in the aquatic environment. These include Section 404 of the Clean Water Act, Title 33 United States Code

(U.S.C.) Section 1344, and Sections 9 and 10 of the Rivers and Harbors Act of 1899, 33 U.S.C. 401 and 403. It may also be possible to implement some ICs through coordination with existing permitting processes. Harbor Master Plans, state-designated port areas, and local authorities may also function to restrict certain uses. In addition, long-term maintenance of structures such as dams or breakwaters may be a necessary component of some sediment remedies. Where this is the case, it is important that project managers clarify how this maintenance is part of the remedy and who is responsible for the remedy. Where maintenance decisions may change through time, contingencies may be needed for additional actions.

Highlight 3-4 summarizes some important points to remember about feasibility studies at sediment sites.

Highlight 3-4: Some Key Points to Remember about Feasibility Studies for Sediment

- Generally, project managers should implement and then evaluate the effectiveness of major source control actions before finalizing the evaluation of alternatives for sediment
- Generally, project managers should evaluate each of the three major approaches: MNR, in-situ capping, and removal through dredging or excavation, at every sediment site
- At sites with multiple water bodies or sections of water bodies with different characteristics or uses, alternatives that combine a variety of remedial approaches are frequently the most promising
- MNR, in-situ capping, and sediment removal may each be capable of achieving acceptable levels of long-term effectiveness and permanence; site-specific site characteristics should be reviewed to ensure that the selected alternative will be effective at a particular site
- Accurate cost estimates, including long-term O&M costs and, where appropriate, materials handling, transport, and disposal costs, are very important to a good comparison of alternatives; a Actual costs from pilot projects at a site and at similar, completed sediment sites are among the best cost resources
- Institutional controls can be used at all stages of the remedial process to reduce exposure to contamination; project managers should consider the effectiveness and enforce ability of controls used at the site and evaluate their role in risk reduction

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4.0 MONITORED NATURAL RECOVERY

4.1 INTRODUCTION

Monitored natural recovery (MNR) is a remedy for contaminated sediment that typically uses ongoing, naturally occurring processes to contain, destroy, or reduce the bioavailability or toxicity of contaminants in sediment. Not all natural processes result in risk reduction; some may increase or shift risk to other locations or receptors. Therefore, to implement MNR successfully as a remedial option, project managers should identify and evaluate those processes that contribute to risk reduction. MNR usually involves acquisition of information over time to confirm that these risk-reduction processes are occurring. Project managers should also be aware of the potential for combining natural recovery with engineering approaches, for example by installing flow control structures to encourage deposition or by the placement of a thin layer of additional clean sediment or additives to enhance sorption or chemical transformation. These combined approaches are discussed further in Section 4.5, Enhanced Natural Recovery.

MNR may rely on a wide range of naturally occurring processes to reduce risk to human and/or ecological receptors. These processes may include physical, biological, and chemical mechanisms that act together to reduce the risk posed by the contaminants. Depending on the contaminants and the environment, this risk reduction may occur in a number of different ways. Highlight 4-1 lists the most common risk reduction processes. Natural processes that reduce toxicity through transformation or reduce bioavailability through increased sorption are usually preferable as a basis for remedy selection to mechanisms that reduce exposure through natural burial or mixing-in-place because the destructive/sorptive mechanisms generally have a higher degree of permanence. However, many contaminants that remain in sediment are not easily transformed or destroyed. For this reason, risk reduction due to natural burial through sedimentation is more common and can be an acceptable sediment management option. Dispersion is the least preferable basis for remedy selection based on MNR. While dispersion may reduce risk in the source area, it generally increases exposure to contaminants and may result in unacceptable risks to downstream areas or other receiving water bodies. As reiterated in Chapter 7, Remedy Selection Considerations, project managers should carefully evaluate the effects of this increased exposure and risk to receiving water bodies before selecting MNR where dispersion is one of the risk reduction mechanisms, to ensure that it is not simply transferring risk to a new area. Project managers should be aware that at most sites, a variety of natural processes are occurring that may reduce risk.

As used in this guidance, MNR is similar in some ways to the Monitored Natural Attenuation (MNA) remedy used for ground water and soils [U.S. Environmental Protection Agency (U.S. EPA 1999d)]. The key difference between MNA for ground water and MNR for sediment is in the type of processes most often being relied upon to reduce risk. Transformation of contaminants is usually the major attenuating process for contaminated ground water, these processes are frequently too slow for the persistent contaminants of concern (COCs) in sediment to provide for remediation in a reasonable time frame. Therefore, isolation and mixing of contaminants through natural sedimentation is the process most frequently relied upon for contaminated sediment.

Highlight 4-1: General Hierarchy of Natural Recovery Processes for Sediment Sites

Many different natural processes may reduce risk from contaminated sediment, including the following, listed from generally most to least preferable, though all potentially acceptable, as a basis for selecting MNR:

A	The contaminant is converted to a less toxic form through transformation processes, such as biodegradation or abiotic transformations
B	Contaminant mobility and bioavailability are reduced through sorption or other processes binding contaminants to the sediment matrix
C	Exposure levels are reduced by a decrease in contaminant concentration levels in the near-surface sediment zone through burial or mixing-in-place with cleaner sediment
D	Exposure levels are reduced by a decrease in contaminant concentration levels in the near-surface sediment zone through dispersion of particle-bound contaminants or diffusive or advective transport of contaminants to the water column or (see caveats in text regarding use of these processes for risk reduction)

To select a MNR remedy, the project manager generally should consider the need for the following:

- A detailed understanding of the natural processes that are affecting sediment and contaminants at the site;
- A predictive tool (generally based either on computer modeling or extrapolation of empirical data) to predict future effects of those processes;
- A means to control any significant ongoing contaminant sources;
- An evaluation of ongoing risks during the recovery period and exposure control, where possible; and
- The ability to monitor the natural processes and/or concentrations of contaminants in sediment or biota to see if recovery is occurring at the expected rate.

Some consider that all sediment site remedies are using natural recovery to some extent because natural processes are ongoing whether or not an active cleanup is underway [e.g., National Research Council (NRC) 2001]. It is true that natural processes in most cases will continue whether or not an active cleanup is underway, but these processes may either reduce, transfer, or increase risk. Natural processes may reduce residual risk following dredging or in-situ capping at many sites, and it can be very valuable to monitor further risk reduction. However, it is also important for project managers to distinguish whether they are relying upon natural processes to reduce risk to an acceptable level (i.e., using MNR as a remedy), or simply noting the fact that natural processes are ongoing at a site and are expected to continue to reduce residual risks. Therefore, the key factors that normally distinguish MNR as a remedy are the presence of unacceptable risk, the ongoing burial or degradation/transformation, or dispersion of the contaminant, and the establishment of a cleanup level that MNR is expected to meet within a particular time frame.

MNR has been selected as a component of the remedy for contaminated sediment at approximately one dozen Superfund sites so far. Historically, at many sites MNR has been combined with dredging or in-situ capping of other areas of a site. Although natural recovery following effective source control has been observed (e.g., decreases in sediment contaminant levels, sediment toxicity, and shellfish tissue contaminant levels), long-term monitoring data on fish tissue are not yet available at most sites to document continued risk reduction (see e.g., Magar et al. 2003). However, monitoring results documented at some sites are promising (e.g., Patmont et al. 2003, U.S. EPA 2001g, U.S. EPA 2001h, Swindoll et al. 2000). When hazardous substances left in place are above levels that allow for unlimited use and unrestricted exposure, a five-year review pursuant to Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) □121(c) may be required (U.S. EPA 2001i).

Although each of the three potential remedy approaches (MNR, in-situ capping, and removal) should be considered at every site at which they might be appropriate, MNR should receive detailed consideration where the site conditions listed in Highlight 4-2 are present.

Highlight 4-2: Some Site Conditions Especially Conducive to Monitored Natural Recovery

- Anticipated land uses or new structures are not incompatible with natural recovery
- Natural recovery processes have a reasonable degree of certainty to continue at rates that will contain, destroy, or reduce the bioavailability or toxicity of contaminants within an acceptable time frame
- Expected human exposure is low and/or can be reasonably controlled by institutional controls
- Sediment bed is reasonably stable and likely to remain so
- Sediment is resistant to resuspension (e.g., cohesive or well-armored sediment)
- Contaminant concentrations in biota and in the biologically active zone of sediment are moving towards risk-based goals on their own
- Contaminants already readily biodegrade or transform to lower toxicity forms
- Contaminant concentrations are low and cover diffuse areas
- Contaminants have low ability to bioaccumulate

4.2 POTENTIAL ADVANTAGES AND LIMITATIONS

In most cases, the two key advantages of MNR are its relatively low implementation cost and its non-invasive nature. While costs associated with site characterization and modeling can be extensive, the costs associated with implementing MNR are primarily associated with monitoring. However, implementation costs may also include the cost of implementing institutional controls and public education to increase the effectiveness of those controls. MNR typically involves no man-made physical disruption to the existing biological community, which may be an important advantage for some wetlands or sensitive environments where the harm to the ecological community due to sediment disturbance may outweigh the risk reduction of an active cleanup.

Other advantages of MNR may include no construction or infrastructure is needed, and may, therefore, be much less disruptive of communities than active remedies such as dredging or in-situ capping. No property should be needed for materials handling, treatment, or disposal facilities, and no contaminated materials should be transported through communities.

Two key limitations of MNR may include it generally leaves contaminants in place and that it can be slow in reducing risks in comparison to active remedies. Any remedy that leaves untreated contaminants in place probably includes some risk of reexposure of the contaminants. When MNR is based primarily on natural burial, there is some risk of buried contaminants being reexposed or dispersed if the sediment bed is significantly disturbed by unexpectedly strong natural or man-made (anthropogenic) forces. The potential effects of reexposure may be greater if high concentrations of contaminants remain in the sediment, and likewise, lower if contaminant concentrations or risks are low. There is also some risk of dissolved contaminants being transported to the surface water at levels that could cause unacceptable risk. The time frame for natural recovery may be slower than that predicted for dredging or in-situ capping. However, time frames for various alternatives may overlap when uncertainties are taken into account. In addition, realistic estimates of the longer design and implementation time for active remedies should be factored in to the comparison. Like any remedy that takes a period of time to reach remediation goals, remedies that include MNR frequently rely upon institutional controls, such as fish consumption advisories, to control human exposure during the recovery period. These controls may have limited effectiveness and usually have no ability to reduce ecological exposures.

Major areas of uncertainty frequently noted for MNR include the ability to 1) predict future sedimentation rates in dynamic environments and 2) predict rates of contaminant flux through stable sediment. It can be especially difficult to predict rates of natural recovery where contaminant levels and risks are already low because small additional factors become relatively more important. However, a higher level of uncertainty may be more acceptable in these situations as well.

4.3 NATURAL RECOVERY PROCESSES

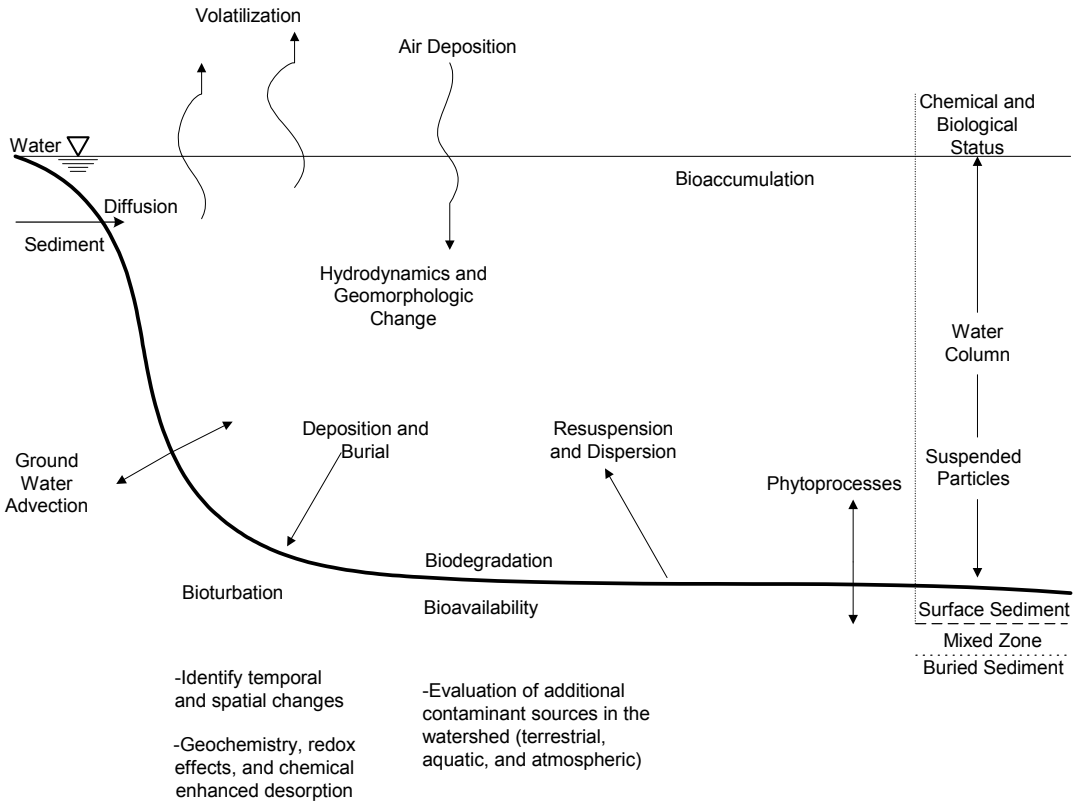
The success of MNR as a risk reduction approach typically is dependent upon understanding the dynamics of the contaminated environment and the fate and mobility of the contaminant in that environment. The natural processes of interest for MNR may include a variety of processes that, under favorable conditions, act without human intervention to reduce the mass, toxicity, mobility, or concentration of contaminants in the sediment bed. These natural processes may include the following:

- *Physical processes*: Sedimentation, advection, diffusion, dilution, dispersion, bioturbation, volatilization;
- *Biological processes*: Biodegradation, biotransformation, phytoremediation, biological stabilization; and
- *Chemical processes*: Oxidation/reduction, sorption, or other processes resulting in stabilization or reduced bioavailability.

Highlight 4-3 illustrates some of the natural processes the project manager should consider when evaluating MNR. With few exceptions, these processes interact in aquatic systems, sometimes increasing

the risk-reduction effects of a process compared to what they might be for that process in isolation, and sometimes reducing those risk-reduction effects. For example, as recognized by the U.S. Environmental Protection Agency s (EPA) Science Advisory Board (SAB) Environmental Engineering Committee, *Monitored Natural Attenuation: USEPA Research Program - An EPA Science Advisory Board Review* (U.S. EPA 2001j), sustained burial processes remove contaminants from the bioavailable zone, but can also impede certain degradation processes, such as aerobic biodegradation. Likewise, contaminant sorption to sediment particles may reduce both bioavailability and rates of contaminant transformation. In addition, in the case of mixed contaminants, the same natural process may result in very different environmental fates. When dealing with mixed contaminants at a site, the project manager should not focus unduly on one contaminant without understanding the effects of natural processes on the other contaminants, including breakdown products. Understanding the interactions between effects and prioritizing the significance of these effects to the MNR remedy should be part of a natural process analysis.

Highlight 4-3: Sample Conceptual Model of Natural Processes Potentially Related to MNR



4.3.1 Physical Processes

Generally, physical processes do not directly change the chemical nature of contaminants. Instead, physical processes may bury, mix, dilute, or transfer contaminants to another medium. Physical processes of interest for MNR include sedimentation, erosion, diffusion, dilution, dispersion, bioturbation, advection, and volatilization (including temperature-induced desorption of semi-volatiles). All of these processes may reduce contaminant concentrations in surface sediment, and thus reduce risk associated with the sediment. Sedimentation normally reduces risk physically by containing contaminants in place. Other physical processes, such as erosion, dispersion, dilution, bioturbation, advection, and volatilization may reduce contaminant concentrations in sediment as a result of transferring the contaminants to another medium or dispersing them over a wider area (e.g., via ground water or surface water). These processes may reduce, increase, or transfer the risk posed by the contaminants. As discussed previously in Section 4.1, project managers should carefully evaluate the potential for increased exposure and risk to receiving water bodies before selecting MNR where dispersion is one of the risk reduction mechanisms.

Physical processes in sediment can operate at vastly different rates. Some may occur faster than others, but may or may not have more impact on risk. In general, processes in which contaminants are transported by bulk movement of particles or pore water (e.g., erosion, dispersion, bioturbation, advection) occur at faster rates than processes in which contaminants are transported by diffusion or volatilization and, therefore, are frequently, but not always, more important when evaluating MNR. Processes that result in particle movement are particularly important for hydrophobic or other contaminants that are strongly sorbed to sediment particles. Some physical processes are continuous, and others seasonal or episodic. Depending on the environment, any of these types of processes (i.e., continuous, seasonal, or episodic) may have the most impact on natural recovery of a site. For example, project managers should not assume that episodic flooding will have a positive or negative effect on risk over an entire site. Flooding is most likely to cause erosion in some areas, while causing significant deposition in others.

Transport and deposition of cleaner sediment in a watershed may lead to natural burial of contaminated sediment in a quiescent environment. Natural burial may reduce the availability of the contaminants to aquatic plants and animals and, therefore, may reduce toxicity and bioaccumulation. The overlaying cleaner sediment also serves to reduce the flux of contaminants into the surface water by creating a longer pathway that the desorbed contaminants must travel to reach the water column. However, while bioturbation by burrowing organisms may promote mixing and dilution of contaminated sediment with the newly deposited cleaner sediment, for bioaccumulative contaminants it may also result in continued bioaccumulation into the food web until contaminant isolation occurs.

The long-term protectiveness provided by sedimentation depends upon the physical stability of the new sediment bed and the rates of movement of contaminants through the new sediment. Major events, such as severe floods or ice movements may scour the buried sediment, exposing contaminated sediment and releasing the contaminants into the water column. Ground water that flows through the sediment bed also may transport dissolved contaminants into the water column. Depending upon their extent, processes such as these may extend the natural recovery period or, in some cases, inhibit it altogether. Project managers should consider the potential influence of these processes on exposure rates and risk. A site-specific evaluation of both sediment and contaminant fate and transport are important to evaluating MNR as a remedy. There are a variety of empirical and modeling methods to assess rates of

various physical processes at specific sites. These are discussed in Chapter 2, Section 2.8, Sediment and Contaminant Fate and Transport, and Section 2.9, Modeling.

4.3.2 Biological and Chemical Processes

Like most natural processes, biological processes also depend on site-specific conditions and are highly variable. During biodegradation, a chemical change is facilitated by microorganisms living in the sediment. One of the important limitations to the usefulness of biodegradation as a risk-reduction mechanism is that the greater the molecular weight of the organic contaminants, the greater partitioning to sorption sites on sediment particles (Mallhot and Peters 1988) and the lower the contaminant availability to microorganisms. Some degradation of high molecular weight organic compounds occurs naturally in soil and sediment with anaerobic and aerobic microorganisms (Brown et al. 1987, Abramowicz and Olsen 1995, Bedard and May 1996, Shuttleworth and Cerniglia 1995, Cerniglia 1992, Seech et al. 1993). Degradation rates vary with depth in sediment partly due to the change from aerobic or anaerobic conditions. This changes frequently occur at depths of a few millimeters to a few centimeters where sediments have substantial organic content and conditions are quiescent, and may occur deeper in some circumstances. Longer residence times of contaminants in the sediment (aging) also usually result in increased sequestration (Luthy et al. 1997, Dec and Bollag 1997). These processes reduce the availability of the organic compounds to microorganisms and, therefore, reduce the extent and rates of biodegradation (Luthy et al. 1997, Tabak and Govind 1997). However, this can also reduce the availability of the contaminant to receptors living in the sediment and as well as at higher trophic levels.

Chemical processes in sediment are especially important for metals. Many environmental variables govern the chemical state of metals in sediment, which in turn affects their mobility, toxicity, and bioavailablity making natural recovery due to chemical processes difficult to predict. Much of the current understanding of the role of chemical processes in controlling risk is focused on the important geochemical changes resulting from changes in redox potential that can affect the bioavailability of metal and organic metal compounds. Formation of relatively insoluble metal sulfides under reducing conditions can often effectively control the risk posed by metal contaminants if reducing conditions are maintained. Environmental variables include pore water pH and alkalinity, sediment grain size, oxidation-reduction (redox) conditions, and the amount of sulfides and organic carbon present in the sediments. Furthermore, many chemical processes in sedimentary environments are also affected by the biological community.

Biochemical Processes for Polycyclic Aromatic Hydrocarbons (PAHs)

The class of hydrocarbons known as polycyclic aromatic hydrocarbons (PAHs) is a common contaminant in sediment and biota at Superfund sites. Many organisms are capable of accumulating PAH contaminants in their tissue, but biomagnification does not generally occur in vertebrate species (Suedel et al. 1994). Fish do not generally accumulate higher tissue PAH concentrations than their prey due to their ability to metabolize and eliminate PAHs; however, the PAH metabolites may themselves cause chronic toxicity, such as reduced growth and reproduction as well as increased incidence of neoplasms in fish. The potential exists for bioaccumulation in some invertebrate species because of their lesser ability to metabolize and eliminate PAHs (Meador et al. 1995).

PAHs may be subject to physical, chemical and biological breakdown in the environment and where these processes are effective, may be especially amenable to natural recovery. The type of process that dominates may depend on time. For example, following a release of PAHs into the environment,

physical-chemical processes such as dispersion, volatilization, and photodegredation may dominate. Where these processes are effective in attenuating the contaminants to less toxic levels, tolerant microbial species may cause further biodegradation. There is a wide variation in rates of biodegradation and toxicity reduction, depending on the levels of microbial activity and the physical and chemical conditions of the site (Swindoll et al. 2000). PAHs biodegrade more quickly through aerobic than anaerobic processes, although the degradation rate usually decreases as the number of aromatic rings increases (Shuttleworth and Cerniglia 1995, Cerniglia 1992, Seech et al. 1993). While biodegradation of PAHs may occur under anaerobic conditions, PAHs usually persist longer in anaerobic sediment compared to aerobic environments (U.S. EPA 1996d, Safe 1980).

Although low PAH degradation rates are often attributed to low bioavailability (see review by Reid et al. 2000), evidence reported by Schwartz and Scow (2001) demonstrates that it may be the lack of enzyme induction amongst the PAH-degrading bacteria that is responsible for low rates below a threshold PAH concentration. Other researchers have reported this phenomenon for PAHs (Ghiorse et al. 1995, Langworthy et al. 1998) and other aromatic organics (Zaidi et al. 1988, Roch and Alexander 1997). At elevated PAH concentrations in sediment, there is selective pressure for PAH-degrading bacteria, which can increase the capacity to attenuate PAHs naturally. However, there is uncertainty about whether and how fast this degradation may reach acceptable risk levels. Because of the variation among sites, site-specific studies may be needed to resolve uncertainties concerning degredation rates and whether these rates will contribute to recovery within an acceptable time frame.

Biochemical Processes for Polychlorinated Biphenyls (PCBs)

Release of a PCB Aroclor (see PCB data information in Chapter 2, Section 2.1.2, Types of Data) into the environment may result in a change in its congener composition. This is a result of the combined weathering effects and such processes as differential volatilization, solubility, sorption, anaerobic dechlorination, and metabolism, and results in changes in the composition of the PCB mixture in sediment, water, and biota over time and between trophic levels (NRC 2001).

Highly chlorinated congeners of PCBs may gradually partially dechlorinate naturally in anaerobic sediment (Brown et al. 1987, Abramowicz and Olsen 1995, Bedard and May 1996). In general, less-chlorinated PCBs bioaccumulate less than the highly chlorinated congeners, but are more soluble and, therefore, more readily transported into and within the water column than highly chlorinated PCBs. The less chlorinated PCBs exhibit significantly less potential human carcinogenic and dioxin-like (coplanar structure) toxicity (Abramowicz and Olsen 1995, Safe 1992), but may be transformed in humans into forms with potential for other toxicity (Bolger 1993).

Aerobic processes may then biodegrade the less chlorinated PCB congeners (Flanagan and May 1993, Harkness et al. 1993). The sediment concentrations of other chemicals and the total organic content tend to control these processes. However, little evidence exists that lower chlorinated congeners under the anaerobic or anoxic conditions found in most sediment are significantly transformed. Therefore, these partially dechlorinated organics tend to accumulate and persist (U.S. EPA 1996d, Harkness et al. 1993). Although desirable, it is unclear whether biologically mediated dechlorination of PCBs would be effective in achieving remedial objectives in a reasonable time frame and may result in the production of more toxic byproducts.

4.4 EVALUATION OF NATURAL RECOVERY

An evaluation of MNR as a potential remedy or remedy component should generally focus on considering, at a minimum, the following questions:

- Is there evidence that the system is recovering?
- Why is the system recovering or not recovering?
- What is the pattern of recovery or non-recovery expected in the future?

This evaluation should be supported with a variety of types of site-specific characterization data and, often, modeling. The lines of evidence approach for evaluation of natural attenuation of contaminants in soil and ground water can provide a general framework for evaluating MNR in sediment (e.g., U.S. EPA 1999d). Swindoll and his colleagues include a chapter on natural remediation of sediment that presents a useful summary discussion (Swindoll et al. 2000). EPA s Office of Research and Development (ORD) is in the process of drafting a technical resource document specifically for MNR in sediments and may also include suggested protocols. In addition, members of the joint industry–EPA Sediments Action Team of the Remedial Technologies Development Forum (RTDF) has developed a series of working papers on MNR that can be found at http://www.rtdf.org/public/sediment/mnrpapers.htm (Davis et al. 2003, Dekker et al. 2003, Erickson et al. 2003, Magar et al. 2003, Patmont et al. 2003).

As with the evaluation of any sediment alternative, an evaluation of MNR should be generally based on a thorough conceptual site model that includes current and future pathways of human and ecological exposure to the contaminants. This conceptual understanding should be based on site-specific data collected over a number of years and, for factors known to fluctuate seasonally, data collected during different seasons. Lines of evidence that can be used to construct a plausible case for the use of MNR include those listed in Highlight 4-4. It is important to note that not all lines of evidence or types of information are appropriate at every site, but, generally, multiple lines of evidence are needed. Project managers should be aware that a substantial spacial and temporal record may be useful to establish a reliable trend, especially for surface sediment data, which typically vary widely.

Highlight 4-4: Potential Lines of Evidence of Monitored Natural Recovery

- Long-term decreasing trend of contaminant levels in higher trophic level biota (e.g., piscivorous fish)
- Long-term decreasing trend of water column contaminant concentrations averaged over a typical low-flow period of high biological activity (e.g., trend of summer low flow concentrations)
- Sediment core data demonstrating a decreasing trend in historical surface contaminant concentrations through time
- Long-term decreasing trends of surface sediment contaminant concentration, sediment toxicity, or contaminant mass within the sediment

Examples of types of site-specific information that could be collected to support the lines of evidence listed in Highlight 4-4 include the following:

- Identification and characterization of ongoing sources of contamination;
- Characterization of sediment types (e.g., bed mapping) and stratigraphic structure of the sediment bed;
- Evaluation of historical and current contaminant levels in biota and surface water;
- Evaluation of geomorphology, long-term accretion, and erosion;
- Evaluation of sequestration mechanisms (e.g., sorption, precipitation) and rates of degradation or transformation;
- Determination of the depth of the surface mixed layer;
- Measurement of suspended solids and contaminant transport during high-energy (e.g., storm) events;
- Measurement of sediment erosion properties and impacts of ice on sediment transport;
- Evaluation of impacts of ground water advection or movement of non-aqueous phase liquids (NAPL); and
- Development of a tool to allow prediction of future recovery and risk reduction (e.g., sediment and contaminant fate and transport modeling).

The amount of physical, biological, and chemical process information needed to assess the applicability of MNR adequately is site specific. An important step in documenting the potential for MNR as a management alternative normally is to show observed reductions in exposure and risk can be reasonably expected to continue into the future. In systems where the mechanisms causing the recovery are uncertain, or where the fate and transport processes driving recovery may be complex and changing with time, simple extrapolation of historical trends may not be appropriate. In such cases, a well-constructed model can be a useful tool for predicting future behavior of the system. The use of models is discussed further in Chapter 2, Section 2.9 Modeling.

Integration of the data quality objective (DQO) process with risk evaluation can help identify which natural processes are most critical to the evaluation of MNR at a site. Generally, the identification of MNR data needs and preparation of study design can be structured similarly to the DQO process (U.S. EPA 2000a) that is normally integrated within the remedial investigation and feasibility study (RI/FS). The DQO process is discussed in greater detail in Chapter 2, Section 2.1.1.

4.5 ENHANCED NATURAL RECOVERY

In some areas, natural recovery may appear to be the most appropriate remedy, yet the rate of sedimentation or other natural processes is insufficient to reduce risks within an acceptable time frame. Where this is the case, project managers may consider accelerating the recovery process by engineering means, for example by the addition of a thin layer of clean sediment. This approach is sometimes referred to as thin-layer placement or particle broadcasting. Thin-layer placement normally accelerates natural recovery by adding a layer of clean sediment over contaminated sediment. The acceleration can occur through several processes, including increased dilution through bioturbation of clean sediment mixed with underlying contaminants. Thin-layer placement is typically different than the isolation caps discussed in Chapter 5, In-situ Capping, because it is not designed to provide long-term isolation of contaminants from benthic organisms. While thickness of an isolation cap can range up to several feet, the thickness of the material used in thin layer placement could be as little as a few inches. The grain size and organic carbon content of the clean sediment to be used for thin-layer placement should be carefully considered in consultation with aquatic biologists. In most cases, natural materials (as opposed to manufactured materials) approximating common substrates found in the area should be used. Clean sediment can be placed in a uniform thin layer over the contaminated area or it can be placed in berms or windrows, allowing natural sediment transport processes to distribute the clean sediment to the desired areas.

Project managers might also consider the addition of flow control structures to enhance deposition in certain areas of a site. Enhancement or inception of contaminant degradation through additives might also be considered to speed up natural recovery. However, when evaluating the feasibility of these approaches, project managers should consult state and federal water programs regarding the introduction of clean sediment or additives to the water body. For example, in some areas, potentially erodible clean sediment already is a major nonpoint source pollution problem, especially in areas near sensitive environments such as those with significant subaquatic vegetation or shellfish beds.

4.6 ADDITIONAL CONSIDERATIONS

MNR is likely to be effective most quickly in depositional environments after source control actions and active remediation of any high risk sediment have been completed. Where external sources were controlled many years previously and no discernable high risk sediment areas can be identified, yet site risks remain unacceptable, it may be questionable whether natural processes alone will reduce risks satisfactorily in the future. At these sites, it can be especially important to evaluate the effectiveness of previous source control actions and to evaluate potential additional active sediment source control or remediation methods for selected areas. For MNR, as for other sediment remedies, effective source control is often critical to reaching remedial objectives in a reasonable time frame and to preventing re-contamination.

As discussed in Chapter 7, Remedy Selection Considerations, when evaluating MNR, the short-term effects on human health and the environment during the recovery period (i.e., the baseline risks for the site) should be compared to the short-term effects of other approaches such as effects of resuspension of contaminants due to dredging and habitat changes caused by capping. Section 7.3, Considering Remedies, discusses the process of comparing short-term and long-term risks associated with various approaches in a net comparative risk analysis.

In most cases, the long-term effectiveness of MNR is dependent on the dynamic processes of mixing and burial over time remaining dominant over sediment resuspension or contaminant movement via advective flow or other mechanisms. Assessment of sediment and contaminant fate and transport are, therefore, very important at most sites. Some potential mechanisms for physical disruption of overlying cleaner sediment, such as keel drag or pipeline construction, may be amenable to human management controls. Others mechanisms for physical disruption, such as ice scour or flooding, may be only partly manageable or not manageable. The importance of contaminant movement through overlying sediment to surficial sediment and the overlying water can depend on several factors, including the chemical characteristics of the contaminant, physical characteristics of the sediment, and patterns of ground water flow. These issues can also be of concern for in-situ capping and are discussed further in Chapter 2, Section 2.8, Sediment and Contaminant Fate and Transport, in Chapter 5, In-Situ Capping, and in the U.S. Army Corps of Engineers (USACE) Technical Note, *Subaqueous Capping and Natural Recovery: Understanding the Hydrogeologic Setting at Contaminated Sediment Sites* (Winter 2002). In general, the presence of processes, such as erosion or ground water flow, that cause release of contamination to the water column should not eliminate consideration of MNR as a remedy; instead, they should lead to evaluation of the consequences of those processes on exposure and risk.

Generally, regions should consider using MNR either in conjunction with source control or active sediment remediation or as a follow-up measure to an active remedy. For example, MNR may be an appropriate approach for some sediment sites after control of floodplain soils and NAPL seeps. At other sites, MNR may be an appropriate approach to control risk from areas of wide-spread, low-level sediment contamination, following dredging or capping of more highly-contaminated areas. MNR may also be an appropriate measure to reduce residual risk from dredging or excavation in cases where the active cleanup is not expected to achieve risk-based measures alone.

When considering the use of MNR as a follow-up measure, project managers should consider the change in conditions caused by the active remedy. As noted by the SAB (U.S. EPA 2001j): If MNA [or, as used in this guidance, MNR] is to be considered after a remedial action (e.g., the removal of heavily contaminated portions or capping), the effects of the remedial action on the chemistry, biology, and physics of contaminated sediments should be evaluated. The effects include: 1) potential disturbances on reaction conditions and aquatic life when dredging is used, and 2) changes on reaction conditions and mass transfer in the sediment and at the sediment/water interface when capping is used.

MNR should be considered when it would meet remedial objectives within a time frame that is reasonable compared to active remedies. However, the Agency recognizes that MNR may take longer to reach cleanup levels in sediment than dredging or in-situ capping and, therefore, may take longer to reach all remedial action objectives, such as contaminant reductions in fish. It is important to compare time frames on as accurate a basis as possible, including for example, accurate assessments of time for design and implementation of dredging or capping and realistic assumptions concerning dredging residuals. Where possible, estimates of the uncertainty in the recovery time frame associated with each alternative should also be made. Factors that the project manager should consider in determining whether the time frame for MNR is reasonable include the following:

- The extent and likelihood of human exposure to contaminants during the recovery period, and if controlled by institutional controls, the effectiveness of those controls;

- The value of ecological resources that may continue to be impacted during the recovery period;
- The time frame in which affected portions of the site may be needed for future uses which will be available after MNR has achieved cleanup levels; and
- The uncertainty associated with the time frame prediction.

As with any remedy, project managers should carefully evaluate the uncertainties involved and consider the need for contingency measures, contingency remedies, or interim decisions where there is significant uncertainty about effectiveness. For MNR, as for other approaches which take a period of time to reduce risk, project managers should carefully consider how risks can be controlled during the recovery period. For sites with bioaccumulative contaminants, institutional controls such as fish consumption advisories are frequently needed to reduce human exposures during this period. In most cases, no institutional controls are possible for reducing ecological exposure during the recovery period. See Chapter 3, Section 3.6, Institutional Controls, and Chapter 7, Section 7.5, Considering Institutional Controls, for more information concerning institutional controls at sediment sites. Highlight 4-5 lists some important points to remember from this chapter.

Highlight 4-5: Some Key Points to Remember When Considering Monitored Natural Recovery

- Source control should be generally implemented to prevent recontamination
- MNR frequently includes multiple physical, biological, and chemical mechanisms that act together to reduce risk
- Evaluation of MNR should be usually based on site-specific data collected over a number of years. At some sites, this may include an assessment of seasonal variation for some factors
- Project managers should evaluate the long-term stability of the sediment bed, the mobility of contaminants within it, and the likely ecological and human health impacts of disruption
- Multiple lines of evidence are frequently needed to evaluate MNR (e.g., time-series data, core data, modeling)
- Thin-layer placement of clean sediment may accelerate natural recovery in some cases
- Contingency measures should be included as part of an MNR remedy when there is significant uncertainty that the remedial action objectives will be achieved within the predicted time frame
- Generally, MNR should be used either in conjunction with source control or active sediment remediation

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5.0 IN-SITU CAPPING

5.1 INTRODUCTION

For purposes of this guidance, in-situ capping refers to the placement of a subaqueous covering or cap of clean material over contaminated sediment that remains in place. Caps are generally constructed of granular material, such as clean sediment, sand, or gravel. A more complex cap design can include geotextiles, liners, and other permeable or impermeable elements in multiple layers that may include additions of material to attenuate the flux of contaminants (e.g., organic carbon). Depending on the contaminants and sediment environment, a cap is designed to reduce risk through the following primary functions:

- Physical isolation of the contaminated sediment sufficient to reduce exposure due to direct contact and to reduce the ability of burrowing organisms to move contaminants to the surface;
- Stabilization of contaminated sediment and erosion protection of sediment and cap, sufficient to reduce resuspension and transport to other sites; and/or
- Chemical isolation of contaminated sediment sufficient to reduce exposure from dissolved and colloidally bound contaminants transported into the water column.

Caps may be designed with different layers to serve these primary functions or in some cases a single layer may serve multiple functions.

As of 2004, In-situ capping has been selected as a component of the remedy for contaminated sediment at approximately fifteen Superfund sites. At some sites, in-situ capping has served as the primary approach for sediment, and at other sites it has been combined with sediment removal (i.e., dredging or excavation) and/or monitored natural recovery (MNR) of other sediment areas. In-situ capping has been successfully used at a number of sites in the Pacific Northwest, several of which were constructed over a decade ago (see site list at http://www.epa.gov/superfund/resources/sediment/sites.htm). When hazardous substances left in place are above levels allowing for unlimited use and unrestricted exposure, a five-year review pursuant to the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) 121(c) may be required [U.S. Environmental Protection Agency (U.S. EPA 2001i)].

Variations of in-situ capping include installation of a cap after partial removal of contaminated sediment and innovative caps, which incorporate treatment components. Capping is sometimes considered following partial sediment removal where capping alone is not feasible due to a need to preserve a minimum water body depth for navigation or flood control, or where it is desirable to leave deeper contaminated sediment in place to preserve bank or shoreline stability following removal. There are pilot studies underway to investigate the effectiveness of in-situ caps that incorporate various forms of treatment (see Chapter 3, Section 3.1.3, In-Situ Treatment and Other Innovative Alternatives). Application of thin layers of clean material may be used to enhance natural recovery through burial and mixing with clean sediment when natural sedimentation rates are not sufficient (see Chapter 4, Section 4.5, Enhanced Natural Recovery). Placement of a thin layer of clean material is also sometimes used to

backfill dredged areas, where it mixes with dredging residuals and further reduces risk from contamination that remains after dredging. In this application, the material is not often designed to act as an engineered cap to isolate buried contaminants and is, therefore, not considered in-situ capping in this guidance.

Much has been written about subaqueous capping of contaminated sediment. The majority of this work has been performed by, or in cooperation with, the U.S. Army Corps of Engineers (USACE). Comprehensive technical guidance on in-situ capping of contaminated sediment can be found in the EPA s *Assessment and Remediation of Contaminated Sediment (ARCS) Program Guidance for In-Situ Subaqueous Capping of Contaminated Sediments* (U.S. EPA 1998d) and the *Assessment and Remediation of Contaminated Sediments (ARCS) Program Remediation Guidance Document* (U.S. EPA 1994d), available through EPA s Web site at http://www.epa.gov/glnpo/sediment/iscmain. Additional technical guidance is available from the USACE s *Guidance for Subaqueous Dredged Material Capping* (Palermo et al. 1998a)

Although each of the three potential remedy approaches (MNR, in-situ capping, and removal) should be considered at every site at which they might be appropriate, capping should receive detailed consideration where the site conditions listed in Highlight 5-1 are present.

Highlight 5-1: Some Site Conditions Especially Conducive to In-Situ Capping

- Suitable types and quantities of cap material are readily available
- Anticipated infrastructure needs (e.g., piers, pilings, buried cables) are compatible with cap
- Water depth is adequate to accommodate cap with anticipated uses (e.g., navigation, flood control)
- Incidence of cap-disrupting human behavior, such as large boat anchoring, is low or controllable
- Long-term risk reduction outweighs habitat disruption, and/or habitat improvements are provided by the cap
- Hydrodynamic conditions (e.g., floods, ice scour) are not likely to compromise cap or can be accommodated in design
- Rates of ground water flow in cap area are low and not likely to create unacceptable contaminant releases
- Sediment has sufficient strength to support cap (e.g., higher density/lower water content, depending on placement method)
- Contaminants have low rates of flux through cap
- Contamination covers contiguous areas (e.g., to simplify capping)

5.2 POTENTIAL ADVANTAGES AND LIMITATIONS

Two advantages of in-situ capping are that it can quickly reduce exposure to contaminants and that, unlike dredging or excavation, it requires less infrastructure in terms of material handling,

dewatering, treatment, and disposal. A well-designed and well-placed cap should more quickly reduce the exposure of fish and other biota to contaminated sediment as compared to dredging, as there should be no or very little contaminant residual on the surface of the cap. Also, the cap often provides a clean substrate for recolonization by bottom-dwelling organisms. Changes in bottom elevation caused by a cap may create more desirable habitat, or specific cap design elements may enhance or improve habitat substrate. Another possible advantage is that the potential for contaminant resuspension and the risks associated with dispersion and volatilization of contaminated materials during construction are typically lower for in-situ capping than for dredging operations and risks associated with transport and disposal of contaminated sediment are avoided. Most capping projects use conventional equipment and locally available materials, and may be implemented more quickly and may be less expensive than remedies involving removal and disposal or treatment of sediment.

In-situ capping may be less disruptive of local communities than dredging or excavation. Although some local land-based facilities are often needed for materials handling, usually no dewatering, treatment, or disposal facilities need to be located and no contaminated materials are transported through communities. Where clean dredged material is used for capping, a much smaller area of land-based facilities is needed.

The major limitation of in-situ capping is the contaminated sediment remains in the aquatic environment where contaminants could become exposed or be dispersed if the cap is significantly disturbed or if contaminants move through the cap in significant amounts. In addition, in some environments, it can be difficult to place a cap without significant contaminant losses from compaction and disruption of the underlying sediment. If the water body is shallow, it may be necessary to develop institutional controls (ICs), which can be limited in terms of effectiveness and reliability, to protect the cap from disturbances such as boat anchoring and keel drag.

Another potential limitation of in-situ capping may be in some situations, a preferred habitat may not be provided by the surficial cap materials. To provide erosion protection, it may be necessary to use coarse cap materials that are different from native soft bottom materials, which may alter the biological community. In some cases, it may be desirable to select capping materials that discourage colonization by native deep-burrowing organisms to limit bioturbation and release of underlying contaminants.

5.3 EVALUATING SITE CONDITIONS

A good understanding of site-specific conditions typically is critical to predicting the expected feasibility and effectiveness of in-situ capping. Site conditions can affect all aspects of a capping project, including design, equipment and cap material selection, and monitoring and management programs. Some limitations in site conditions can be accommodated in the cap design. General aspects of site characterization are discussed in Chapter 2, Remedial Investigation Considerations. Some specific aspects of site characterization important for in-situ capping are introduced briefly in the following sections.

5.3.1 Physical Environment

Aspects of the physical environment that should be considered include water body dimensions, depth and slope (bathymetry) of sediment bed, and flow patterns, including tides, currents, and other

potential disturbances in cold climates, such as an ice scour. Existing infrastructure such as bridges, utility crossings, and other marine structures are discussed in Section 5.3.3.

The bathymetry of the site influences how far cap material will spread during placement and the cap s stability. Flat bottoms and shallow slopes should allow material to be placed more accurately, especially if capping material is to be placed hydraulically. Water depth also can influence the amount of spread during cap placement. Generally, the longer the descent of the cap material through the water column, the more water is entrained in the plume, resulting in a thinner layer of cap material over a larger area.

The energy of flowing water is also an important consideration. Capping projects are easier to design in low energy environments (e.g., protected harbors, slow-flowing rivers, or micro-tidal estuarine systems). In open water, deeper sites are generally less influenced by wind or wave generated currents and less prone to erosion than shallow, near-shore environments. However, armoring techniques or selection of erosion-resistant capping materials can make capping technically feasible in some high energy environments. Currents within the water column can affect dispersion during cap placement and can influence the selection of the equipment to be used for cap placement. Bottom currents can generate shear stresses that can act on the cap surface and may potentially erode the cap. In addition to ambient currents due to normal riverine or tidal flows, the project manager should consider the effects of storm-induced waves and other episodic events (e.g., floods, ice scour).

The placement of an in-situ cap can alter existing hydrodynamic conditions. In harbor areas or estuaries, the decrease in depth or change in bottom geometry can affect the near-bed current patterns, and thus the flow-induced bed shear stresses. In a riverine environment, the placement of a cap generally reduces depth and restricts flow and may alter the sediment and flood-carrying capacity of the channel. Modeling studies may be useful to assess these changes in site conditions where they are likely to be significant. Project managers are encouraged to draft decision documents that include some flexibility in requirements for how a cap affects carrying capacity of a water body, while still meeting applicable or relevant and appropriate requirements (ARARs). For example, in some water bodies, a cap may be appropriate even though it decreases, but not significantly, the flood-carrying capacity. In depositional areas, the effect of new sediment likely to be deposited on the cap should be considered in predicting future flood-carrying capacity. Clean sediment accumulating on the cap can increase the isolation effectiveness of the cap over the long term and may also increase consolidation of the underlying sediment bed.

5.3.2 Sediment Characteristics

The project manager should determine the physical, chemical, and biological characteristics of the contaminated sediment pursuant to using the data quality objective (DQO) process during the remedial investigation. The results of the characterization, in combination with the remediation goals and remedial action objectives (RAOs), should determine the areal extent or boundaries of the area to be capped.

Shear strength, especially undrained shear strength, of contaminated sediment deposits is of particular importance in determining the feasibility of in-situ capping. Most contaminated sediment is fine-grained, and is usually high in water content and relatively low in shear strength. Although a cap can be constructed on sediment with low shear strengths, the ability of the sediment to support a cap and the

need to construct the cap using appropriate methods to avoid displacement of the contaminated sediment should be carefully considered. The presence of other materials within the sediment bed, such as debris, wood chips, high sludge fractions, or other non-mineral-based sediment fractions, can also present special problems when interpreting grain size and other geotechnical properties of the sediment, but their presence can also improve sediment stability under a cap. It could be necessary to remove large debris prior to placing a cap, for example, if it will extend beyond the cap surface and cause scouring. Side-scan sonar can be an effective tool to identify debris.

The chemical characteristics of the contaminated sediment are an important factor that may affect design or selection of a cap, especially if capping highly mobile or highly toxic sediment. Capping may change the uppermost layer of contaminated sediment from an oxidizing to an anoxic condition, which may change the solubility of metal contaminants and the susceptibility of organic contaminants to microbial decomposition in this upper zone. For example, many of the divalent metal cations (e.g., lead, nickel, zinc) become less soluble in anaerobic conditions, while other metal ions (e.g., arsenic) become more soluble. Mercury, in the presence of pore water sulfate concentrations and organic matter, can become methylated through the action of anaerobic bacteria, and highly chlorinated, polychlorinated biphenyls (PCBs) may degrade to less chlorinated forms in an anaerobic environment. These issues are also discussed in Chapter 4, Section 4.3.2, Biological and Chemical Processes.

When contaminated sediment is capped, chemical conditions in the contaminated zone change. Mercury methylation is generally reduced as organic matter deposition and biological processes are reduced. Organic matter remaining beneath a cap may be decomposed by anaerobic microorganisms and release methane and hydrogen sulfide gases. As these dissolved gases accumulate, they could percolate through the cap by convective or diffusive transport. This process has the potential to solubilize some contaminants and carry them upward, dissolved in the gaseous bubbles. The grain size of the capping material controls in part how these avenues are developed. Finer grained caps may develop fissures whereas coarser grained caps such as sands allow gas to pass through. However, a compensating factor in some cases is caused by the caps insulation ability, which can cause underlying sediment to stay cooler and thus reduce expected decomposition rates. Where gas generation is expected to be significant, these factors should be considered during cap design.

5.3.3 Waterway Uses and Infrastructure

If the site under consideration is adjacent to or within a water body used for navigation, recreation or flood control, the effect of cap placement on those uses should be evaluated. As described in Section 5.3.1, the flood-carrying capacity of a water body could be reduced by a cap. If water depths are reduced in a harbor or river channel, some commercial and recreational vessels may have to be restricted or banned. The acceptable draft of vessels allowed to navigate over a capped area depends on water level fluctuations (e.g., seasonal, tidal, and wave) and the potential effects of vessel groundings on the cap. Potential cap erosion caused by propeller wash should be evaluated. Where circumstances dictate, an analysis should be conducted for activities that may affect cap integrity such as the potential for routine anchoring of large vessels. Anchoring by recreational vessels may or may not compromise the integrity of a cap, depending on its design. Such activities may indicate the need for restrictions (see Chapter 3, Section 3.6, Institutional Controls) or a modification of the cap design to accommodate certain activities. It may be necessary to restrict fishing and swimming to prevent recreational boaters from dragging anchors across a cap. In some situations, partial dredging prior to cap placement may minimize these limitations of capping.

Other activities in and around the water body may also impact cap integrity and maintenance needs and should be evaluated. These include the following:

- Water supply intakes;
- Storm water or effluent discharge outfalls;
- Utilities crossings;
- Construction of bulkheads, piers, docks, and other waterfront structures;
- Navigational dredging adjacent to the cap area; and
- Future development of commercial navigation channels in the vicinity of the cap.

Utilities (e.g., storm drains) and utility crossings (e.g., water, sewer, gas, oil, telephone, cable, and electric lines) are commonly located in urban waterways. It may be necessary to relocate existing utility crossings under portions of water bodies if their deterioration or failure might impact cap integrity. More commonly however, pipes or utilities are left in place under caps, and long-term operation and maintenance (O&M) plans include repair of cap damage caused by the need to remove, replace, or repair the pipes or utilities. Future construction or maintenance of utility crossings would have to consider the cap, and it may be necessary to consider limiting those activities through institutional controls (ICs) if cap repair cannot be assured. The presence of the cap can also place constraints on future waterfront development if dredging would be needed as part of the development activity.

In designing caps to be placed within federal navigation channels, horizontal and vertical separation distances may be developed by USACE based on considerations of normal dredging accuracy and depth allowances. This can provide a factor of safety to protect the cap surface from damage during potential future maintenance dredging.

To date, environmental agencies have little experience with the ability to enforce use restrictions necessary to protect the integrity of an in-situ cap (e.g., vessel size limits, bans on anchoring, etc.), although experience is growing. Generally, a state or local enforcement mechanism is necessary to implement specific use restrictions. Project managers should consider mechanisms for compliance assurance, enforcement, and the consequences of non-compliance, on use restrictions when evaluating in-situ capping.

5.3.4 Habitat Alterations

In-situ capping alters the aquatic environment and, therefore, can affect aquatic organisms in a variety of ways. As is discussed further in Chapter 6, Dredging and Excavation, while a project may be designed to minimize habitat loss or degradation, or even to enhance habitat, both sediment capping and sediment removal do alter the environment. Where baseline risks are relatively low, it is important to determine whether the potential loss of a contaminated habitat is a greater impact than the benefit of providing a new, modified but less contaminated habitat. Habitat considerations are especially important when evaluating materials for the uppermost layers of a cap. Sandy sediment and stone armor layers are often used to cap areas with existing fine-grained sediment. Through time, sedimentation and other

natural processes will change the uppermost layer of the cap. At least initially, changes in organic carbon content of the capping material may change the feeding behavior of bottom-dwelling organisms in the capped area. Generally, the uppermost cap layers become a substrate for recolonization. Where possible, caps should be designed to provide habitat for desirable organisms. In some cases it is possible to provide a habitat layer over an erosion protection layer by filling the interstices of armor stones with materials such as crushed gravel. In some cases, natural sedimentation processes after cap placement can create desirable habitat characteristics. For example, placement of a rock cap in some riverine systems can result in a final cap surface that is similar to the previously existing surface because the rock may become embedded with sands/silts through natural sedimentation.

Desirable habitat characteristics for cap surfaces vary by location. Providing a layer of appropriately sized rubble that can serve as hard substrate for attached molluscs (e.g., oysters, mussels) can greatly enhance the ecological value at some sites. Material suitable for colonization by foraging organisms, such as bottom-dwelling fish, can also be appropriate. A mix of cobbles and boulders may be desirable for aquatic environments in areas with substantial flow. In addition, the potential for attracting burrowing organisms incompatible with the cap design or ability to withstand additional physical disturbances should be considered. Habitat enhancements should not impair the function of the cap or its ability to withstand the shear stresses of storms, floods, propeller wash, or other disturbances. Project managers should consult with local resource managers and natural resource trustee agencies to determine what types of modifications to the cap surface would provide suitable substrate for local organisms.

Habitat considerations are also important when evaluating post-capping bottom elevations. Capping often increases bottom elevations, which in itself can alter the pre-existing habitat. For example, a remediated subtidal habitat can become intertidal, or lake habitat can become a wetland (Cowardin et al. 1979). Changes in bottom elevation may either enhance or degrade desirable habitat, depending on the site.

Project managers should consult EPA staff familiar with implementing the Clean Water Act, as well as natural resource trustees and USACE, where Section 404 of the Clean Water Act is either applicable or relevant and appropriate [see Chapter 3, Section 3.3, Applicable or Relevant and Appropriate Requirements (ARARs) for Sediment Alternatives]. Where remedies under consideration degrade aquatic habitat, substantive requirements may include minimizing the permanent loss of habitat and mitigating it by creation or restoration of a similar habitat elsewhere. However, it should not be assumed that in-situ caps result in a permanent loss of habitat; this is a site-specific decision. In addition, project managers should be aware that any mitigation related to meeting the substantive requirements of ARARs for the site, such as the Clean Water Act, may be independent of the Natural Resource Trustees natural resource damage assessment process.

5.4 FUNCTIONAL COMPONENTS OF A CAP

As introduced in Section 5.1 of this chapter, caps are generally designed to fulfill three primary functions: physical isolation, stabilization/erosion protection, and chemical isolation. In some cases, multiple layers of different materials are used to fulfill these function and in some cases, a single layer may serve multiple functions. Project managers are encouraged to consider the use of performance-based measures for caps in remedy decisions to preserve flexibility in how the cap may be designed to fulfill these functions.

5.4.1 Physical Isolation Component

The cap should be designed to isolate contaminated sediment from the aquatic environment order to reduce exposure to protective levels. The physical isolation component of the cap should also include a component to account for consolidation of cap materials.

To provide long-term protection, a cap should be sufficiently thick to effectively separate contaminated sediment from most aquatic organisms that dwell or feed on, above, or within the cap. This serves two purposes: 1) to decrease exposure of aquatic organisms to contaminants, and 2) to decrease the ability of burrowing organisms to move buried contaminants to the surface (i.e., bioturbation). To design a cap component for this second purpose, the depth of the effective mixing zone (i.e., the depth of effective sediment mixing due to bioturbation and/or frequent sediment disturbance) and the population density of organisms within the sediment profile should be estimated and considered in selecting cap thickness. Especially in marine environments, the potential for colonization by deep burrowing organisms (e.g., certain species of mud shrimp) could lead to a decision to design a thicker cap. Measures to prevent colonization or disturbance of the cap by deep burrowing bottom-dwelling organisms can be considered in cap design, and in developing biological monitoring requirements for the project. Project managers should refer to Chapter 2, Section 2.8.3 and consult with aquatic biologists with knowledge of local conditions for evaluation of the bioturbation potential. In some cases, a site-specific biological survey of bioturbators would be appropriate. In addition, the USACE Technical Note *Subaqueous Cap Design: Selection of Bioturbation Profiles, Depths and Process Rates* [Clarke et al. 2001, (Dredging Operations and Environmental Research (DOER)-C21 at http://el.erdc.usace.army.mil/dots/doer/technote.html], provides information on designing in-situ caps and also provides many useful references on bioturbation. Although not usually a major pathway for contaminant release, project managers should also be aware of the potential for wetland/aquatic plants to penetrate a cap and create pathways for some contaminant migration.

The project manager should consider consolidation when designing the cap. Fine-grained granular capping materials can undergo consolidation due to their own weight. The thickness of granular cap material should have an allowance for consolidation so that the minimum required cap thickness is maintained following consolidation. An evaluation of consolidation is important in interpreting monitoring data to differentiate between changes in cap surface elevation or cap thickness due to consolidation, as opposed to erosion.

Even if the cap material is not compressible, most contaminated sediment is compressible and some may be highly compressible. Underlying contaminated sediment will almost always undergo some consolidation due to the added weight of the capping material or armor stone. The degree of consolidation should provide an indication of the volume of pore water expelled through the contaminated layer and capping layer to the water column due to consolidation. The consolidation-driven advection of pore water should be considered in the evaluation of short-term contaminant flux. Also, consolidation may decrease the vertical permeability of the capped sediment and thus reduce long-term flux. Methods used to define and quantify consolidation characteristics of sediment and capping materials, such as standard laboratory tests and computerized models, are available (U.S. EPA 1998d, Palermo et al. 1998a, Liu and Znidarcic 1991).

5.4.2 Stabilization/Erosion Protection Component

This functional component of the cap is intended to stabilize both the contaminated sediment and the cap itself to prevent either from being resuspended and transported from the capping location. The potential for erosion generally depends on the magnitude of the applied bed shear stresses due to river, tidal, and wave-induced currents, turbulence generated by ships/vessels (due to propeller action and vessel draft), and sediment properties such as particle size, mineralogy and bed bulk density. At some sites, there is also the potential for seismic disturbance, especially where contaminated sediment and/or cap material are of low shear strength. These and other aspects of investigating sediment stability are discussed in Chapter 2, Section 2.8, Sediment Stability and Contaminant Fate and Transport. Conventional methods for analysis of sediment transport are available to evaluate erosion potential of caps, ranging from simple analytical methods to complex numerical models (U.S. EPA 1998d, Palermo et al. 1998a). Uncertainty in the estimate of erosion potential should be evaluated as well.

The design of the erosion protection features of an in-situ cap (i.e., armor layers) should be based on the magnitude and probability of occurrence of relatively extreme erosive forces estimated at the capping site. Generally, in-situ caps should be designed to withstand forces with a probability of 0.01 per year, for example, the 100-year storm. As is discussed further in Chapter 2 (Section 2.8, Sediment Stability and Contaminant Fate and Transport), in some circumstances, higher or lower probability events should also be considered.

Another consideration for capping, especially capping of contaminated sediment with high organic content is whether significant gas generation due to anaerobic degradation will occur. Gas generation in sediment beneath caps, especially those constructed of low permeable materials, could either generate significant uplift forces and threaten the physical stability of the overlying capping material, or carry some contaminants through the cap. Little has been documented in this area to date, but the possible influence of this process on cap effectiveness presents an uncertainty the project manager should consider in the analysis of remedial alternatives.

5.4.3 Chemical Isolation Component

If a cap has a properly designed physical isolation component, contaminant migration associated with the movement of sediment particles should be controlled. However, the vertical movement of dissolved contaminants by advection (flow of ground water or pore water) through the cap is possible, while some movement of contaminants by molecular diffusion (movement across a concentration gradient) over long periods usually is inevitable. However, in assessing these processes, it is important to also assess the sorptive capacity of the cap material, which will act to retard contaminant flux through the cap, and the long-term fate of capped contaminants that may transform through time. Slow releases of dissolved contaminants through a cap at low levels will generally not create unacceptable exposures. If reduction of contaminant flux is necessary to meet remedial action objectives, however, a more involved analysis to include capping effectiveness testing and modeling should be conducted as a part of cap design. Because of the uncertainties involved in predicting future flux rates over very long time periods, this guidance does not advocate a particular minimum rule of thumb for the appropriate time frame for design with respect to chemical isolation. In general, it is reasonable for the physical isolation component (i.e., physical stability) of a cap design to be based on a shorter time frame (e.g., a disruptive event with a more frequent recurrence interval) than the much longer time frames considered in design for chemical isolation (e.g., the time required for accumulation of contaminants in the cap material or that required to

attain the maximum chemical flux through the cap), in part because erosion of small areas of a cap is easier to repair.

Nevertheless, both advective and diffusive processes should be considered in cap design. If a ground water/surface water interaction study indicates that advection is not significant over the area to be capped (e.g., migration of ground water upward through the cap would not prevent attaining the RAOs), the cap design may need to address only diffusion and the physical isolation and stabilization of the contaminated sediment. In this case, it may not be necessary to design for control of dissolved and/or colloidally facilitated transport due to advection (Ryan et al. 1995).

In contrast, where ground water flow upward through the cap is expected to be significant, the hydraulic properties of the cap should also be determined and factored into the cap design. These properties should include the hydraulic conductivity of the cap materials, the contaminated sediment, and underlying clean sediment or bedrock. According to a USACE laboratory study, ground water flow velocities exceeding 10^{-5} cm/sec potentially result in conditions in which equilibrium partitioning processes important to cap effectiveness could not be maintained (Myers et al. 1991). Such conditions should be carefully considered in the cap design. High rates of ground water flow through contaminated sediment may cause unacceptable exposures. In these areas, in-situ capping may not be an effective remedial approach without additional protective measures. Use of amended caps (caps containing reactive or sorptive material to sequester organic or inorganic contaminants) is one potential measure undergoing pilot studies. Project managers should refer to the Remediation Technologies Development Forum (RTDF) Web site at http://www.rtdf.org for the latest in-situ cleanup developments. More information on the interactions of ground water and in-situ caps can be found in the USACE Technical Note, *Subaqueous Capping and Natural Recovery: Understanding the Hydrogeologic Setting at Contaminated Sediment Sites* (Winter 2002).

Where non-aqueous phase liquids (NAPL) are present in part of an area to be capped, the process for potential contamination migration should be carefully considered. NAPL may be mobilized by consolidation-induced or ground water-induced advective forces. Field sampling and bench-scale tests such as the Seepage Induced Consolidation Test can be designed to test these issues (e.g., Hedblom et al. 2003). In situations where conventional cap designs are not likely to be effective, it may be possible to consider impervious materials (e.g., geomembranes, clay, concrete, steel, or plastic) or reactive materials for the cap design. Where this is done, however, care must be taken such that head increases along the edges of the impervious area do not lead to additional NAPL migration. Project managers are encouraged to draw on the experience of others who have conducted pilot or full scale caps in the presence of NAPL.

Laboratory tests can be used to calculate sediment- and capping material-specific diffusion and chemical partitioning coefficients. Several numerical models are available to predict long-term movement of contaminants due to advection and diffusion processes into or through caps, including caps with engineered components. The models can evaluate the effectiveness of varying thicknesses of granular cap materials with differing properties [grain size and total organic carbon (TOC)]. The results generated by such models include flux rates to overlying water and sediment and pore water concentrations in the entire sediment and cap profile as a function of time. These results can be compared to sediment remediation goals or applicable water quality criteria in overlying surface water, or interpreted in terms of a mass loss of contaminants as a function of time. Results could also be compared to similar calculations for other remediation technologies.

5.5 OTHER CAPPING CONSIDERATIONS

In preparing a feasibility study to evaluate in-situ capping for a site, project managers should consider the following:

- Identifying candidate capping materials physically and chemically compatible with the environment in which they will be placed;
- Evaluating geotechnical considerations including consolidation of compressible materials and potential interactions and compatibility among cap components;
- Assessing placement methods that will minimize short-term risk from release of contaminated pore water and resuspension of contaminated sediment during cap placement; and
- Identifying performance objectives and monitoring methods for cap placement and long-term assessment of cap integrity and biota effects.

In addition to evaluation during the feasibility study, these aspects should be addressed in more detail during design. These topics are discussed briefly below. In addition, project managers should refer to Chapter 8, Section 8.4.2 for a discussion of general monitoring considerations for in-situ capping, and to Chapter 3, Section 3.6 for a discussion of ICs that may relate to caps.

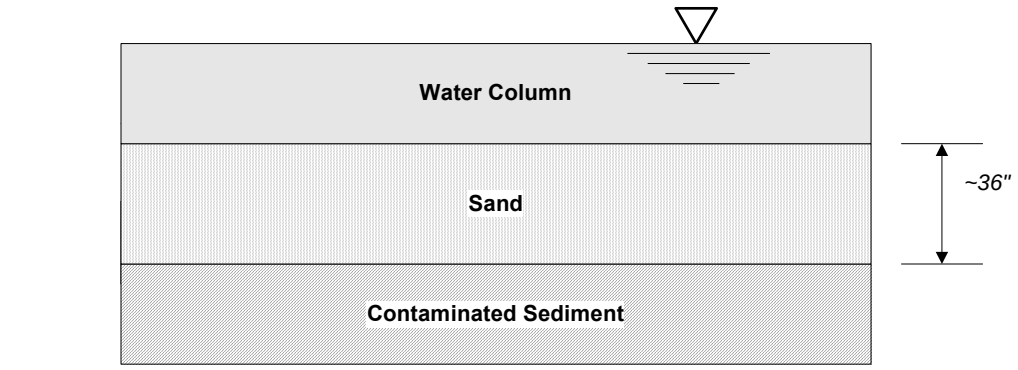
5.5.1 Identification of Capping Materials

Caps are generally composed of clean granular materials, such as upland sand-rich soils or sandy sediment; however, more complex cap designs could be required to meet site-specific RAOs. The project manager should take into consideration the expected effects of bioturbation, consolidation, erosion, and other related processes on the short- and long-term exposure and risk associated with contaminants. For example, if the potential for erosion of the cap is significant, the level of protection could be raised by increasing cap thickness or by engineering the cap to be more erosion-resistant through use of cap material with larger grain size, or by using an armor layer. Porous geotextiles do not contribute to contaminant isolation, but serve to reduce the potential for mixing and displacement of the underlying sediment with the cap material. A cap composed of naturally occurring sand is generally preferred over processed sand because the associated fine fraction and organic carbon content found in natural sands are more effective in providing chemical isolation by sequestering contaminants migrating through the cap. However, sand containing a significant fraction of finer material may also increase turbidity during placement.

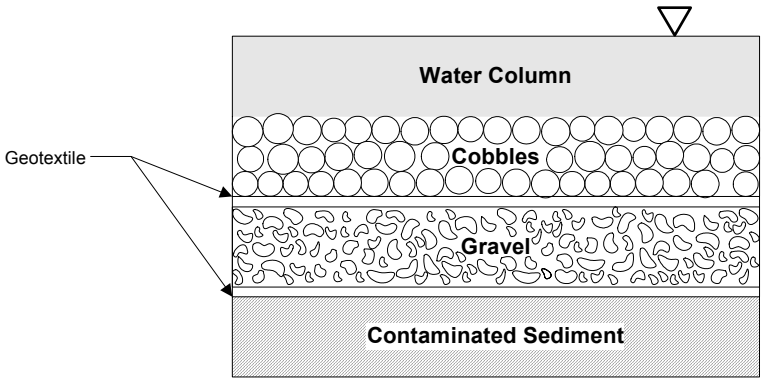
Specialized materials may be used to enhance the chemical isolation capacity or otherwise decrease the thickness of caps compared to sand caps. Examples include engineered clay aggregate materials (e.g., AquaBlok□), and reactive/adsorptive materials such as activated carbon, apatite, coke, organoclay, zero-valent iron and zeolite. Composite geotextile mats containing one or more of these materials (i.e., reactive core mats) are becoming available commercially.

Highlight 5-2 illustrates some examples of cap designs.

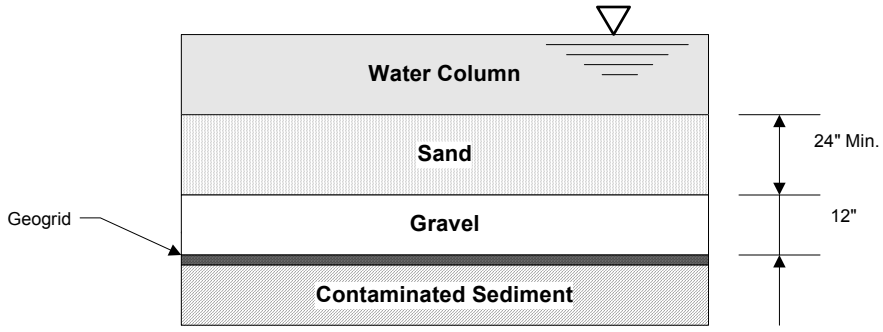
Highlight 5-2: Sample Cap Designs



A. Eagle Harbor, WA



B. Sheboygan, WI



C. Convair Lagoon, CA

Source: Modified from U.S. EPA 1998d

5.5.2 Geotechnical Considerations

Usually, contaminated sediment is predominately fine-grained, and often has high water content and low shear strength. These materials are generally compressible. Unless appropriate controls are implemented, contaminated sediment can be easily displaced or resuspended during cap placement. Following placement, cap stability and settlement due to consolidation can become two additional geotechnical issues that may be important for cap effectiveness.

As with any geotechnical problem of this nature, the shear strength of the underlying sediment will influence its resistance to localized bearing capacity or sliding failures, which could cause localized mixing of capping and contaminated materials. Cap stability immediately after placement is critical, before any excess pore water pressure due to the weight of the cap has dissipated. Usually, gradual placement of capping materials over a large area will reduce the potential for localized failures. Information on the behavior of soft deposits during and after placement of capping materials is limited, although some field monitoring data have shown successful sand capping of contaminated sediment with low shear strength. Conventional geotechnical design approaches should, therefore, be applied with caution (e.g., by building up a cap gradually over the entire area to be capped). Similarly, caps with flatter transition slopes at the edges are not generally subject to a sliding failure normally predicted by conventional slope stability analysis.

5.5.3 Placement Methods

Various equipment types and placement methods have been used for capping projects. The use of granular capping materials (i.e., sand, sediment, and soil), geosynthetic fabrics, and armored materials are all in-situ cap considerations discussed in this section. Important considerations in selection of placement methods include the need for controlled, accurate placement of capping materials. Slow, uniform application that allows the capping material to accumulate in layers is often necessary to avoid displacement of or mixing with the underlying contaminated sediment. Uncontrolled placement of the capping material can also result in the resuspension of contaminated material into the water column and the creation of a fluid mud wave that moves outside of the intended cap area.

Granular cap material can be handled and placed in a number of ways. Mechanically excavated materials and soils from an upland site or quarry usually have relatively little free water. Normally, these materials can be handled mechanically in a dry state until released into the water over the contaminated site. Mechanical methods (e.g., clamshells or release from a barge) rely on gravitational settling of cap materials in the water column, and could be limited by depth in their application. Granular cap materials can also be entrained in a water slurry and carried to the contaminated site wet, where they can be discharged by pipe into the water column at the water surface or at depth. These hydraulic methods offer the potential for a more precise placement, although the energy required for slurry transport could require dissipation to prevent resuspension of contaminated sediment. Armor layer materials can be placed from barges or from the shoreline using conventional equipment, such as clamshells. Placement of some cap components, such as geotextiles, could require special equipment. Examples of equipment types used for cap placement are shown in Highlight 5-3. The *Guidance for In-Situ Subaqueous Capping of Contaminated Sediments* (U.S. EPA 1998d) contains more detailed information about cap placement techniques.

Monitoring sediment resuspension and contaminant releases during cap placement is important. Cap placement can resuspend some contaminated sediment. Contaminants can also be released to the water column from compaction or disruption of underlying sediment during cap placement. Both can lead to increased risks during and following cap placement. Applying cap material slowly and uniformly can minimize the amount of sediment disruption and resuspension. Therefore, designs should include plans to minimize and monitor impacts during and after construction.

5.5.4 Performance Monitoring

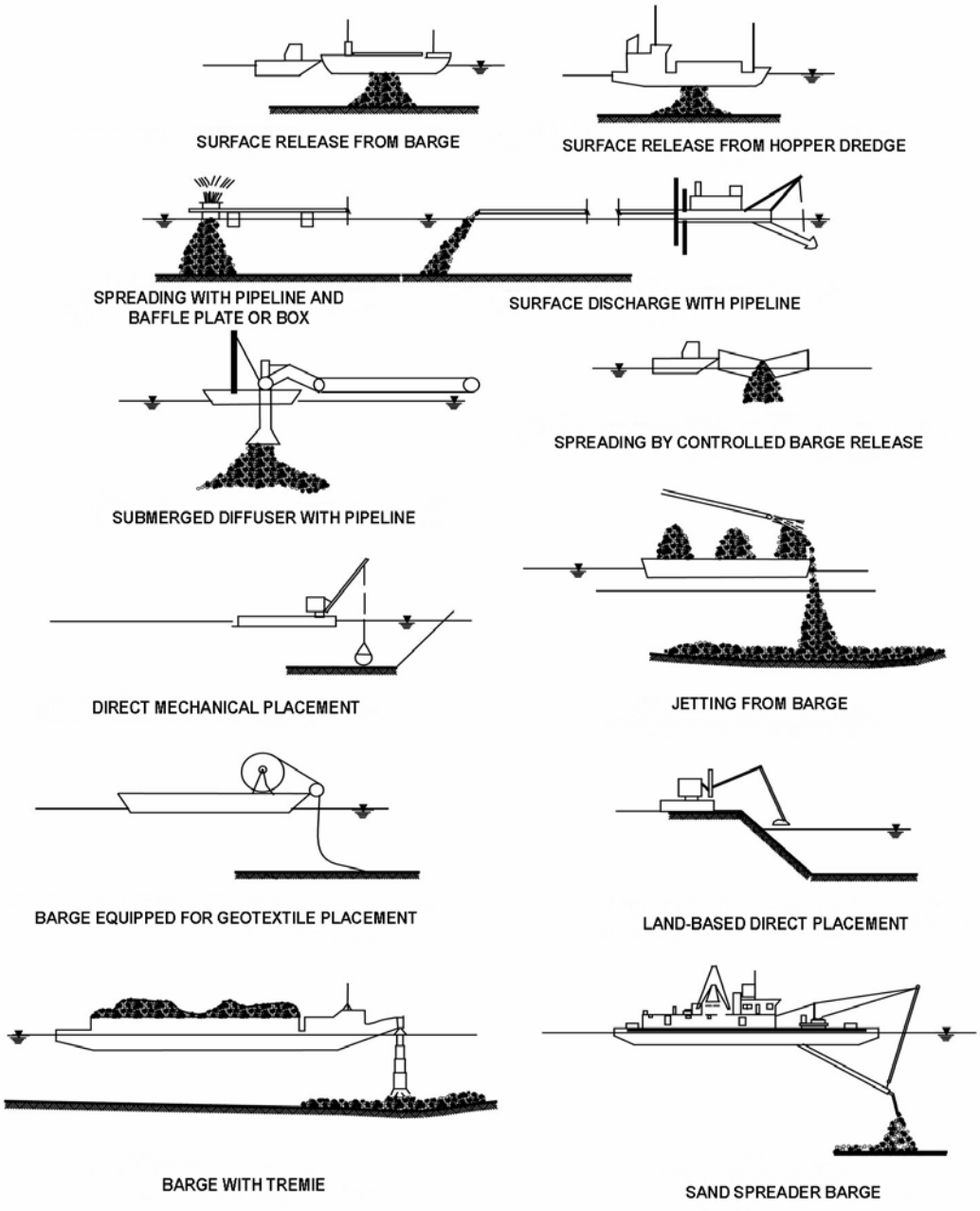
Performance objectives for an in-situ cap relate to its ability to provide sufficient physical and chemical isolation and stabilization of contaminated sediment to reduce exposure and risk to protective levels. Broader RAOs for the site such as decreases in contaminant concentrations in biota or reduced toxicity should be monitored when applicable. The following processes should be considered when evaluating the performance of a cap, and in developing a cap monitoring program:

- Erosion or other physical disturbance of cap;
- Contaminant flux into cap material and into the surface water from underlying contaminated sediment (e.g., ground water advection, molecular diffusion); and
- Recolonization of cap surface and resulting bioturbation.

General considerations related to monitoring caps and an example of cap monitoring elements are presented in Chapter 8, Remedial Action and Long-Term Monitoring.

Performance monitoring of a cap should be related to the design standards and remedial action objectives related to the site. Generally, physical monitoring is initially conducted on a more frequent schedule than chemical or biological monitoring because it is less expensive to perform. Some processes (i.e., contaminant flux) are not generally assessed directly because they are very difficult to measure, but are assessed by measuring contaminant concentrations in bulk samples from the cap surface, in shallow cores into the surface layer of a cap, and by bathymetric surveys and various photographic techniques. It is often desirable to establish several permanent locational benchmarks so that repeated surveys can be accurately compared. In some cases, contaminant flux and the resulting contaminant concentration in surface sediment, cap pore water, or overlying surface water can be compared to site-specific sediment cleanup levels or water quality standards (e.g., federal water quality criteria or state promulgated standards). In addition, the concentration of contaminants accumulating in the cap material as a function of time can be compared to site-specific target cleanup levels during long-term cap performance monitoring. Both analytical and numerical models exist to predict cap performance and have been compared and validated with laboratory tests and field results (e.g., Ruiz et al. 2000). However, project managers should be aware that representative chemical monitoring of caps is difficult, in part because of the need to distinguish between vertical migration into the cap and the mixing that occurs at the cap/sediment interface during placement. In some cases, physical measurement of cap integrity and water column chemical measurement may be sufficient for routine monitoring.

Highlight 5-3: Sample Capping Equipment and Placement Techniques



Source: U.S. EPA 1998d

Highlight 5-4 presents some general points to remember from this chapter.

Highlight 5-4: Some Key Points to Remember When Considering In-Situ Capping

- Source control generally should be implemented to prevent recontamination
- In-situ caps generally reduce risk through three primary functions: physical isolation, stabilization, and reduction of contaminant transport
- Caps may be most suitable where water depth is adequate, slopes are moderate, ground water flow gradients are low or contaminants are not mobile, substrates are capable of supporting a cap, and an adequate source of cap material is available
- Evaluation of capping alternatives and design of caps should consider buried infrastructure, such as water, sewer, electric and phone lines, and fuel pipelines
- Alteration of substrate and depth from capping should be evaluated for effects on aquatic biota
- Evaluation of a capping project in natural riverine environments, should include consideration of a fluvial system's inherent dynamics, especially the effects of channel migration, flow variability including extreme events, and ice scour
- Evaluation of capping alternatives should include consideration of cap disruption from human and natural sources, including at a minimum, the 100-year flood and other events such as seismic disturbances with a similar probability of occurrence
- Selection of cap placement methods should minimize the resuspension of contaminated sediment and releases of dissolved contaminants from compacted sediment
- Use of experienced contractors skilled in marine construction techniques is very important to placement of an effective cap
- Monitor in-situ caps during and after placement to evaluate long-term integrity of the cap, recolonization by biota, and evidence of recontamination
- Maintenance of in-situ caps is expected periodically

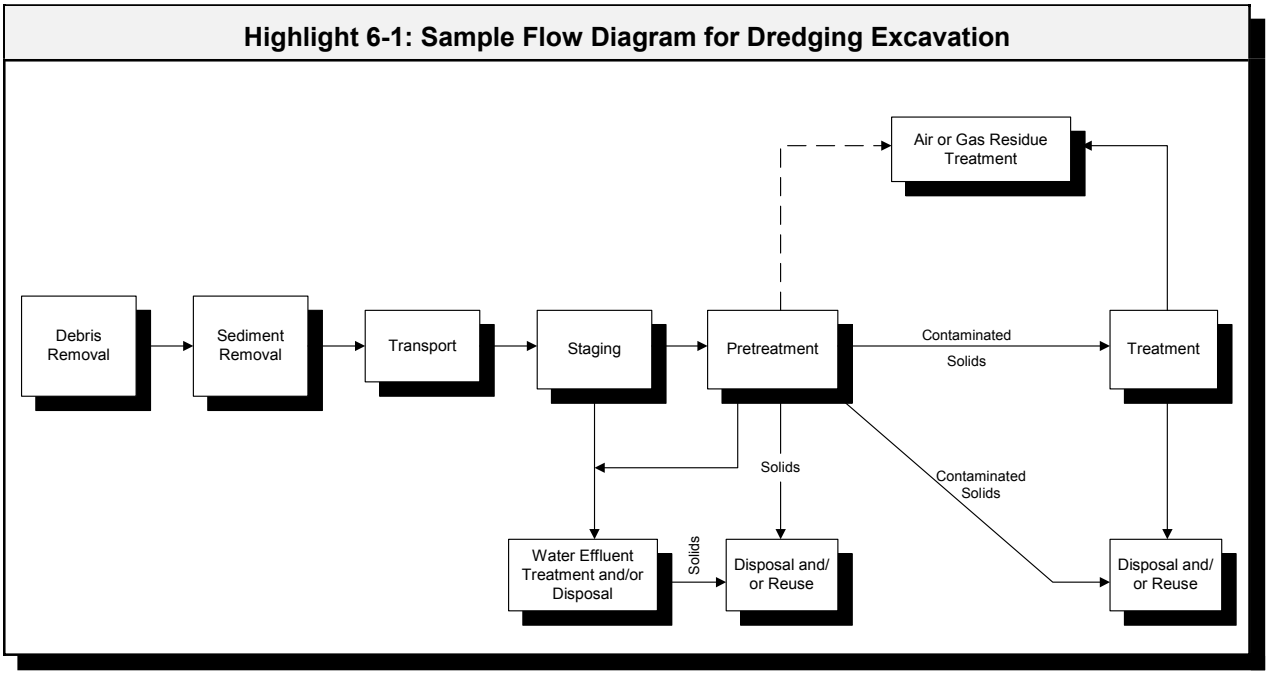
6.0 DREDGING AND EXCAVATION

6.1 INTRODUCTION

Dredging and excavation are the two most common means of removing contaminated sediment from a water body, either while it is submerged (dredging) or after water has been diverted or drained (excavation). Both methods typically necessitate transporting the sediment to a location for treatment and/or disposal. They also frequently include treatment of water from dewatered sediment prior to discharge to an appropriate receiving water body. Sediment is dredged by the U.S. Army Corps of Engineers (USACE) on a routine basis at numerous locations for the maintenance of navigation channels. The objective of navigational dredging is to remove sediment as efficiently and economically as possible to maintain waterways for recreational, national defense, and commercial purposes. Use of the term environmental dredging has evolved in recent years to characterize dredging performed specifically for the removal of contaminated sediment. Environmental dredging is intended to remove sediment contaminated above certain action levels while minimizing the spread of contaminants to the surrounding environment during dredging [National Research Council (NRC 1997)].

Some of the key components to be evaluated when considering dredging or excavation as a cleanup method include sediment removal, transport, staging, treatment (pretreatment, treatment of water and sediment, if necessary), and disposal (liquids and solids). Highlight 6-1 provides an sample flow diagram of the possible steps in a dredging or excavation alternative. The simplest dredging or excavation projects may consist of as few as three of the components shown in Highlight 6-1. More complex projects may include most or all of these components. Efficient coordination of each component typically is very important for a cost-effective cleanup. Project managers should recognize, in general, fewer sediment rehandling steps leads to lower implementation risks and lower cost.

Highlight 6-1: Sample Flow Diagram for Dredging Excavation



Sediment removal by dredging or excavation has been the most frequent cleanup method used by the Superfund program at sediment sites. Dredging or excavation has been selected as a cleanup method for contaminated sediment at more than 100 Superfund sites (some as an initial removal action). At approximately fifteen to twenty percent of these sites, an in-situ cleanup method [i.e., capping or monitored natural recovery (MNR)] was also selected for sediment at part of the site. When dredging is the selected remedy and hazardous substances left in place are above levels that allow for unlimited use and unrestricted exposure, a five-year review pursuant to the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) 121(c) may be required (U.S. EPA 2001i).

Project managers should also refer to the U.S. Environmental Protection Agency s (EPA s) *Assessment and Remediation of Contaminated Sediments (ARCS) Program Remediation Guidance Document* (U.S. EPA 1994d), and *Handbook: Remediation of Contaminated Sediments* (U.S. EPA 1991c), the NRC s *Contaminated Sediments in Ports and Waterways: Cleanup Strategies and Technologies* (NRC 1997), and *Operational Characteristics and Equipment Selection Factors for Environmental Dredging* (Palermo et al. 2004) for detailed discussions of the processes and technologies available for dredging and excavation.

Although each of the three potential remedy approaches (MNR, in-situ capping, and removal) should be considered at every site at which they might be appropriate, sediment removal by dredging or excavation should receive detailed consideration where the site conditions listed in Highlight 6-2 are present.

Highlight 6-2: Some Site Conditions Especially Conducive to Dredging or Excavation

- Suitable disposal site(s) is available and nearby
- Suitable area is available for staging and handling of dredged material
- Existing shoreline areas and infrastructure can accommodate dredging or excavation needs; maneuverability and access not unduly impeded by piers, buried cables, or other structures
- Navigational dredging is scheduled or planned
- Water depth is adequate to accommodate dredge but not so great as to be infeasible; or excavation in the dry is feasible
- Long-term risk reduction of sediment removal outweighs sediment disturbance and habitat disruption
- Water diversion is practical, or current velocity is low or can be minimized, to reduce resuspension and downstream transport during dredging
- Contaminated sediment overlies clean or much cleaner sediment (so that over-dredging is feasible)
- Sediment contains low incidence of debris (e.g., logs, boulders, scrap material) or is amenable to effective debris removal prior to dredging or excavation
- High contaminant concentrations cover discrete areas of sediment
- Contaminants are highly correlated with sediment grain size (to facilitate separation and minimize disposal costs)

6.2 POTENTIAL ADVANTAGES AND LIMITATIONS

One of the advantages of removing contaminated sediment from the aquatic environment often is that, if it achieves cleanup levels for the site, it may result in the least uncertainty about long-term effectiveness of the cleanup, particularly regarding future environmental exposure to contaminated sediment. Removal of contaminated sediment can minimize the uncertainty associated with predictions of sediment bed or in-situ cap stability and the potential for future exposure and transport of contaminants.

Another potential advantage of removing contaminated sediment is the flexibility it may leave regarding future use of the water body. In-situ cleanup methods such as MNR and capping frequently include institutional controls (ICs) that limit water body uses. Although remedies at sites with bioaccumulative contaminants usually require the development or continuation of fish consumption advisories for a period of time after removal, other types of ICs that would be needed to protect a cap or layer of natural sedimentation might not be necessary if contaminated sediment is removed.

Another advantage, especially where dredging residuals are low, concerns the time to achieve remedial action objectives (RAOs). Active cleanup methods such as sediment removal and, particularly, capping may reduce risk more quickly and achieve RAOs faster than would be achieved by natural recovery. (However, in comparing time frames between approaches, it is important to include accurate estimates of the time for design and implementation of active approaches.) Also, sediment removal is the only cleanup method that can allow for treatment and/or beneficial reuse of dredged or excavated material. (However, caps that incorporate treatment measures, sometimes called active caps, are under development by researchers. See Chapter 3, Section 3.1.3, In-Situ Treatment and Other Innovative Alternatives.)

There are also some potential sediment removal limitations that can be significant. Implementation of dredging or excavation is usually more complex and costly than MNR or in-situ capping because of the removal technologies themselves (especially in the case of dredging) and the need for transport, staging, treatment (where applicable), and disposal of the dredged sediment. Treatment technologies for contaminated sediment frequently offer implementation challenges because of limited full-scale experience and high cost. In some parts of the country, disposal capacity may be limited in existing municipal or hazardous waste landfills, and it may be difficult to locate new local disposal facilities. Dredging or excavation may also be more complex and costly than other approaches due to accommodation of equipment maneuverability and portability/site access. Operations and effectiveness may be affected by utilities and other infrastructures, surface and submerged structures (e.g., piers, bridges, docks, bulkheads, or pilings), overhead restrictions, and narrow channel widths.

Another possible limitation of sediment removal is the level of uncertainty associated with estimating the extent of residual contamination following removal that can be high at some sites. For purposes of this guidance, residual contamination is contamination remaining in the sediment after dredging within or adjacent to the dredged area. The mass and contaminant concentration of residuals is generally a result of many factors including dredge equipment, dredge operator experience, proper implementation of best management practices, sediment characteristics, and site conditions.

Residual contamination is likely to be greater in the presence of cobbles, boulders, or buried debris, in high energy environments, at greater water depths, and where more highly contaminated sediment lies

near the bottom of the dredge thickness or directly overlies bedrock or a hard bottom. Residuals may also be greater in very shallow waters and when dredging sediment with high water contents. These complicating factors can make the sediment removal process difficult and costly. The continued bioaccumulation of residual contaminants can also affect the achievement of risk-based remediation goals. Dredging residuals have been underestimated at some sites, even when obvious complicating factors are not present. For some sites, this has resulted in not meeting selected cleanup levels without also backfilling with clean material.

Another potential limitation of dredging effectiveness includes contaminant losses through resuspension and, generally to a lesser extent, through volatilization. Resuspension of sediment from dredging normally results in releases of both dissolved and particle-associated contaminants to the water column. Resuspended particulate material may be redeposited at the dredging site or, if not controlled, transported to downstream locations in the water body. Some resuspended contaminants may also dissolve into the water column where they are more available for uptake by biota. While aqueous resuspension generally is much less of a concern during excavation, there may be increased concern with releases to air. Losses en route to and/or at the disposal or treatment site may include effluent or runoff discharges to surface water, leachate discharges to ground water, or volatile emissions to air. Each component of a sediment removal alternative typically necessitates additional handling of the material and presents a possibility of contaminant loss, as well as other potential risks to workers and communities.

Finally, similar to in-situ capping, dredging or excavation includes at least a temporary destruction of the aquatic community and habitat within the remediation area.

Where it is feasible, excavation often has advantages over dredging for the following reasons:

- Excavation equipment operators and oversight personnel can much more easily see the removal operation. Although in some cases diver-assisted hydraulic dredging or video-monitored dredging can be used, turbidity, safety and other technological constraints typically result in dredging being performed without visual assistance;

- Removal of contaminated sediment is usually more complete (i.e., residual contamination tends to be lower when sediment is removed after the area is dewatered);

- Far fewer waterborne contaminants are released when the excavation area has been dewatered; and

- Bottom conditions (e.g., debris) and sediment characteristics (e.g., grain size and specific gravity) typically require much less consideration.

However, site preparation for excavation can be more lengthy and costly than for a dredging project due to the need for dewatering or water diversion. For example, coffer dams, sheet pile walls, or other diversions/exclusion structures would need to be fabricated and installed. Maneuvering around diversion/exclusion structures may be required because earth moving equipment cannot access the excavation area or double handling may be required to move material outside of the area. In addition, excavation is generally limited to relatively shallow areas.

6.3 SITE CONDITIONS

6.3.1 Physical Environment

Several aspects of the physical environment may make sediment removal more or less difficult to implement. In the remedial investigation, the following types of information should be collected, as they can affect the type of equipment selected and potentially the feasibility of sediment removal:

- Bathymetry, slope of the sediment surface and water depth;
- Currents and tides;
- Bottom conditions, especially the presence of debris and large rocks both on top of and within the sediment bed;
- Depth to and (un)evenness of bedrock or hard bottom (e.g., stiff glacial till);
- Sediment particle size distribution, degree of consolidation, and shear strength;
- Thickness and vertical delineation of contaminated sediment;
- Distance between dredging and disposal locations;
- The presence and maintenance condition of structures such as piers, pilings, cables, or pipes; and
- Land access to water body.

Additionally, sediment removal may change the hydrodynamics and slope stability of the remediation area. These changes should be evaluated to ensure that the removal activity does not cause significant bank or structural instability, shoreline facility damages, or other unacceptable adverse effects in or near the removal operation.

Data on both the horizontal and vertical characterization of the physical and chemical sediment characteristics are generally needed during the remedial investigation to evaluate the feasibility, cost, and potential effectiveness of dredging or excavation. The results of this characterization should help determine the area, depth, and volume to be removed, and the volume of sediment requiring treatment and/or disposal. Some aspects of sediment characterization are discussed in Chapter 2, Section 2.1, Site Characterization.

The project manager should refer to *Evaluation of Dredged Material Proposed for Disposal at Island, Nearshore or Upland Confined Disposal Facilities - Testing Manual* (USACE 2003) and *Evaluation of Dredged Material Proposed for Discharge in Waters of the U.S. - Inland Testing Manual* (U.S. EPA and USACE 1998) for further information. In addition, several guidance documents on estimating contaminant losses from dredging and disposal have been developed by the EPA and USACE. For example, the project manager should refer to *Estimating Contaminant Losses from Components of Remediation Alternatives for Contaminated Sediments* (U.S. EPA 1996e).

6.3.2 Waterway Uses and Infrastructures

Any evaluation of the feasibility of a dredging or excavation remedy should consider impacts to existing and reasonably anticipated future uses of a waterway. Waterway uses that may need to be considered when evaluating a sediment removal alternative include the following:

- Navigation (e.g., commercial, military, recreational);
- Residential/commercial/military moorage and anchorage;
- Flood control;
- Recreation;
- Fishing (e.g., subsistence, commercial, recreational);
- Water supply, such as presence of intakes;
- Storm water or effluent discharge outfalls;
- Use by fish and wildlife, especially sensitive or important aquatic habitats;
- Waterfront development;
- Utility crossings; and
- Existing dredge disposal sites.

Evaluation of the feasibility of a sediment removal remedy should include an analysis of whether impacts to these potential uses may be avoided or minimized both during construction and in the long term.

6.3.3 Habitat Alteration

The project manager should consider the impact of habitat loss or alteration in evaluating a dredging or excavation alternative. As is also discussed in Chapter 5, In-Situ Capping, while a project may be designed to minimize habitat loss, or even enhance habitat, sediment removal and disposal do alter the environment. It is important to determine whether the loss of a contaminated habitat is a greater impact than the benefit of providing a new, modified but less contaminated habitat. For example, a sediment removal alternative may or may not be appropriate where extensive damage to an existing forested wetland will occur. If the contaminated sediment in the wetland is bioavailable and may be impacting wildlife populations, the short-term disruption of the habitat may be warranted to limit ongoing long-term impacts to wildlife. Comparatively, if the wetland is functioning properly and is not acting as a contaminant source to the biota and the surrounding area, it may be appropriate to leave the wetland intact rather than remove the contaminated sediment. Deliberations to alter wetland and aquatic habitats should be considered in the remedial decision process. Appropriate coordination with natural resource agencies

will typically assist the project manager in determining the extent of impacts that a dredging project may have on aquatic organisms or their habitat, and how to minimize these impacts.

Another consideration is avoidance of short-term ecological impacts during dredging. This may involve timing the project to avoid water quality impacts during migration and breeding periods of sensitive species or designing the dredging project to minimize suspended sediment during dredging and disposal.

6.4 EXCAVATION TECHNOLOGIES

Excavation of contaminated sediment generally involves isolating the contaminated sediment from the overlying water body by pumping or diverting water from the area, and managing any continuing inflow followed by sediment excavation using conventional dry land equipment. However, excavation may be possible without water diversion in some areas such as wetlands during dry seasons or while the sediment and water are frozen during the winter. Typically, excavation is performed in streams, shallow rivers and ponds, or near shore areas.

Prior to pumping out the water, the area can be isolated using one or more of the following technologies:

- Sheet piling;
- Earthen dams;
- Cofferdams;
- Geotubes, inflatable dams;
- Rerouting the water body using temporary dams or pipes; or
- Permanent relocation of the water body.

Sediment isolation using sheet piling commonly involves driving interlocking metal plates (i.e., sheet piles) into the subsurface, and thereby either blocking off designated areas or splitting a stream down the center. Highlight 6-3 shows an example of where this technology has been used. If a stream is split down its center, then one side of the stream may be excavated in the dry, after pumping out the trapped water. When the excavation of the first side of the stream is completed, water may be diverted back to the excavated side and sediment on the other side may be excavated. Sheet piling may not be feasible where bedrock or hard strata are present at or near the bottom surface. Where sheet piling is used to isolate a dredging or excavation action, project managers should consider potential hydraulic impacts of the diverted flow. Such diversion in most cases will increase natural flow velocity, which may scour sediment outside the diversion wall. If the sediment is also contaminated, as is likely to be the case, the increased dispersion of the sediment should be considered in design choices. Temporarily rerouting a water body with dams is sometimes done for small streams or ponds (Highlight 6-4). This includes the use of temporary dams to divert the water flow allowing excavation of now dry contaminated sediment. The ability and cost to provide hydraulic isolation of the contaminated area during remediation is a major factor in selecting the appropriate removal technology.

Once isolated, standing water within the excavation area will need to be removed. Although surface water flows are eliminated, ground water may infiltrate the confined area. The ground water can be collected in sumps or dewatering wells. After collection, the ground water should be characterized, managed, treated (if necessary), and discharged to an appropriate receiving water body. Management of water within the confined area is another important logistical and cost factor that can influence the decision of wet versus dry removal techniques.

Highlight 6-3: Example of Excavation Following Isolation Using Sheet Piling

Source: Pine River/Velsicol, EPA Region 5

Isolation and dewatering of the area is normally followed by excavation using conventional earthmoving equipment such as a backhoe or dragline. Where sediment is soft, support of the excavation equipment in the dewatered area can be problematic because underlying materials may not have the strength to support equipment weight. This also may reduce excavation depth precision. Both factors should be accounted for in design. When the excavation activities are complete, temporary dam(s) or sheet piling(s) are removed, and the water body is restored to its original hydraulic condition.

Another less common type of excavation project involves permanent relocation of a water body (also shown in Highlight 6-4). This, for example, was accomplished at the Triana/Tennessee River Superfund Site in Alabama and is being implemented at the Moss-American Superfund site in Wisconsin. The initial phases of such a project may be similar to excavation projects that temporarily reroute a water body. However, in a permanent stream relocation project, a replacement stream normally is constructed and then the original water body is excavated or capped and converted into an upland area. To the extent the original water body is covered over, direct exposure to residual contamination is generally eliminated.

Highlight 6-4: Examples of Permanent or Temporary Rerouting of a Water Body

A: Permanent River Relocation □ Triana Tennessee River Site

The Triana/Tennessee River site consists of an 11-mile stretch of two tributaries, the Huntsville Spring Branch (HSB) and Indian Creek, which both empty into the Tennessee River. Remedial actions involved rerouting of the channel in Huntsville Spring Branch (HSB mile 5.4 to 4.0), the filling and burial in place of the total DDT (dichloro diphenyl trichloroethane and its metabolites) in the old channel, the construction of diversion structures at the upper and lower end of the stream to prevent stream reversion to the former stream channel, and the diversion of storm water runoff to prevent flow across the filled channel. Remedial actions for HSB mile 4.0 to 2.4 consisted of constructing four diversion structures; excavating a new channel between HSB mile 3.4 and 2.4; filling three areas; constructing a diversion ditch around the fill areas; and excavating portions of the sediment from the channel.

These remedial actions effectively isolated in place 93% of the total DDT in the Huntsville Spring Branch-Indian Creek system of the Tennessee River. These remedial actions began on April 1, 1986, and were completed on October 16, 1987. Through March 1, 2001, the remedial actions have been inspected yearly by a federal and state Review Panel. The remedial action has not required any repair of the structures to maintain their integrity, and monitoring has shown that total DDT concentrations in fish and water continue to decline.

B: Temporary ReRouting of a River □ Bryant Mill Pond Project at the Allied Paper, Inc. Portage Creek Kalamazoo River Site

In EPA Region 5, an EPA-conducted removal and onsite containment action removed polychlorinated biphenyls (PCBs)-contaminated sediment from the Bryant Mill Pond area of Portage Creek. During the removal action, that was conducted from June 1998 - May 1999, Portage Creek was temporarily diverted from its normal streambed so that 150,000 yds^3 of the creek bed and floodplain soils could be excavated using conventional excavation equipment. PCB concentrations remaining after the removal action were below 1 ppm.

Source: U.S. EPA Region 5

Excavation may also include excavation of sediment in areas that experience occasional dry conditions, such as intermittent streams and wetlands. These types of projects generally are logistically similar to upland construction projects and frequently use conventional earthmoving equipment.

6.5 DREDGING TECHNOLOGIES

For purposes of this guidance the term dredging means the removal of sediment from an underwater environment, typically using floating excavators called dredges. Dredging involves mechanically grabbing, raking, cutting, or hydraulically scouring the bottom of a waterway to dislodge the sediment. Once dislodged, the sediment may be removed from a waterway either mechanically with buckets or hydraulically by pumping. Therefore, dredges may be categorized as either mechanical or hydraulic depending on the basic means of removing the dredged material. Some dredges employ

pneumatic (compressed air) systems to pump the sediment out of the waterway (U.S. EPA 1994d); however, these have not gained general acceptance on environmental dredging projects.

6.5.1 Mechanical Dredging

The fundamental difference between mechanical and hydraulic dredging equipment is how the sediment is removed. Mechanical dredges offer the advantage of removing the sediment at nearly the same solids content and, therefore, volume as the in-situ material. Little additional water is entrained with the sediment as it is removed. Thus, the volumes of contaminated material and process water to be disposed, managed, and/or treated are minimized. However, the water that is present in the bucket above the sediment must either be collected, managed, and treated, or be permitted to leak out, which generally leads to higher contaminant losses during dredging.

The mechanical dredges most commonly used in the U.S. for environmental dredging are the following (Palermo et al. 2004):

- *Clamshell:* Wire supported, conventional open clam bucket, circular shaped cutting action;

- *Enclosed bucket:* Wire supported, near watertight or sealed bucket as compared to conventional open clam bucket (recent designs also incorporate a level cut capability as compared to a circular-shaped cut for conventional buckets, for example, the Cable Arm and Boskalis Horizontal Closing Environmental Grab); and

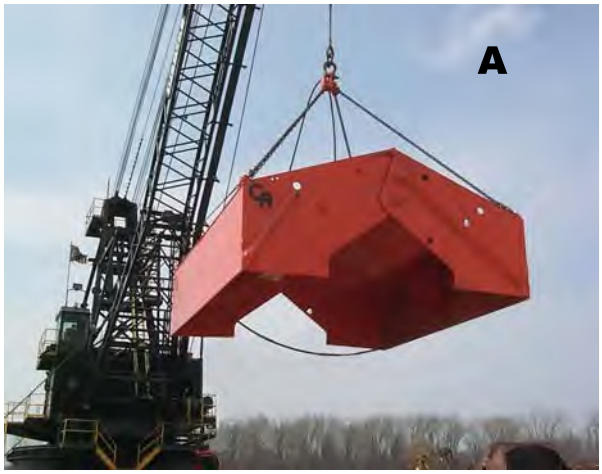
- *Articulated mechanical:* Backhoe designs, clam-type enclosed buckets, hydraulic closing mechanisms, all supported by articulated fixed-arm (e.g., Ham Visor Grab, Bean Horizontal Profiling Grab (HPG), Toa High Density Transport, and the Dry Dredge).

The mechanical dredge types listed above reflect equipment used for environmental dredging and generally are readily available in the U.S. The enclosed bucket dredges were designed to address a number of issues often raised relative to remedial dredging including contaminant removal efficiency and minimizing sediment resuspension. However, newly redesigned dredging equipment may not be cost-effective or preferred at every site. For example, in some environments, an enclosed bucket may be most useful for soft sediment but may not close efficiently on debris. A conventional clamshell dredge may have greater leverage and be able to close on or cut debris in some cases; however, material mounded over the top may be resuspended. An articulated mechanical dredge may have advantage in stiffer sediment since the fixed-arm arrangement can push the bucket into the sediment to the desired cut-level, and not rely on the weight of the bucket for penetration. Highlight 6-5 shows two examples of mechanical dredges.

6.5.2 Hydraulic Dredging

Hydraulic dredges remove and transport sediment in the form of a slurry through the inclusion or addition of high volumes of water at some point in the removal process (Zappi and Hayes 1991). The total volume of material processed may be greatly increased and the solids content of the slurry may be considerably less than that of the in-situ sediment although solids content varies between dredges (U.S. EPA 1994d). The excess water is usually discharged as effluent at the treatment or disposal site and often

Highlight 6-5: Examples of Mechanical Dredges



Note: A = Cable Arm Corp. dredge (Source: Cable Arm, Corp.)
B = Bean Company Horizontal Profiling Grab (HPG) dredge, New Bedford Harbor Site (Source: Barbara Bergen, U.S. EPA)

needs treatment prior to discharge. Hydraulic dredges may be equipped with rotating blades, augers, or high-pressure water jets to loosen the sediment (U.S. EPA 1995b). The hydraulic dredges most commonly used in the U.S. for environmental dredging are the following (Palermo et al. 2004):

- *Cutterhead:* Conventional hydraulic pipeline dredge, with conventional cutterhead;
- *Horizontal auger:* Hydraulic pipeline dredge with horizontal auger dredgehead (e.g., Mudcat);
- *Plain suction:* Hydraulic pipeline dredge using dredgehead design with no cutting action, plain suction (e.g., cutterhead dredge with no cutter basket mounted, Matchbox dredgehead, articulated Slope Cleaner, Scoop-Dredge BRABO, etc.);

- *Pneumatic:* Air operated submersible pump, pipeline transport, either wire supported or fixed-arm supported (e.g., Japanese Oozer, Italian Pneuma, Dutch d, Japanese Refresher, etc.);

- *Specialty dredgeheads:* Other hydraulic pipeline dredges with specialty dredgeheads or pumping systems (e.g., Boskalis Environmental Disc Cutter, Slope Cleaner, Clean Sweep, Water Refresher, Clean Up, Swan 21 Systems, etc.); and

- *Diver assisted:* Hand-held hydraulic suction with pipeline transport.

Some of the hydraulic dredges included above have been specifically developed to reduce resuspension during the removal process. As with modified mechanical dredges, project managers should be aware that there may be tradeoffs in terms of production rate and ability to handle debris with many of these modifications. Highlight 6-6 presents examples of hydraulic dredges.

6.5.3 Dredge Equipment Selection

The selection of appropriate dredging equipment is generally essential for an effective environmental dredging operation. The operational characteristics of the three types of mechanical and six types of hydraulic dredges presented in the guidance sections above are listed in Highlights 6-7a and 6-7b. This information was reviewed by an expert panel and attendees at a special session on environment dredging at the Meeting of the Western Dredging Association (WEDA XXI) and the 33rd Annual Texas A&M Dredging Seminar in Houston, Texas. The operational characteristics and identified selection factors presented in Highlights 6-7a and 6-7b have been drawn from information compiled for this guidance as well as earlier published reviews of dredge characteristics. Quantitative operational characteristics (both capabilities and limitations) are summarized for conditions likely to be encountered for many environmental dredging projects. The numbers are not representative of all dredge designs and sizes available, but represent those most commonly used for environmental dredging. Qualitative selection factors for each dredge type are presented based on the best professional judgment of the panel and/or their interpretation of readily available data. Site-specific results and supporting references are available in *Operational Characteristics and Equipment Selection Factors for Environmental Dredging* (Palermo et al. 2004).

The information in Highlights 6-7a and 6-7b is intended to help project managers make initial screening assessments of general dredge capabilities and identify equipment types for further evaluation at the feasibility study stage or for pilot field testing. Note that whenever an equipment type receives a rating of high, it means that a particular dredge type should perform better for that selection factor. It is not intended as a guide for final equipment selection for remedy implementation. There are many site-specific circumstances that dictate which equipment type is most appropriate for any given situation, and each type can be applied in different ways to adapt to site conditions. Project managers should use their own experience and judgment in using this information, and may find it useful to consider other sources of information for purposes of comparison. In addition, because new equipment is being continuously developed and tested, project managers will need to consult with experts who are familiar with the latest in equipment technologies. Experience has shown that an effective environmental dredging operation also depends on the use of highly skilled dredge operators familiar with the goals of environmental remediation, in addition to close monitoring and management of the dredging operation.

Highlight 6-6: Examples of Hydraulic Dredges

Note: A = Fox River, WI; horizontal auger hydraulic dredge deployment (Source: Jim Hahnenberg U.S. EPA)
B = Manistique, MI; closeup of twin-vortex pump, hydraulic dredge cutterhead (Source: Ernie Watkins U.S. EPA)
C = Closeup of swinging ladder hydraulic dredge cutterhead (Source: Ellicott Corporation)

Highlight 6-7a: Sample Environmental Dredging Operational Characteristics and Selection Factors[1]

	EQUIPMENT TYPE[2]									
	Mechanical Dredges (2 to 8 cubic meter buckets)			Hydraulic Pneumatic Dredges (15 to 30 cm pump sizes)						Dry Excavation
	Conventional Clamshell (Wire)[3]	Enclosed Bucket (Wire)[4]	Articulated Mechanical (Fixed Arm)[5]	Cutter-head[6]	Horizontal Auger[7]	Plain Suction[8]	Pneumatic[9]	Specialty[10]	Diver[11]	Various Mechanical Excavators[12]
OPERATIONAL CHARACTERISTICS[13]										
Operating Production Rate (m^3/hr)[14]	48 (2 m^3 bucket) 95 (4 m^3 bucket) 143 (6 m^3 bucket) 193 (8 m^3 bucket)			23 (15 cm pump) 41 (20 cm pump) 64 (25 cm pump) 93 (30 cm pump)			Site Specific	Equipment Specific	10	Site Specific
Percent Solids (by weight)[15]	Near In-Situ	Near In-Situ	Near In-Situ	5	5	5	15 or Higher	Equipment Specific	<5	In-Situ or Greater
Vertical Operating Accuracy (cm)[16]	15	15	10	10	10	10	15	10	--	5
Horizontal Operating Accuracy (cm)[17]	10	10	10	10	10	10	10	10	–	5
Maximum Dredging Depth (m)[18]	Stability Limitations	Stability Limitations	15	15	5	15	45	15	30	Stability Limitations
Minimum Dredging Depth (m)[19]	--	--	--	1	0.5	1	5	1	0.5	--

	EQUIPMENT TYPE[2]									
	Mechanical Dredges (2 to 8 cubic meter buckets)			Hydraulic Pneumatic Dredges (15 to 30 cm pump sizes)						Dry Excavation
	Conventional Clamshell (Wire)[3]	Enclosed Bucket (Wire)[4]	Articulated Mechanical (Fixed Arm)[5]	Cutter-head[6]	Horizontal Auger[7]	Plain Suction[8]	Pneumatic[9]	Specialty[10]	Diver[11]	Various Mechanical Excavators[12]
EQUIPMENT SELECTION FACTORS[20]										
Limit Sediment Resuspension[21]	Low	High	High	Medium	Medium	High	High	High	High	High
Control Contaminant Release [22]	Low	High	High	Medium	Medium	Medium	Medium	Medium	High	High
Minimize Residual Sediment[23]	Low	Medium	Medium	Medium	Medium	Medium	Medium	Medium	High	High
Transport by Pipeline[24]	Medium	Medium	Medium	High	High	High	High	High	High	Medium
Transport by Barge[25]	High	High	High	Medium	Medium	Medium	Medium	Medium	Low	High
Positioning Control in Currents/Wind/ Tides[26]	High	High	High	High	Medium	High	High	High	Medium	High
Maneuverability[27]	High	High	High	Low	Low	Low	Low	Low	High	High
Portability/ Access[28]	High	High	High	High	High	High	High	Medium	High	High
Availability[29]	High	High	High	High	High	High	Medium	Medium	High	High

	EQUIPMENT TYPE[2]									
	Mechanical Dredges (2 to 8 cubic meter buckets)			Hydraulic Pneumatic Dredges (15 to 30 cm pump sizes)						Dry Excavation
	Conventional Clamshell (Wire)[3]	Enclosed Bucket (Wire)[4]	Articulated Mechanical (Fixed Arm)[5]	Cutter-head[6]	Horizontal Auger[7]	Plain Suction[8]	Pneumatic[9]	Specialty[10]	Diver[11]	Various Mechanical Excavators[12]
Debris/Loose Rock/ Vegetation[30]	High	High	High	Low	Low	Low	Low	Low	Low	High
Hardpan/Rock Bottom[31]	Low	Low	Low	Low	Low	Medium	Medium	Medium	High	High
Flexibility for Varying Conditions[32]	High	High	Medium	High	Medium	Low	Low	Low	Low	High
Thin Lift/Residual Removal[33]	Low	Medium	Medium	Medium	High	High	High	High	High	High

Note: For additional information on development and technical basis for the entries in this table refer to: Palermo, M., N. Francingues, and D. Averett. 2004. Operational Characteristics and Equipment Selection Factors for Environmental Dredging. *Journal of Dredging Engineering*, Western Dredging Association.

Highlight 6-7b: Footnotes for Sample Environmental Dredging Operational Characteristics and Selection Factors	
1	This table provides some of the currently available general information that can help project managers initially assess dredge capabilities, and screen and select equipment types for evaluation at the feasibility study stage or for pilot field testing. This table is NOT intended as a guide for final equipment selection for remedy implementation, and regions may find it useful to consider other sources of information for purposes of comparison. There are many site-specific, sediment-specific, and project-specific circumstances that will indicate which equipment is most appropriate for any given situation, and each equipment type can be applied in different ways to adapt to site and sediment conditions. In addition, because new equipment is being continuously developed, project managers should consult with experts who are familiar with the latest technologies.
2	Equipment types shown here are considered the most commonly used for environmental dredging in the U.S. Other dredge types are available. Equipment used for environmental dredging is usually smaller in size than that commonly used for navigation dredging. Information presented here is tailored for mechanical bucket sizes from 3 to 10 cubic yards (about 2 to 8 m^3), and hydraulic/pneumatic pump sizes from 6 to 12 inches (about 15 to 30 cm). Larger sizes are available for many equipment types.
3	Clamshell - conventional clamshell dredges, wire supported, conventional open clam bucket.
4	Enclosed Bucket - wire supported, near watertight or sealed bucket usually incorporating a level cut capability.
5	Articulated Mechanical - backhoe designs, clam-type enclosed buckets, hydraulic closing mechanisms, all supported by articulated fixed-arm.
6	Cutterhead - conventional hydraulic pipeline dredge, with conventional cutterhead.
7	Horizontal Auger - hydraulic pipeline dredge with horizontal auger dredgehead.
8	Plain Suction - hydraulic pipeline dredge using dredgehead design with no cutting action.
9	Pneumatic – air operated submersible pump, pipeline transport, either wire supported or fixed-arm supported.
10	Specialty Dredgeheads - other hydraulic pipeline dredges with specialty dredgeheads or pumping systems
11	Diver Assisted - hand-held hydraulic suction with pipeline transport.
12	Dry Excavation - conventional excavation equipment operating within dewatered containments such as sheet-pile enclosures or cofferdams.
13	OPERATIONAL CHARACTERISTICS - quantitative entries, reflecting capabilities and limitations of dredge types, and are solely a function of the equipment itself.
14	Production Rate - in-situ volume of sediment removed per unit time. Rates shown are for production cuts as opposed to □cleanup passes□ and are for active periods of operation under average conditions. Rates for two bucket or pump sizes are shown for comparison. For mechanical dredges, the rates were calculated assuming 80% bucket fill with a bucket cycle time of 2 minutes. For hydraulic dredges, the rates were calculated assuming in-situ sediment 35% solids by weight, 5% solids by weight for slurry, and pump discharge velocity of 10 ft/sec. The rate shown for diver-assisted assumes a maximum pump size of 15 cm and roughly 50% efficiency of diver effort while working. Production rate for dry excavation is would be largely dictated by the time required to isolate and dewater the areas targeted for excavation. A variety of factors may influence the effective operating time per day, week, or season, and should be considered in calculating times required for removal.
15	Percent Solids by Weight - ratio of weight of dry solids to total weight of the dredged material as removed, expressed as a percentage. Percent solids for mechanical dredging is a function of the in-situ percent solids and the effective bucket fill (expressed as a percentage of the bucket capacity filled by in-situ sediment as opposed to free water), and near in-situ percent solids is possible for production cuts. A wide range of percent solids for hydraulic dredges is reported, but 5% solids can be expected for most environmental dredging projects.

Highlight 6-7b: Footnotes for Sample Environmental Dredging Operational Characteristics and Selection Factors	
16	Vertical Operating Accuracy - the ability to position the dredgehead at a desired depth or elevation for the cut and maintain or repeat that vertical position during the dredging operation. Although positioning instrumentation is accurate to within a few cm, the design of the dredge and the linkages between the dredgehead and the positioning system will affect the accuracy attainable in positioning the dredgehead. A vertical accuracy of cut of approximately 15 cm (one-half foot) is considered attainable for most project conditions. Fixed arm equipment holds some advantage over wire-supported in maintaining vertical operating accuracy. The accuracies achievable for sediment characterization should be considered in setting performance standards for environmental dredging operating accuracy (both vertical and horizontal).
17	Horizontal Operating Accuracy - the ability to position and operate the dredgehead at a desired location or within a desired surface area. Considerations are similar to those for vertical accuracy.
18	Maximum Dredging Depth - physical limitation to reach below a given depth. Wire-supported buckets or pumps can be deployed at substantial depths, so the maximum digging depth generally is limited by stability of the excavation. Reach of fixed arm supported buckets or hydraulic dredges is limited by the length of the arm or ladder. Conventional backhoe equipment is generally limited to about 15 m reach. Smaller hydraulic dredges are usually designed for a maximum dredging depth of about 15 m. Hydraulic dredges usually also have a limiting depth of removal of about 50 ft due to the limitation of atmospheric pressure, but this limitation can often be overcome by addition of a submerged pump on the ladder. The table entries should NOT be considered as hard and fast limits. Larger dredge sizes and designs are available for deeper depths.
19	Minimum Dredging Depth - constraints on draft limitations of some floating dredges or potential loss of pump prime for hydraulic dredges. Such limitations can be managed if the dredge □digs its way into the area.□ For smaller dredges, these limitations typically are at approximately the 1m water depth. Pneumatic dredges require a minimum water depth of about 5 m for efficient pump operation.
20	SELECTION FACTORS - qualitative entries, reflecting the potential performance of a given dredge type, and are a function of both the capability of the equipment type and the site and/or sediment conditions. Entries defined as follows: (High) - indicating the given dredge type is generally suitable or favorable for a given issue or concern, (Medium) - indicating the given dredge type addresses the issue or concern, but it may not be preferred, and (Low) - indicating the given dredge type may not be a suitable selection for addressing this issue or concern.
21	Limit Sediment Resuspension - potential of a given dredge type in minimizing sediment resuspension. Clamshell (Low) - Circular-shaped cutting action, cratered bottom subject to sloughing, open bucket design subject to washout and spillage, scows and workboats working in shallow areas. Enclosed Bucket (High) - Seal around the lips of the bucket and an enclosed top when in the shut position, level cut design minimizes sloughing. Articulated Mechanical (High) - Less resuspension as compared to conventional clamshell dredges. Cutterhead/Horizontal Auger (Medium) - Conventional cutterhead dredges and horizontal augers result in less resuspension as compared to conventional clamshell dredges. May be fitted with hoods or shrouds to partially control resuspension. Plain Suction/Pneumatic (High) - No mechanical action to dislodge the material. Specialty (High) - Although designs vary, all the so-called specialty dredges have features specifically intended to reduce resuspension. Diver Assisted (High) - Precision of diver assisted hydraulic dredging, the smaller size of the dredgeheads used, and inherently slow speed of operation. Dry Excavation (High) - Completely isolates the excavation process from the water column.
22	Control Contaminant Release - the inherent ability to control sediment resuspension and dissolved and volatile releases for the given equipment type and associated operation. Clamshell (Low) - Can be operated such that the excavation and water column exposure of the bucket is within a silt curtain containment or enclosure; however, high suspended solids within the silt curtain may be released when the curtain is moved. Enclosed Bucket/Articulated Mechanical (Medium) - can be operated such that the excavation and water column exposure of the bucket is within a silt curtain enclosure with relatively small footprint. Enclosed buckets act as a control and greatly reduce resuspension within the enclosures and potential for release. Cutterhead/Plain Suction/Horizontal Auger/Pneumatic/Specialty Dredgeheads (Medium) - Capable of transporting the material directly by pipeline, minimizing exposure to the water column and to volatilization. Can be operated within enclosures, but the footprint of such enclosures would be necessarily larger than that for mechanical dredges. Diver assisted (High) - scale of diver-assisted dredging would seldom require contaminant release controls. Dry Excavation (High) - Dewatering of the dredging area effectively eliminates dissolved releases. Sediment surface exposed to the atmosphere has lower volatile emission rates as compared to the same surface ponded with elevated suspended sediment concentrations.

Highlight 6-7b: Footnotes for Sample Environmental Dredging Operational Characteristics and Selection Factors	
23	Minimize Residual Sediment - efficiency of the dredge is in removing material without leaving a residual, and potentially meeting a cleanup level. Clamshell (Low) - High potential to leave residual sediment because of the circular-shaped cutting action and the tendency to leave a cratered bottom subject to sloughing. Enclosed Bucket/Articulated Mechanical/Cutterhead/Horizontal Auger/Plain Suction/Pneumatic/Specialty Dredgeheads (Medium) - All dredges with active dredgeheads and/or movement in contact with the bottom sediment will leave some residual sediment. The control offered by the articulated arm provides an advantage for removal of thin residual layers. Diver Assisted (High) - Hand-held action of diver-assisted work has a low potential for generating residual sediment. Dry Excavation (High) - Any fallback of sediment excavated under dry conditions can be readily observed and managed.
24	Transport by Pipeline - compatibility of the dredge with subsequent transport by pipeline. Clamshell/ Enclosed Bucket/Articulated Mechanical (Medium) - All mechanical dredges remove material at near in-situ density, and additional reslurry and rehandling equipment must be employed to allow for pipeline transport. Cutterhead/Plain Suction/Horizontal Auger/Pneumatic/Specialty Dredgeheads/Diver Assisted (High) - All hydraulic and pneumatic dredges are designed for pipeline transport. Dry Excavation (Medium) - Additional reslurry and rehandling equipment must be employed to allow for pipeline transport.
25	Transport y Barge - compatibility of the dredge with subsequent transport by barge. Clamshell/Enclosed Bucket/Articulated Mechanical (High) - Material excavated with mechanical dredges is close to in-situ density and may be directly placed in barges for transport. Cutterhead/Plain Suction/Horizontal Auger/Pneumatic/Specialty Dredgeheads/Diver Assisted (Medium) - Barge transport of hydraulically dredged material is inefficient. Although pneumatic and some specialty dredges are capable of removing soft sediment at high water content, intermittent operation for change-out of barges will significantly reduce efficiency. Dry Excavation (High) - Material excavated in the dry may be placed directly in barges using conveyers or front-end loaders.
26	Positioning Control in Currents/Wind/Tides - ability of the dredge to hold a desired position of the dredgehead horizontally with current, wind, or vertically with fluctuating tides. Clamshell/Enclosed Bucket/Articulated Mechanical (High) - Operate with spuds or jack-up piles and are inherently stable against movement by normal winds and currents. Cutterhead/Plain Suction/Specialty Dredgeheads (High) - Equipped with spuds and use □walking spud□ method of operation inherently stable against movement by normal winds and current. Horizontal Auger (Medium) - Free floating and operate using an anchor and cable system, subject to movement with longer anchor sets. Pneumatic (High) - Operate from spudded barges or platforms and are inherently stable against movement by normal winds and currents. Diver Assisted (Medium) - Ability of divers to maintain a desired position will be hampered by currents. Dry Excavation (High) - Not affected by wind and currents.
27	Maneuverability - ability of the dredge to operate effectively in close proximity or around utilities and other infrastructure, narrow channel widths, surface and submerged obstructions, and overhead restrictions. Clamshell/Enclosed Bucket/Articulated Mechanical (High) - Buckets are wire supported or fixed-arm articulated and may be operated close in to infrastructure and within tightly restricted areas. Cutterhead/Plain Suction/Horizontal Auger/Pneumatic/Specialty Dredgeheads (Low) - Swinging action of the walking spud method of operation for hydraulic pipeline dredges and the need for long anchor and cable setup for horizontal auger dredges limits their ability to operate near infrastructure or within tightly restricted areas. Diver Assisted (High) - Can be conducted close to infrastructure and within tightly restricted areas. Dry Excavation (High) - Containments for dry excavation can be designed for areas near infrastructure and tightly restricted areas may be completely contained.
28	Portability/Access - ability of the dredge to pass under bridges, through narrow channels, or to be transported by truck and easily launched to the site. Clamshell/Enclosed Bucket/Articulated Mechanical/Cutterhead/Plain suction/Horizontal Auger/Pneumatic/Diver Assisted/Dry Excavation (High) - Dredge types considered here are the smaller size and are generally truck transportable. Specialty Dredgeheads (Medium) - Some specialty dredge designs are too large for truck transport.
29	Availability - this factor refers to the potential availability of dredges types to contractors and the potential physical presence of the equipment in the U.S. Clamshell/Enclosed Bucket/Articulated Mechanical/Cutterhead/Plain Suction/Horizontal Auger/Pneumatic/Diver Assisted/Dry Excavation (High) - Most dredge types are readily available. Specialty Dredgeheads (Medium) - Some specialty dredges are available through only one contractor or may be subject to restrictions under the Jones Act.

Highlight 6-7b: Footnotes for Sample Environmental Dredging Operational Characteristics and Selection Factors	
30	Debris/Loose Rock/Vegetation - susceptibility of a given dredge type to clogging by debris and subsequent loss of operational efficiency. Clamshell/Enclosed Bucket/Articulated Mechanical (High) - Mechanical dredges can effectively remove sediment containing debris, although leakage may result. Mechanical equipment is the only approach for debris-removal passes. Cutterhead/Plain Suction/Horizontal Auger/ Pneumatic/ Specialty Dredgeheads (Low) - Subject to clogging by debris and are incapable of removing larger pieces of loose rock and larger debris. Loose rock and large debris can also cause inefficient sediment removal. Diver Assisted (Low) - Presence of logs and large debris may present dangerous conditions for diver-assisted dredging. Although divers can remove sediment from around large debris or rocks, this type of operation would be inefficient. Dry Excavation (High) - Dry excavation allows use of conventional excavation equipment. Leakage from buckets caused by debris is not a consideration for dry excavation.
31	Hardpan/Rock Bottom - ability of a dredge type to remove a sediment layer overlying hardpan or rock bottom effciently without leaving excessive residual sediment. Clamshell/Enclosed Bucket/Articulated Mechanical/Cutterhead/Horizontal Auger (Low) - Closing action of buckets and cutting action of dredgeheads result in problems maintaining a desired vertical cutting position and would tend to leave behind excessive residual sediment. Power associated with articulated mechanical has advantage in removing hard materials. Plain Suction/ Pneumatic/ Specialty Dredges (Medium) - Lack an active closing or cutting action and can operate over an uneven hard surface, although removal efficiency may be low. Diver Assisted (High) - May be the most effective approach for precise cleanup of a hard face, since the divers can feel the surface and adjust the excavation accordingly. Dry Excavation (High) - Allows the visual location of pockets of residual remaining on an uneven hard surface.
32	Flexibility for Varying Conditions - flexibility of a given dredge type in adapting to differing conditions, such as sediment stiffness, variable cut thicknesses, and the overall ability to take thick cuts. Clamshell/Enclosed Bucket (High) - Buckets are capable of taking thin cuts or thicker cuts in proportion to the bucket size, and bucket sizes can be easily switched. Articulated Mechanical (Medium) - Ability to change bucket sizes for articulated mechanical is limited. Cutterhead (High) - Capable of taking variable cut thicknesses by varying the burial depth of the cutter. Different cutterhead sizes or designs can be used to adapt to changing cut thicknesses or sediment stiffness. Horizontal Auger (Medium) - Designed for a set maximum cut thickness, and attempts to remove thick cuts may result in plowing actions with excessive resuspension and residual. Plain Suction/ Pneumatic (Low) - No cutting action limits ability to take thicker cuts or remove stiffer materials. Specialty Dredgeheads (Low) - Specialty dredges are designed for a specific application and have limited flexibility. Diver Assisted (Low) - Removal is limited to thin cuts. Dry Excavation (High) - Allows use of a full range of conventional excavation equipment.
33	Thin Lift/Residual Removal - ability of a given dredge type to removal thin layers of contaminated material without excessive over dredging. Clamshell (Low) - Circular shaped cut not suited for efficient removal of thin layers. Enclosed Bucket/Articulated Mechanical (Medium) - Level cutting action is capable of removing thin layers, but the buckets would be only partially filled, resulting in inefficient production and higher handling and treatment costs. Cutterhead/Horizontal Auger (Medium) - Capable of removing thin layers, but the percent solids is reduced under these conditions. Plain Suction/Pneumatic (High) - Well suited for removal of thin lifts, especially loose material such as residual sediment. Specialty Dredgeheads (High) - Some specialty dredges are designed specifically for removal of thin lifts. Diver Assisted (High) - Precision of diver-assisted dredging is well suited for removal of thin layers, especially residuals. Dry Excavation (High) - Allows for a precise control of cut thickness, amenable to removal of thin layers.
Source: Palermo et al. 2004	

6.5.4 Dredge Positioning

An important element of sediment remediation is the precision of the dredge cut, both horizontally and vertically. Technological developments in surveying (vessel) and positioning (dredgehead) instruments have improved the dredging process. Vertical control may be particularly important when contamination occurs in a relatively thin or uneven layer to avoid an unnecessary amount of over-dredging and excess handling of uncontaminated sediment. Video cameras are sometimes useful in monitoring dredging operations, although turbidity effects and lack of spatial references may present limitations on their use. The working depth of the dredgehead may be measured using acoustic instrumentation and by monitoring dredged slurry densities. In addition, surveying software may be used to generate pre- and post-dredging bathymetric charts, determine the volume of dredged sediment, locate

obstacles, and calculate linear dimensions of surface areas (see, e.g., St. Lawrence Centre 1993). Also available are digital positioning systems that enable dredge operators to follow a complex sediment contour (see, e.g., Van Oostrum 1992).

Depending on site conditions (e.g., currents, winds, tides), the horizontal position of the dredge may need to be continuously monitored during dredging. Satellite- or transmitter-based positioning systems, such as differential global positioning systems (DGPS), can be used to define the dredge position. In some cases, however, the accuracy of these systems is inadequate for precise dredging control. Where the accuracy of site characterization data or the high cost of disposal warrant very precise control, it is possible to use optical (laser) surveying instruments set up at one or more locations on shore. These techniques, in conjunction with on-vessel instruments and spuds (if water depths are less than about 50 ft) and anchoring systems may enable the dredge operator to more accurately target specific sediment deposits. The effectiveness of anchoring systems diminishes as water depth increases.

The positioning technology described above enhances the accuracy of dredging. The accuracies achievable for sediment characterization should be considered in setting performance standards for environmental dredging vertical and horizontal operating accuracy (Palermo et al. 2004). However, project managers should not develop unrealistic expectations of dredging accuracy. Contaminated sediment cannot be removed with surgical accuracy even with the most sophisticated equipment. Equipment may not be the only factor affecting the accuracy of the dredging operation. Site conditions (e.g., weather, currents), sediment conditions (e.g., bathymetry, physical characteristics), and the skill of the dredge operator are all important factors. In addition, the distribution of sediment contaminants may be only defined at a crude level and there could be a substantial margin for error. Accurately dredging to pre-established cut-lines is an important component of meeting remedial action objectives for sediment, but alone is not generally sufficient to show that the objectives have been met. Generally, post-dredging sampling should be conducted for that purpose. The section below describes the equally important factors of controlling dredging losses and residual contamination.

6.5.5 Predicting and Minimizing Sediment Resuspension and Contaminant Release and Transport During Dredging

Sediment resuspension and the resulting unwanted contaminant release and transport in the water body arise due to a variety of activities associated with a dredging remedy. These frequently include resuspension caused by operation of the dredgehead, by operation of work boats and tug boats, and by deployment and movement of control measures such as silt screens or sheet piles. Contaminated sediment may also be lost from barges used during the dredging operation. In environments with significant water movement due to tides or currents, resuspended sediment may be transported away from a dredging site; therefore, limiting resuspension or increasing containment (so that resuspended sediment is later redeposited and dredged) can be an important consideration in remedy selection and design. Storm events may also result in transport of contaminants beyond the dredging area. Use of containment barriers to limit transport of resuspended contaminated sediment is discussed in Section 6.5.6 of this chapter.

When evaluating resuspension due to dredging, it generally is important to compare the degree of resuspension to the natural sediment resuspension that would continue to occur if the contaminated sediment was not dredged, and the length of time over which increased dredging-related suspension would occur. Typically, two types of contaminant release are associated with resuspended sediment:

particulate and dissolved. Particulate release refers to the transport of contaminants associated with the particle phase (i.e., sorbed to suspended sediment). Dissolved refers to the release of dissolved contaminants from the particles into the water column. This latter form of release can be significant because dissolved contaminants are the most readily bioavailable and are more easily transported away from the site. Consequently, resuspension can result in the release of bioavailable organic and inorganic contaminants into the water column, which may cause toxicity or enhanced bioaccumulation. Research is currently being performed to address the risk associated with resuspension at contaminated sites and some existing models have been developed by the USACE. Until further guidance is available, at most sites, the project manager should monitor resuspension during dredging and to evaluate its potential effects on water quality. Project managers should be aware that most engineering measures implemented to reduce resuspension also reduce dredging efficiency. Estimates of production rates, cost, and project time frame should take these measures into account.

Some contaminant release and transport during dredging is inevitable and should be factored into the alternatives evaluation and planned for in the remedy design. Releases can be minimized by choice of dredging equipment, dredging less area, and/or using certain operational procedures (e.g., slowing the dredge clamshell descent just before impact with the sediment bed). Generally, the project manager should assess all causes of resuspension and realistically predict likely contaminant releases during a dredging operation. The magnitude of sediment resuspension and resulting transport of contaminants during a dredging operation is influenced by many factors, including:

- Physical properties of the sediment [e.g., grain size distribution, organic carbon content, Acid Volatile Sulfides (AVS) concentration];
- Vertical distribution of contaminants in the sediment;
- Water velocity and degree of turbulence;
- Type of dredge;
- Methods of dredge operation;
- Skill of operators;
- Extent of debris;
- Water salinity; and
- Extent of workboat/tugboat activity.

To compare various remedies for a site, to the extent possible, the project manager should attempt to estimate the downstream mass transport and the degree of increase (if any) in downstream surface water and surface sediment contaminant concentrations. However, at present, no fully verified empirical or predictive tools are available to quantify the predicted releases accurately. As research in predicting resuspension and contaminant release associated with dredging progresses, project managers should watch for verified methods to be developed to assist in this estimate. Although the degree of resuspension will be site specific, recent analyses of field studies and available predictive models of the mass of

sediment resuspended range from generally less than one percent of the mass dredged (Hays and Wu 2001, Palermo and Averett 2003) to between 0.5 and 9 percent (NRC 2001). The methods contained in EPA s *Estimating Contaminant Losses from Components of Remediation Alternatives for Contaminated Sediments* (U.S. EPA 1996g), may be useful to estimate the dredgehead component of resuspension losses. To the extent possible, the project manager should estimate total dredging losses on a site-specific basis and consider them in the comparison of alternatives during the feasibility study.

If conventional clamshell dredges may cause a high level of resuspension, a special purpose dredge may be considered. These dredges generally resuspend less material than conventional dredges, but associated costs may be greater, and dredges may not be usable in the presence of significant debris or obstructions. As in the case of conventional dredges, the selection of a special purpose dredge will be likely dictated by site-specific conditions, economics, and availability (Palermo et al. 1998b). Other factors unrelated to resuspension, such as maneuverability requirements, hydrodynamic conditions, or others listed in Section 6.5.3, Dredge Equipment Selection, may also dictate the type of dredge that should be used. The strategy for the project manager should be to minimize the resuspension levels generated by any specific dredge type, while also ensuring that the project can be implemented in a reasonable time frame. The EPA s Office of Research and Development (ORD) and others are in the process of evaluating resuspension and its effects, both in field and modeling studies. The results of this research should help project managers to understand better and control effects of resuspension during future cleanup actions.

Another potential route of contaminant release during dredging or excavation may be the volatilization of contaminants, either near the dredge or excavation site or in a holding facility like a confined disposal facility (CDF) (Chiarenzeli et al. 1998). At sites with high concentrations of volatile contaminants, dredging or excavation may present special challenges for monitoring and operational controls if they may pose a potential risk to workers and the nearby community. This exposure route may be minimized by reducing dredging production rates so that resuspension is minimized. Covering the surface of the water with a physical barrier or an absorbent compound may also minimize volatilization. At the New Bedford Harbor site, a cutterhead dredge was modified by placing a cover over the dredgehead that retained polychlorinated biphenyl (PCB)-laden oils, thus reducing the air concentrations of PCBs during dredging to background levels; see *Report on the Effects of the Hot Spot Dredging Operations: New Bedford Harbor Superfund Site, New Bedford, MA* (U.S. EPA 1997e and available through EPA s Web site at http://www.epa.gov/region01/nbh/techdocs.html). In addition, the CDF that the dredged sediment was pumped into was fitted with a plastic cover that effectively reduced air emissions. To minimize the potential for volatile releases further, dredging operations were conducted during cooler weather periods and at night. During excavation, volatilization could be of greater concern as contaminated materials may be exposed to air. Care should be taken during dewatering activities to ensure that temperatures are not elevated (e.g., cautious application of lime or cement for dewatering), and other control measure should be taken as needed (e.g., foam).

6.5.6 Containment Barriers

Transport of resuspended contaminated sediment released during dredging can often be reduced by using physical barriers around the dredging operation. Barriers commonly used to reduce the spread of contaminants during the removal process include oil booms, silt curtains, silt screens, sheet-pile walls, cofferdams, and bubble curtains (U.S. EPA 1994d, Francingues 2003). Under favorable site conditions, these barriers help limit the areal extent of particle-bound contaminant migration resulting from dredging

resuspension and enhance the long-term benefits gained by the removal process. Conversely, because the barriers contain resuspended sediment, they may increase, at least temporarily, residual contaminant concentrations inside the barrier compared to what it would have been without the barriers.

Structural barriers, such as sheet pile walls, have been used for sediment excavation and in some cases (e.g., high current velocities) for dredging projects. The determination of whether these types of barriers are necessary should be made based on a thorough evaluation of the site. This can be accomplished by evaluating the relative risks posed by the anticipated release of contaminants from the dredging operation absent use of such structural barriers, the predicted extent and duration of such releases, and the potential for trapping and accumulating residual contaminated sediment within the barrier. The project manager should consult the ARCS program s *Risk Assessment and Modeling Overview Document* (U.S. EPA 1993c) and *Estimating Contaminant Losses from Components of Remediation Alternatives for Contaminated Sediment* (U.S. EPA 1996e) for further information about evaluating the need for structural barriers.

Sheet pile containment structures are more likely to provide reliable containment of resuspended sediment than silt screens or curtains, although at significantly higher cost and with different technological limitations. Where water is removed on one side of the wall, project managers should be aware of the hydraulic loading effects of water level variations inside and outside of these walls. Project managers should also be aware of the increased potential for scour to occur around the outside of the containment area, and the resuspension that will occur during placement and removal of these structures. In addition, use of sheet piling may significantly change the carrying capacity of a stream or river and make it temporarily more susceptible to flooding.

Oil booms are appropriate for sediment that may likely release oils or floatables [i.e., light non-aqueous-phase liquids (LNAPL)] when disturbed. Such booms typically consist of a series of synthetic foam floats encased in fabric and connected with a cable or chains. Oil booms may be supplemented with oil absorbent materials, such as polypropylene mats (U.S. EPA 1994d). However, booms do not aid in retaining the soluble portion of floatables [i.e., polycyclic aromatic hydrocarbons (PAHs) from oils].

Silt curtains and silt screens are flexible barriers that hang down from the water surface. Both systems use a series of floats on the surface and a ballast chain or anchors along the bottom. Although the terms silt curtain and silt screen may be frequently used interchangeably, there are fundamental differences. Silt curtains are made of impervious materials, such as coated nylon, and primarily redirect flow around the dredging area. In contrast, silt screens are made from synthetic geotextile fabrics, which allow water to flow through, but retain a large fraction of the suspended solids (Averett et al. 1990). Silt curtains or silt screens may be appropriate when site conditions dictate the need for minimal transport of suspended sediment, for example, when dredging hot spots of high contaminant concentration.

Silt curtains have been used at many locations with varying degrees of success. For example, silt curtains were found to be effective in limiting suspended solids transport during in-water dike construction of the CDF for the New Bedford Harbor pilot project. However, the same silt curtains were ineffective in limiting contaminant migration during dredging operations at the same site primarily as a result of tidal fluctuation and wind (Averett et al. 1990). Problems were experienced during installation of silt curtains at the General Motors site (Massena, New York) due to high current velocities and back eddies. Dye tests conducted after installation revealed significant leakage, and the silt curtains were removed. Sheet piling was then installed around the area to be dredged with silt curtains used as

supplemental containment for hot spot areas. A silt curtain and silt screen containment system were effectively applied during dredging of the Sheboygan River in 1990 and 1991, where water depths were 2 m or less. A silt curtain was found to reduce suspended solids from approximately 400 mg/L (inside) to 5 mg/L (outside) during rock fill and dredging activities in Halifax Harbor, Canada (MacKnight 1992). At some sites, changes in dredging operating procedures may offer more effective control of resuspension than containment barriers.

The effectiveness of silt curtains and screens is primarily determined by the hydrodynamic conditions at the site. Conditions that may reduce the effectiveness of these and other types of barriers include the following:

- Significant currents;
- High winds;
- Changing water levels (i.e., tidal fluctuation);
- Excessive wave height, including ship wakes; and
- Drifting ice and debris.

Silt curtains and screens are generally most effective in relatively shallow, undisturbed water. As water depth increases and turbulence caused by currents and waves increases, it becomes difficult to isolate the dredging operation effectively from the ambient water. The St. Lawrence Centre (1993) advises against the use of silt curtains in water deeper than 6.5 m or in currents greater than 50 cm/sec.

The effectiveness of containment barriers is also influenced by the quantity and type of suspended solids, the mooring method, and the characteristics of the barrier. To be effective, barriers should be deployed around the dredging operation and remain in place until the operation is completed, although it may need to be opened to allow transport of barges in and out of the dredge site, which may release some resuspended contaminants. For large projects, it may be necessary to relocate the barriers as the dredge moves to new areas. Where possible, barriers should not impede navigation traffic. Containment barriers may also be used to protect specific areas, for example, valuable habitat, water intakes, or recreational areas, from suspended sediment contamination.

6.5.7 Predicting and Minimizing Dredging Residuals

All dredging operations leave behind some residual contamination in sediment, usually both within the dredged area and spread to adjacent areas. This residual contaminated sediment is often soft, unconsolidated, has a high water content, and may exist, at least temporarily, as a fluid mud or nephloid layer. The primary sources of the dredging residuals typically include: 1) contaminated sediment below the dredge line that was not removed, 2) sediment loosened by the dredge head or bucket, but not captured and removed, 3) sediment on steep slopes that fall into the dredged area, and 4) resettling of sediment from the dredging operation. Similar to resuspension releases discussed in Section 6.5.5, the extent of the residual contamination is dependent on a number of factors including:

- Skill of operator and type and size of dredging equipment;

- Steepness of dredge cut slopes;
- Amount of contaminated sediment resuspended by the dredging operation;
- Extent of controls on dispersion of resuspended sediment (e.g., silt curtains, sheet piling);
- Vertical profile of contaminant concentrations in sediment relative to the thickness of sediment to be removed;
- Contaminant concentrations in surrounding undredged areas;
- Characteristics of underlying sediment or bedrock (e.g., whether over-dredging is feasible); and
- Extent of debris, obstructions, or confined operating area (e.g., which may limit effectiveness of dredge operation).

Project managers should factor a realistic estimate of dredging residuals into their evaluation of alternatives. Field results for some completed environmental dredging pilots and projects suggest that average post-dredging residual contamination levels have not met desired cleanup levels. However, aside from past experience, there is no commonly accepted method to predict accurately the degree of residual contamination likely to result from different dredge types under given site conditions. Additional guidelines are needed in this area and are likely to be developed in the future. Some preliminary research has shown that the residual concentration may be expected to be similar to the average contaminant concentration within the dredging prism (Desrosiers et al. 2005). In situations where more highly contaminated sediment is removed in a first dredging pass and deeper lower-level contamination is removed in a second dredging pass, lower residuals may be attainable. If the buried sediment is significantly more contaminated than the near-surface sediments, and if over dredging into clean sediment is not accomplished or feasible, the residual concentration may be greater than the average baseline surface concentration although significant contaminant mass may have been removed. When comparing alternatives and selecting of the best risk reduction alternative for the site, project managers should consider whether conditions are favorable for achieving desired post-dredging residual concentrations.

In cases where residuals may cause an unacceptable risk, additional passes of the dredge may be needed to achieve the desired results. Placement of a thin layer (e.g., 6–24 in) of clean material designed to mix with underlying sediment or the addition of reactive/sorptive materials to surface sediment can also be used to reduce the residual contamination. Project managers should consider developing a contingency remedy if there is sufficient uncertainty concerning the ability to achieve low cleanup levels. Where a contingency remedy involves containment of residuals by in-situ capping, project managers should consider whether containment without dredging may be a more appropriate solution to manage long-term risks in that area.

It is generally important to conduct post-dredging sampling to confirm residual contamination levels. If resuspension and transport is expected, generally, it is also important to sample outside of the

dredged area to assess contaminant levels to which biota will be exposed from these areas. These data are often needed to assess the likelihood of achieving all RAOs.

6.6 TRANSPORT, STAGING, AND DEWATERING

After removal, sediment often is transported to a staging or rehandling area for dewatering (if necessary), and further processing, treatment, or final disposal. Transport links all dredging or excavation components and may involve several different modes of transport. The first element in the transport process is to move sediment from the removal site to the disposal, staging, or rehandling site. Sediment may then be transported for pretreatment, treatment, and/or ultimate disposal (U.S. EPA 1994d). As noted previously, where possible, project managers should design for as few rehandling operations as possible to decrease risks and cost. Project managers should also consider community concerns regarding these operations (e.g., odor, noise, lighting, traffic, and other issues). Health and safety plans should address both workers and community members.

Modes of transportation may include one or more of the following waterborne or overland methods:

- *Pipeline:* Direct placement of material into disposal sites by pipeline is economical only when the disposal and/or treatment site is located near the dredging areas (typically a few kilometers or less, unless booster pumps are used). Mechanically dredged material may also be reslurried from barges and pumped into nearshore disposal sites by pipeline;

- *Barge:* A rehandling facility located on shore is a commonly considered option. With a rehandling facility, dredging can be accomplished with mechanical (bucket) dredges where the sediment is excavated at near in-situ density (water content) and placed in a barge or scow for transport to the rehandling facility;

- *Conveyor:* Conveyors may be used to move material relatively short distances. Materials should be in a dewatered condition for transport by conveyor;

- *Railcar:* Rail spurs may be constructed to link rehandling/treatment facilities to the rail network. Many licensed landfills have rail links, so long-distance transport by rail is generally an option; and/or

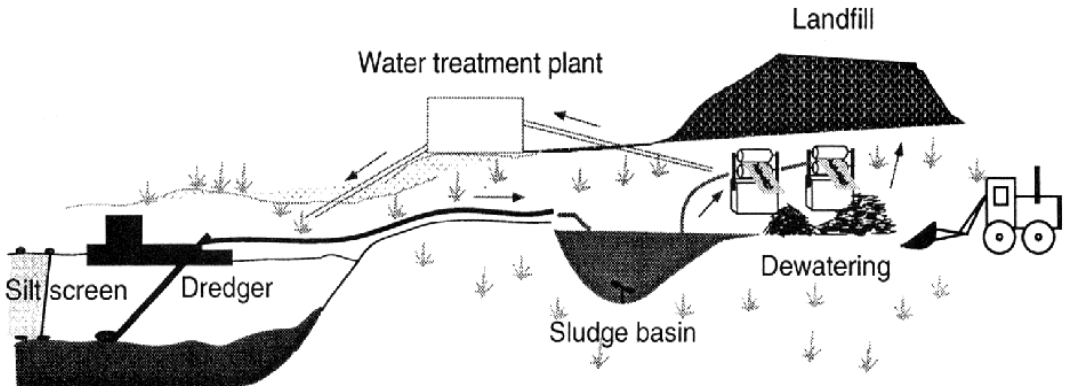
- *Truck/Trailer:* Dredged material can be rehandled directly from the barges to roll-off containers or dump trucks for transport to a CDF by direct dumping or unloading into a chute or conveyor. Truck transport of treated material to landfills may also be considered. The material should be dewatered prior to truck transport over surface streets. In some smaller sites where construction of dewatering beds may be difficult or the cost of disposal is not great, addition of non-toxic absorbent materials such as lime or cement may be feasible.

A wide variety of transportation methods are available for moving sediment and residual wastes with unique physical and chemical attributes. In many cases, contaminated sediment is initially moved using waterborne transportation. Exceptions are the use of land-based or dry excavation methods. Project managers should consider the compatibility of the dredge with the subsequent transport of the

dredged sediment. For example, hydraulic and pneumatic dredges produce contaminated dredged-material slurries that can be transported by pipeline to either a disposal or rehandling site. Mechanical removal methods typically produce dense, contaminated material hauled by barge, railcar, truck/trailer, or conveyor systems. The feasibility, costs of transportation, and need for additional equipment are frequently influenced by the scale of the remediation project (Churchward et al. 1981, Turner 1984, U.S. EPA 1994f).

Temporary storage of contaminated sediment may also be necessary in order to dewater it prior to upland disposal or to allow for pretreatment and equalization prior to treatment. For example, a temporary CDF may be designed to store dredged material for periods when dredging or excavation is not possible due to weather or environmental concerns, while the treatment process may continue on a near 24-hour operating schedule. Storage may be temporary staging (e.g., pumping onto a barge with frequent off-loading) or more permanent disposal (e.g., moving the sediment to a land-based CDF where it may be dewatered and treated). A typical dewatering schematic is shown in Highlight 6-8.

Highlight 6-8: Sample of Dredging Dewatering Process



Depending upon the quality of the water after it is separated from sediment and upon applicable or relevant and appropriate requirements (ARARs), it may be necessary to treat water prior to discharge. Where water treatment is required, it can be a costly segment of the dredging project and should be included in cost estimates for the alternative. Water treatment costs may also affect choices regarding dredging operation and equipment selection, as both can affect the amount of water entrained.

The project manager should consider potential contaminant losses to the water column and atmosphere during transport, dewatering, temporary storage, or treatment. For example, conventional mechanical dredging methods and equipment often rely on gravity dewatering of the sediment on a dredge scow, with drainage water and associated solids flowing into the surrounding water. Project managers should evaluate what engineering controls are necessary and cost-effective, and include these controls in planning and design. Implementation risks, both to workers and to the community, differ significantly between the various transport methods listed above. These risks should be evaluated and included when comparing alternatives. Best management practices for protection of water quality should also be followed.

The risks associated with a temporary storage or staging sites are similar to those associated with CDFs, as discussed in Section 6.8.2, Sediment Disposal. In particular, in-water temporary CDFs can prove to be attractive nuisances, especially to waterfowl, by providing attractive habitat that encourages use of the CDF by wildlife and presenting the opportunity for exposure to contaminants. For highly contaminated sites, it may be necessary to provide a temporary cover or sequence dredging to allow for coverage of highly contaminated sediment with cleaner sediment to minimize short-term exposures. This method of control has proven effective for minimizing exposures at upland sanitary landfills. In addition, because some holding areas may not be designed for long-term storage of contaminated sediment, the risk of contaminant transport to ground water may need to be evaluated and monitored.

6.7 SEDIMENT TREATMENT

For the majority of sediment removed from Superfund sites, treatment is not conducted prior to disposal, generally because sediment sites often have widespread low-level contamination, which the NCP acknowledges is more difficult to treat. However, pretreatment, such as particle size separation to distinguish between hazardous and non-hazardous waste disposal options, is common. Although the NCP provides a preference for treatment for principal threat waste, treatment has not been frequently selected for sediment. High cost, uncertain effectiveness, and/or (for on-site operations) community preferences are other factors that lead to treatment being selected infrequently at sediment sites. However, treatment of sediment could be the best option in some circumstances and innovations in ex-situ or in-situ treatment technologies may make treatment a more viable cost-effective option in the future.

The treatment of contaminated sediment is not usually a single process, but often involves a combination of processes or a treatment train to address various contaminant problems, including pretreatment, operational treatment, and/or effluent treatment/residual handling. Some form of pretreatment and effluent treatment/residual handling are necessary at almost all sediment removal projects. Sediment treatment processes of a wide variety of types have been applied in pilot-scale demonstrations, and some have been applied full scale. However, the relatively high cost of most treatment alternatives, especially those involving thermal and chemical destruction techniques, can be a major constraint on their use (NRC 1997). The base of experience for treatment of contaminated sediment is still limited. Each component of a potential treatment train is discussed in the next section.

6.7.1 Pretreatment

Pretreatment modifies the dredged or excavated material in preparation for final treatment or disposal. When pretreatment is part of a treatment train, distinguishing between the two components may be difficult and is not always necessary. Pretreatment is generally performed to condition the material to meet the chemical and physical requirements for treatment or disposal; and/or to reduce the volume and/or weight of sediment that requires transport, treatment, or restricted disposal. Pretreatment processes typically include dewatering and physical or size separation technologies.

Most treatment technologies require that the sediment be relatively homogeneous and that physical characteristics be within a relatively narrow range. Pretreatment technologies may be used to modify the physical characteristics of the sediment to meet these requirements. Additionally, some pretreatment technologies may divide sediment into separate fractions, such as organic matter, sand, silt, and clay. Often the sand fractions contain lower contaminant levels and may be suitable for unrestricted disposal and/or beneficial use if it meets applicable standards and regulations. Selection factors, costs,

pilot-scale demonstrations, and applicability of specific pretreatment technologies are discussed in detail in EPA s *Assessment and Remediation of Contaminated Sediments (ARCS) Program Remediation Guidance Document* (U.S. EPA 1994d).

6.7.2 Treatment

Depending on the contaminants, their concentrations, and the composition of the sediment treatment of the sediment to reduce the toxicity, mobility, or volume of the contaminants before disposal may be warranted. Available disposal options and capacities may also affect the decision to treat some sediment. In general, treatment processes have the ability to reduce sediment contaminant concentrations, mobility, and/or sediment toxicity by contaminant destruction or by detoxification, by extraction of contaminants from sediment, by reduction of sediment volume, or by sediment solidification/stabilization.

Treatment technologies for sediment are generally classified as biological, chemical, extraction or washing, immobilization (solidification/stabilization), and thermal (destruction or desorption). In some cases, particle size separation is also considered a treatment technology. The following treatment technologies are among those which might be evaluated.

Bioremediation

Generally, bioremediation is the process in which microbiological processes are used to degrade or transform contaminants to less toxic or nontoxic forms. In recent years, it has been demonstrated as a technology for destroying some organic compounds in sediment. The project manager should refer to EPA (1994d), Myers and Bowman (1999), and Myers and Williford (2000) for a summarization of bioremediation technologies and their application under site-specific conditions.

Chemical Treatment

Generally, chemical treatment refers to processes in which chemical reagents are added to the dredged or excavated material for the purpose of contaminant destruction. Contaminants may be destroyed completely, or may be altered to a less toxic form. Averett and colleagues (1990) reviewed several general categories of chemical treatment. Of the categories reviewed, treatments including chelation, dechlorination, and oxidation (of organic compounds) were considered most promising.

Extraction/Washing

Generally, the primary application of extraction processes is to remove organic and, in some cases, metal contaminants from the sediment particles. Sediment washing is another term used to describe extraction processes, primarily when water may be a component of the solvent. In the extraction process, dredged or excavated material is slurried with a chemical solvent and cycled through a separator unit. The separator divides the slurry into the three following fractions: 1) particulate solids; 2) water; and 3) concentrated organic contaminants. The concentrated organics are removed from the separator for post-process treatment. Extraction or washing may also generate large volumes of contaminated wastewater that generally must be treated prior to discharge.

Immobilization or Solidification/Stabilization

Generally, immobilization, commonly referred to as solidification/stabilization, alters the physical and/or chemical characteristics of the sediment through the addition of binders, including cements and pozzolans (U.S. EPA 1994d). Immobilization technologies primarily work by changing the properties of the sediment so contaminants are less prone to leaching. Alteration of the physical character of the sediment to form a solid material, such as a cement matrix, reduces the accessibility of the contaminants to water and entraps the contaminated solids in a stable matrix (Myers and Zappi 1989). Another form of immobilization, chemical stabilization, minimizes the solubility of metals primarily through the control of pH and alkalinity. Chemical stabilization of organic compounds may also be possible (Barth et al. 2001, Wiles and Barth 1992, Myers and Zappi 1989, Zimmerman et al. 2004).

Thermal Treatment

Generally, thermal technologies include incineration, pyrolysis, thermal desorption, sintering, and other processes that require heating the sediment to hundreds or thousands of degrees above ambient temperatures. Thermal destruction processes, such as incineration, are generally effective for destroying organic contaminants but are also expensive and have significant energy costs. Generally, thermal treatment does not destroy toxic metals.

Particle Size Separation

Generally, particle size separation involves separation of the fine material from the coarse material by physical screening. A site demonstration of the Bergman USA process resulted in the successful separation of less than 45 micron fines from washed coarse material and a humic fraction (U.S. EPA 1994f). As previously noted, particle size separation may serve as a pretreatment step prior to implementation of a treatment alternative. Many treatment processes require particle sizes of one centimeter or less for optimal operation.

Effluent Treatment/Residue Handling

Generally, treatment of process effluents means treatment of liquid, gas, or solid residues and is a major consideration during selection, design, and implementation of dredging or excavation. As shown in Highlight 6-1, dredging or excavation may require management of several types of residual wastes from the pretreatment and operational treatment processes that include liquid and/or air/gas effluents from dewatering or other pretreatment/treatment processes, residual solids, and runoff/discharges from active CDFs. Generally, these wastes can be handled through the use of conventional technologies for water, air, and solids treatment and disposal. However, the technical, cost, and regulatory requirements can be important considerations during the evaluation of dredging or excavation as a cleanup method.

Pilot and full-scale treatment processes have been conducted at a number of sites, although there is limited experience at Superfund sites. Where treatment has been used at Superfund sites, the most common treatment method is immobilization by solidification or stabilization. Additional information concerning treatment technologies for contaminated sediment may be found in U.S. EPA Office of Water s *Selecting Remediation Technologies for Contaminated Sediment* (U.S. EPA 1993d). Specific applications, limitations, specifications, and efficiencies of many sediment treatment processes are discussed in the ARCS program s *Remediation Guidance Document* (U.S. EPA 1994d). The NY/NJ

Harbor Project is an example of a large-scale demonstration of several dredged decontamination technologies (Highlight 6-9).

Highlight 6-9: NY NJ Harbor - An Example of Treatment Technologies and Beneficial Use

The goal of the NY/NJ Harbor Sediment Decontamination Project is to assemble a complete decontamination system for cost effective transformation of dredged material (mostly from navigational dredging projects) into an environmentally safe material that can be used in the manufacturing of a variety of beneficial use products.

The following four treatment technologies are being used at the NY/NJ site: 1) sediment washing; 2) thermal treatment; 3) solidification; and 4) vitrification. Each technology has a sponsor from the private sector that will provide the capital needed for facility construction and operation.

Sediment washing (extraction) uses high-pressure water jets and proprietary chemical additives to extract both organic and inorganic contaminants from the sediment. The resulting materials can be used to produce manufactured soil for commercial, and in some cases, residential landscaping applications. Advantages to this treatment include modest capital costs and high throughput. The patented washing system has been demonstrated capable of decontaminating sediments containing high quantities of silt and clay.

A thermal treatment being used is a thermo-chemical manufacturing process that, at high temperatures, will destroy organic contaminants. The process will melt a mixture of sediment and modifiers, and the resulting product is a manufactured grade cement comparable to Portland Cement. This is a very effective treatment, but expensive.

A third process is a "treatment train" that includes dewatering, pelletizing, and transport to an existing light-weight aggregate facility. Pelletizing is a type of solidification treatment. After the sediment is dewatered, it is mixed with shale fines and extruded into pellets. The pellets are fed into a rotary kiln, and the organic matter explodes. The resulting material can be used as a structural component in concrete, insulation (pipeline) and for other geotechnical uses.

Finally, the process includes a high temperature vitrification, which uses an electrical current to heat (melt) and vitrify the soil in place. This process can destroy organic contaminants and incorporate metals into a glassy matrix that can be used to produce an architectural tile.

Source: Stern et al. 2000, Mulligan et al. 2001, Stern 2001, NRC 1997

Potential sediment treatment technologies will evolve as new technologies are developed and other technologies are improved. EPA has recognized the need for an up-to-date list of treatment alternatives and has developed the following databases:

- *EPA Remediation and Characterization Innovative Technologies (EPA REACH IT):* Provides information on more than 750 service providers that offer almost 1,300 remediation technologies and more than 150 characterization technologies (includes a variety of media, not just sediment). More information is available at http://www.epareachit.org/index3.html; and

- *EPA National Risk Management Research Laboratory (NRMRL) Treatability Database:* Provides results of published treatability studies that have passed the EPA quality assurance reviews, it is not specific to sediment, and is available on CD from the EPA's ORD National Risk Management Research Laboratory in Cincinnati, Ohio. Detailed contact information is available at http://www.epa.gov/ORD/NRMRL/treat.htm.

6.7.3 Beneficial Use

Although not normally considered a treatment option, beneficial use may be an appropriate management option for treated or untreated sediment resulting from environmental dredging projects. Significant cost savings may be realized if physical and chemical properties of the sediment allow for beneficial use, especially where disposal options are costly. For example, at Rouge River/Newburgh Lake, Michigan, a Great Lakes Area of Concern, significant cost savings were realized by using lightly contaminated dredged sediment as daily cover at a local sanitary landfill, where it did not pose risk within the landfill boundary. The Bark Camp Mine Reclamation Project in Pennsylvania provides another reuse example. Information is available through the Pennsylvania Department of Environmental Protection Web site at http://www.dep.state.pa.us/dep/DEPUTATE/MINRES/BAMR/bark_camp/barkhomepage.htm. However, beneficial use of dredged or excavated sediment has been only implemented infrequently for remedial projects, mainly due to lack of cost-effective uses in most instances. Where beneficial use is considered, the contaminant levels and environmental exposure, including considerations of future land use, should be assessed.

Options for beneficial use may include the following:

- Construction fill;
- Sanitary landfill cover as in the above example;
- Mined lands restoration;
- Subgrade cap material or subgrade in a restoration fill project (topped with clean sediment or other fill);
- Building materials (e.g., architectural tile; see Highlight 6-9); and
- Beach nourishment (for a clean sand fraction).

A series of technical notes on beneficial uses of contaminated material has been developed by the USACE (Lee 2000), and the USACE maintains a Web site of beneficial use case studies currently available at http://el.erdc.usace.army.mil/dots/budm/budm.html. Use of contaminated materials from CDFs (to include treated material) is a major thrust of the USACE Dredging Operations and Environmental Research (DOER) program (http://el.erdc.usace.army.mil/dots/doer). In addition, Barth and associates evaluated beneficial reuse using an effectiveness protocol (Barth et al. 2001).

In some cases, a CDF (see description in Section 6.8.2) can be integrated with site reuse plans to both reduce environmental risk and simultaneously foster redevelopment in urban areas and brownfields sites. For example, at the Sitcum Waterway cleanup project in Tacoma, Washington, contaminated sediment was placed in a near shore fill in the Milwaukee Waterway, which was then developed into a container terminal. Also, there may be innovative and environmentally protective ways to reuse dredged contaminated sediments in habitat restoration projects (e.g., placement of lightly contaminated material over highly contaminated materials to build up elevations necessary for eventual creation of clean emergent marshlands).

6.8 SEDIMENT DISPOSAL

For purposes of this guidance the term disposal refers to the placement of dredged or excavated material and process wastes into a temporary or permanent structure, site, or facility. The goal of disposal is generally to manage sediment and/or residual wastes to prevent contaminants associated with them from impacting human health and the environment. Disposal is typically a major cost and logistical component of any dredging or excavation alternative. The identification of disposal locations can often be the most controversial component of planning and implementing a dredging remedy and, therefore, should be considered very early in the feasibility study.

Historically, contaminated sediment from Superfund sites has been typically managed in upland sanitary landfills, or hazardous or chemical waste landfills, and less frequently, in CDFs. Contaminated sediment has also been managed by the USACE in contained aquatic disposals (CADs). Also, the material may have a beneficial use in an environment other than the aquatic ecosystem from which it was removed (e.g., foundation material beneath a newly constructed brownfields site), especially if the sediment has undergone treatment. As noted below, all disposal options have the potential to create some risk. These risks may result from routine practices (i.e., worker exposure and physical risks and volatilization), while other risks may result from unintended events, such as transportation accidents and contaminant losses at the disposal site. All potential risks should be considered when comparing alternatives. The ARCS program s *Remediation Guidance Document* (U.S. EPA 1994d) provides a discussion of the available disposal technologies for sediment, including an in-depth discussion of costs, design considerations, and selection factors associated with each technology. Averett and colleagues (1990), EPA (1991b), and Palermo and Averett (2000) provide additional discussion of disposal options and considerations.

6.8.1 Sanitary/Hazardous Waste Landfills

Existing commercial, municipal, or hazardous waste landfills are the most widely used option for disposal of dredged or excavated sediment and pretreatment/treatment residuals from environmental dredging and excavation. Landfills also are sometimes constructed onsite for a specific dredging or excavation project. Landfills can be categorized by the types of wastes they accept and the laws regulating their operation. Most solid waste landfills accept all types of waste (including hazardous substances) not regulated as Resource Conservation and Recovery Act (RCRA) hazardous waste or Toxic Substances Control Act (TSCA) toxic materials. Due to typical restrictions on liquids in landfills, most sediment should be dewatered and/or stabilized/solidified before disposal in a landfill. Temporary placement in a CDF or pretreatment using mechanical equipment may therefore be necessary (Palermo 1995).

6.8.2 Confined Disposal Facilities (CDFs)

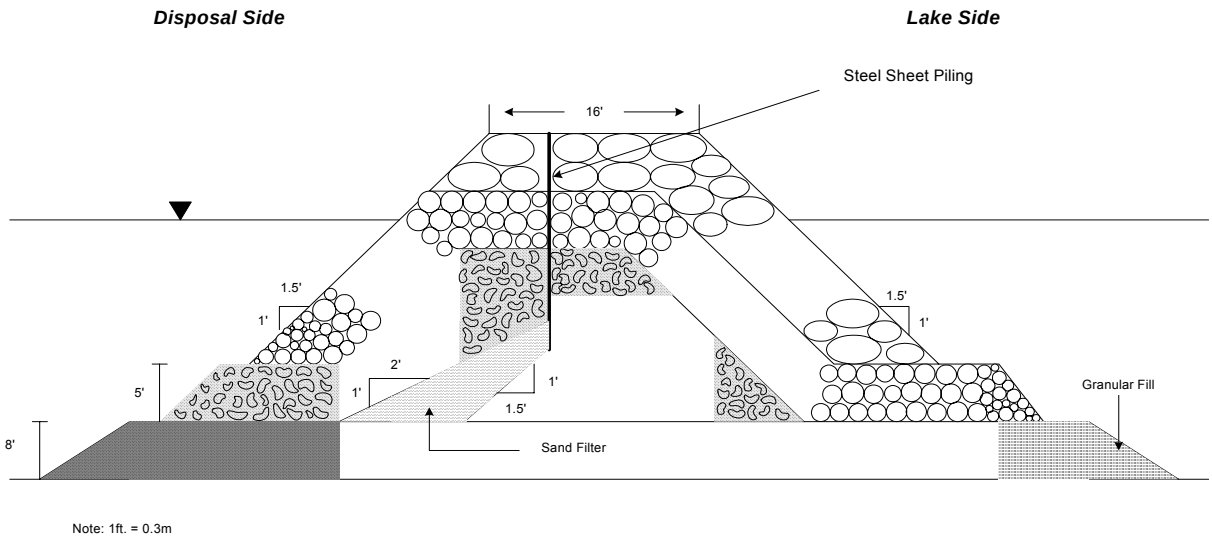
CDFs are engineered structures enclosed by dikes and specifically designed to contain sediment. CDFs have been widely used for navigational dredging projects and some combined navigational/environmental dredging projects but are less common for environmental dredging sites, due in part to siting considerations. However, they have been used to meet the needs of specific sites, as have other innovative in-water fill disposal options, for example, the filling of a previously used navigational waterway or slip to create new container terminal space (e.g., Hylebos Waterway cleanup and Sitcum

Waterway cleanup in Tacoma, Washington). In some cases, new nearshore habitat has also been created as mitigation for the fill.

Under normal operations of a CDF, water is discharged over a weir structure or allowed to migrate through the dike walls while solids are retained within the CDF. Typically effluent guidelines or discharge permits govern the monitoring requirements of the return water. Details regarding the use and engineering design of CDFs are available in the USACE Engineer Manual, *Confined Disposal of Dredged Material* (USACE 1987) and the USACE *Testing Manual* (USACE 2003).

A cross-sectional view of a typical nearshore CDF dike design is shown in Highlight 6-10. CDFs may be located either upland (above the water table), near-shore (partially in the water), or completely in the water (island CDFs). There are several documents available containing thorough descriptions, technical considerations, and costs associated with CDFs (U.S. EPA 1996e, U.S. EPA 1994d, U.S. EPA 1991c, and Averett et al. 1990). Additionally, USACE and EPA (2003) describes a history and evaluation of the design and performance of CDFs used for navigational dredging projects in the Great Lakes Basin, including a review and discussion of relevant contaminant loss and contaminant uptake studies.

Highlight 6-10: Cross Section of a Typical Confined Disposal Facility Dike with a Filter Layer



Note: Adapted from US. EPA 1998d

6.8.3 Contained Aquatic Disposal (CAD)

For purposes of this guidance, contained aquatic disposal is a type of subaqueous capping in which the dredged sediment is placed into a natural or excavated depression elsewhere in the water body. A related form of disposal, known as level bottom capping, places the dredged sediment on a level bottom elsewhere in the water body, where it is capped. CAD has been used for navigational dredging projects (e.g., Boston Harbor, Providence River), but has been rarely considered for environmental dredging

projects. However, there may be instances when neither dredging with land disposal nor capping contaminated sediment in-situ is feasible, and it may be appropriate to evaluate CADs. The depression used in the case of a CAD should provide lateral containment of the contaminated material, and also should have the advantage of requiring less maintenance and being more resistant to erosion than level-bottom capping. The depression for the CAD cell may be excavated using conventional dredging equipment or natural or historically dredged depressions may be used. Uncontaminated material excavated from the depression may be subsequently used for the cap (U.S. EPA 1994d).

6.8.4 Losses from Disposal Facilities

Evaluation of a new on-site disposal facility for placement of contaminated sediment should include an assessment of contaminant migration pathways and should incorporate management controls in the facility design as needed. Landfill disposal options may have short-term releases, which include spillages during transport and volatilization to the atmosphere as the sediment is drying. As for any disposal option, longer-term releases depend in large part on the characteristics of the contaminants and the design and maintenance of the disposal facility.

For CDFs, contaminants may be lost via effluent during filling operations, surface runoff due to precipitation, seepage through the bottom and the dike wall, volatilization to the air, and uptake by plants and animals. The USACE has developed a suite of testing protocols for evaluating each of these pathways (U.S. EPA and USACE 1992), and these procedures are included in the ARCS program s *Estimating Contaminant Losses from Components of Remediation Alternatives for Contaminated Sediments* (U.S. EPA 1996e). The USACE has also developed the *Testing Manual* (USACE 2003), which describes contaminant pathway testing. Depending on the likelihood of contaminants leaching from the confined sediment, a variety of dike and bottom linings and cap materials may be used to minimize contaminant loss (U.S. EPA 1991c, U.S. EPA 1994d, Palermo and Averett 2000). Depending on contaminant characteristics, CDFs for sediment remediation projects may need control measures such as bottom or sidewall liners or low permeability dike cores. Project managers should also be aware that permeability across these barriers can decline significantly with time due to the consolidation process and blockage of pore spaces with fine materials. Therefore, site-specific evaluation is important.

Contaminants may be released as a mud wave outside of the boundaries of the CAD, or to the water column or air during placement of the contaminated sediment. Seepage of pore water may also occur during the initial consolidation of the sediment following placement. Other releases common to in-situ caps, such as through erosion of the cap or movement of contaminants through the cap (see Chapter 5, In-Situ Capping) may also occur. Whatever disposal options are evaluated, the rate and potential effects of contaminant losses during construction and in the long term should be considered.

Highlight 6-11 presents some general points to remember from this chapter.

Highlight 6-11: Some Key Points to Remember When Considering Dredging and Excavation

- Source control should be generally implemented to prevent recontamination
- A dredging or excavation alternative should include details concerning all phases of the project, including sediment removal, staging, dewatering, water treatment, sediment transport, and sediment treatment, reuse, or disposal
- Transport and disposal options may be complex and controversial; options should be investigated early and discussed with stakeholders
- In predicting risk reduction effects of dredging or excavation of deeply buried contaminants, exposure and risk are related to contaminants that are accessible to biota. Contaminants that are deeply buried have no significant migration pathway to the surface, and are unlikely to be exposed in the future may not need removal
- Environmental dredging should take advantage of methods of operation, and in some cases specialized equipment, that minimize resuspension of sediment and transport of contaminants. The use of experienced operators and oversight personnel is very important to an effective cleanup
- A site-specific assessment or pilot study of anticipated sediment resuspension, contaminant release and transport, and its potential ecological impacts should be conducted prior to full scale dredging
- Realistic, site-specific predictions should be made of residual contamination based on pilot studies or data from comparable sites. Where residuals are a concern, thin layer placement/backfilling, MNR, or capping may also be needed
- Excavation (conducted after water diversion) often leads to lower levels of residual contamination than dredging (conducted under standing water)
- A dredging or excavation project should be monitored during implementation to assess resuspension and transport of contaminants, immediately after implementation to assess residuals, and after implementation to measure long-term recovery of biota and to test for recontamination

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7.0 REMEDY SELECTION CONSIDERATIONS

> No two sites are identical and therefore the risk-management strategy will vary from site to site... The strategy selected should be one that actually reduces overall risk, not merely transfers the risk to another site or another affected population. The decision process necessary to arrive at an optimal management strategy is complex and likely to involve numerous site-specific considerations...
>
> Management decisions must be made, even when information is imperfect. There are uncertainties associated with every decision that need to be weighed, evaluated, and communicated to affected parties. Imperfect knowledge must not become an excuse for not making a decision.

In these two statements from the National Research Council s (NRC s) report *A Risk Management Strategy for PCB-Contaminated Sediments* (NRC 2001), the NRC identifies some of the key challenges faced by many project managers at the remedy selection stage. The program goal of the Superfund remedy selection process is to select remedies that are protective of human health and the environment, that maintain protection over time, and that minimize untreated waste [Title 40 Code of Federal Regulations (40 CFR) 300.430(a)(1)(i)]. Superfund remedies must also be cost-effective and use permanent solutions to the maximum extent practicable [Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) 121(b)]. The best route to meeting these and other requirements, as well as the best route to overall risk reduction, depends on a large number of site-specific considerations, some of which may be subject to significant uncertainty. Although final decision making in the face of imperfect knowledge may be necessary, it may be appropriate to postpone a final decision if there is significant doubt about the proposed action s ability to reduce site risks substantially in light of the potential magnitude of costs associated with addressing certain sediment sites. Postponing a final decision may provide an opportunity to conduct additional investigation or pilot studies, and would not necessarily preclude carrying out appropriate interim response actions at the same time.

7.1 RISK MANAGEMENT DECISION MAKING

Consistent with the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), each of the risk management principles in the U.S. Environmental Protection Agency s (EPA s) *Principles for Managing Contaminated Sediment Risks at Hazardous Waste Sites* (U.S. EPA 2002a; see Appendix A), is important to consider for achieving a successful sediment cleanup. Several of the principles apply more directly to the remedy selection stage, especially Principle 7, Select Site-Specific, Project-Specific, and Sediment-Specific Risk Management Approaches that will Achieve Risk-based Goals. Any decision regarding the specific choice of a remedy for a contaminated sediment site should be based on a careful consideration of the advantages and limitations of available approaches and a balancing of tradeoffs among alternatives.

A risk management process should be used to select a remedy designed to reduce the key human and ecological risks effectively. Another important risk management function generally is to compare and contrast the costs and benefits of various remedies. As noted in EPA s *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessment* (U.S. EPA 1997d), risk assessments should provide a basis for comparing, ranking, and prioritizing risks. The

results can also be used in cost-effectiveness analyses that offer additional interpretation of the effects of alternative management options.

In addition, risk management goals should be developed that can be evaluated within a realistic time period, acknowledging that it may not be practical to achieve all goals in the short term. Risk management of contaminated sediment should comprehensively evaluate the broad range of risks posed by contaminated sediment and associated remedial actions, while recognizing that some risks may be reduced in a shorter time frame than others.

EPA s *Rules of Thumb for Superfund Remedy Selection* (U.S. EPA 1997c, also referred to as the Rule of Thumb Guidance) is a helpful guidance for project managers to review when making risk-management decisions and selecting remedies at sediment sites. The Rules of Thumb Guidance describes key principles and expectations, interspersed with best practices based on program experience and policies. In addition, this guidance discusses how remedy selection may also be applicable to the Resource Conservation and Recovery Act (RCRA) Corrective Action Program. For more information on the two cleanup programs, the project manager should refer to Office of Solid Waste and Emergency Response (OSWER) Directive 9200.0-25, *Coordination Between RCRA Corrective Action and Closure and CERCLA Site Activities* (U.S. EPA 1996f).

Decisions regarding risk management and remedy selection should also consider pertinent recommendations from stakeholders, which frequently include the local community, local government, states, Indian tribes, and responsible parties. Remediation may significantly impact day-to-day activities of residents and recreation-seekers, and operations of commercial establishments near the water body for extended periods. Stakeholders should be involved when designing and scheduling remedial operations, not just during the remedy selection process. Documenting and communicating how and why remedy decisions are made are very important tasks at sediment sites. For guidance on documenting remedy decisions under CERCLA, project managers should refer to EPA s *A Guide to Preparing Superfund Proposed Plans, Records of Decision, and other Remedy Selection Documents*, also referred to as the ROD Guidance (U.S. EPA 1999a).

7.2 NCP REMEDY SELECTION FRAMEWORK

In the NCP, EPA provides a series of expectations (see Highlight 7-1) to reflect the principal requirements under CERCLA 121 and to help focus the remedial investigation/feasibility study (RI/FS) on appropriate cleanup options. EPA developed nine criteria for evaluating remedial alternatives to ensure that all important considerations are factored into remedy selection decisions. Chapter 3, Section 3.2 outlines the NCP s nine remedy selection criteria. These criteria are derived from the statutory requirements under CERCLA 121, as well as technical and policy considerations that have proven to be important for selecting among the remedial alternatives. In general, the nine criteria analysis comprises the following two steps: 1) an evaluation of all alternatives with respect to each criterion; and 2) a comparison among the alternatives to determine the relative performance of the alternatives and identify major trade-offs among them (i.e., relative advantages and limitations). Generally this comparison is made on a qualitative basis, although some have attempted a quantitative analysis (e.g., Linkov et al. 2004). Ultimately, the remedy selected must be protective of human health and the environment, attain (or waive) applicable or relevant and appropriate requirements (ARARs), be cost effective, use permanent solutions and alternative treatment technologies or resource recovery technologies to the maximum extent

practicable, and satisfy a preference for treatment or provide an explanation as to why this preference was not met.

Consistent with CERCLA and the NCP, each remedial action selected should be cost-effective. The NCP provides several threshold criteria that should be satisfied (40 CFR §300.430(f)(ii)(D)). Cost-effectiveness is generally determined by evaluating three of the five balancing criteria: 1) long-term effectiveness and permanence; 2) reduction of toxicity, mobility, or volume of hazardous substances through treatment; and 3) short-term effectiveness. A remedy typically is considered cost effective when its cost is proportional to its overall effectiveness. As described in the preamble to the NCP, more than one alternative may be considered cost-effective (55 *Federal Register* (*FR*) 8728, March 8, 1990). The relationship between overall effectiveness and cost should be examined across all alternatives to identify which options can best afford effectiveness proportional to their cost. The evaluation of an alternative s cost effectiveness is usually concerned with the reasonableness of the relationship between the effectiveness afforded by each alternative and its costs when compared to other available options (U.S. EPA 1999a).

For some complex sediment sites, there may be a high degree of uncertainty about the predicted effectiveness of various remedial alternatives. Where this is the case, it is especially important to identify and factor that uncertainty into site decisions. Project managers are encouraged to consider a range of probable effectiveness scenarios that includes both optimistic and non-ideal site conditions and remedy performance.

The NCP lists six expectations that EPA generally considers in developing appropriate remedial alternatives at Superfund sites (40 CFR §300.430(a)(1)(iii)). Highlight 7-1 discusses how the six expectations may be relevant for sites with contaminated sediment. Generally, the expectations are addressed by seeking the best balance of trade-offs among the alternatives evaluated.

7.3 CONSIDERING REMEDIES

If the baseline risk assessment determines that contaminated sediment presents an unacceptable risk to human health or the environment, remedial alternatives should be developed to reduce those risks to acceptable levels. As discussed in Chapter 3, Section 3.1, Developing Remedial Alternatives for Sediment, due to the limited number of approaches available for contaminated sediment, generally, project managers should evaluate each of the three major approaches monitored natural recovery (MNR), in-situ capping, and removal through dredging or excavation at every sediment site. Depending on site-specific conditions, contaminant characteristics, and/or health or environmental risks at issue, certain methods or combinations of methods may prove more promising than others. Each site and the various sediment areas within it presents a unique combination of circumstances that should be considered carefully in selecting a comprehensive site-wide cleanup strategy. At large or complex sediment sites, the remedy decision frequently involves choices between areas of the site and how they are best suited to particular cleanup methods rather than a simple one-size-fits-all choice between approaches for the entire site.

Project managers should keep in mind that deeper contaminated sediment that is not currently bioavailable or bioaccessible, and that analyses have shown to be stable to a reasonable degree, do not necessarily contribute to site risks. In evaluating whether to leave buried contaminated sediment in place, project managers should include an analysis of several factors, including the depth to which significant

Highlight 7-1: NCP Remedy Expectations and Their Potential Application to Contaminated Sediment

EPA expects to use treatment to address the principal threats posed by a site, wherever practicable:

- In general, wastes, including contaminated sediment, may be considered a principal threat where toxicity and mobility combine to pose a potential human health risk of 10^{-3} or greater for carcinogens (U.S. EPA 1991d). For these areas, project managers should evaluate an alternative that includes treatment. However, the practicability of treatment, and whether a treatment alternative should be selected, should be evaluated against the NCP's nine remedy selection criteria. Based on available technology, treatment is not considered practicable at most sediment sites

EPA expects to use engineering controls, such as containment, for waste that poses a relatively low long-term threat or where treatment is impracticable:

- Containment options for sediment generally focus on in-situ capping. A project manager should evaluate in-situ capping for every sediment site that includes low-level threat waste. Where a containment alternative is clearly not appropriate for a detailed evaluation, project managers should evaluate ex-situ containment (i.e., disposal without treatment). It should be recognized that in-situ containment can also be effective for principal threat wastes, where that approach represents the best balance of the NCP nine remedy selection criteria

EPA expects to use a combination of methods, as appropriate, to achieve protection of human health and the environment:

- Large or complex contaminated sediment sites or operable units frequently require development of alternatives that combine various approaches for different parts of the site. For a broader discussion on this topic, refer to Chapter 3, Section 3.1.1, Alternatives that Combine Approaches

EPA expects to use institutional controls, such as water use and deed restrictions, to supplement engineering controls as appropriate for short- and long-term management to prevent or limit exposure to hazardous substances, pollutants, or contaminants:

- Institutional controls such as fish consumption advisories, fishing bans, ship draft/anchoring/wake controls, or structural maintenance requirements (e.g., dam or breakwater maintenance) are frequently a part of sediment alternatives, especially where contaminated sediment is left in place, or where remedial goals in fish tissue cannot be met for some time. See Chapter 3, Section 3.6, Institutional Controls, for additional discussion

EPA expects to consider using innovative technology when such technology offers the potential for comparable or superior treatment performance or implementability, fewer or lesser adverse impacts than other available approaches, or lower costs for similar levels of performance than demonstrated technologies:

- Innovative technologies are technologies whose limited number of applications may result in less cost and performance data, frequently due to limited field application. Additional cost and performance data may be needed for many sediment remedies, and field demonstrations of new techniques and approaches may be especially needed, including both innovative in-situ and ex-situ technologies. Although most innovations for sediment remedies are currently in the research phase, as they become available, project managers should consider using them

EPA expects to return reusable ground waters to their beneficial uses wherever practicable, within a time frame that is reasonable given the circumstances for the site. When restoration of ground water to beneficial uses is not practicable, EPA expects to prevent further migration of the plume, prevent exposure to the contaminated ground water, and evaluate further risk reduction:

- Ground water may be a continuing source of sediment and surface water contamination. Where this is the case, ground water migration prevention may be very important to a successful sediment cleanup and to protect benthic biota. Ground water restoration may also be needed to return the ground water to a beneficial use

populations of organisms burrow, the potential for erosion due to natural or anthropogenic (man-made) forces, the potential for contaminant movement via ground water, and the effectiveness of any institutional controls (ICs) to limit sediment disturbance. In some cases, the most appropriate approach may be long-term monitoring, with contingency actions, if necessary.

To assist project managers in evaluating cleanup options, two summary highlights are presented below. Highlight 7-2 provides general site, sediment, and contaminant characteristics or conditions especially conducive to each of the three common sediment approaches. This highlight is intended as a general tool for project managers as they look more closely at particular approaches when most of these characteristics are present. Project managers should note that these characteristics are not requirements. It is important to remain flexible when evaluating sediment alternatives and when considering approaches that at first may not appear the most appropriate for a given environment. When an approach is selected for a site that has one or more site characteristics or conditions appearing problematic, additional engineering or ICs may be available to enhance the remedy. Some of these situations are discussed in the remedy-specific chapters (Chapters 4, 5, and 6).

Highlight 7-2: Some Site Characteristics and Conditions Especially Conducive to Particular Remedial Approaches for Contaminated Sediment

Characteristics	Monitored Natural Recovery	In-situ Capping	Dredging Excavation
General Site Characteristics	Anticipated land uses or new structures are not incompatible with natural recovery Natural recovery processes have a reasonable degree of certainty to continue at rates that will contain, destroy, or reduce the bioavailability or toxicity of contaminants within an acceptable time frame	Suitable types and quantities of cap material are available Anticipated infrastructure needs (e.g., piers, pilings, buried cables) are compatible with cap Water depth is adequate to accommodate cap with anticipated uses (e.g., navigation, flood control) Incidence of cap-disrupting human behavior, such as large boat anchoring, is low or controllable	Suitable disposal sites are available Suitable area is available for staging and handling of dredged material Existing shoreline areas and infrastructure (e.g., piers, pilings, buried cables) can accommodate dredging or excavation needs Navigational dredging is scheduled or planned
Human and Ecological Environment	Expected human exposure is low and/or reasonably controlled by ICs Site includes sensitive, unique environments that could be irreversibly damaged by capping or dredging	Expected human exposure is substantial and not well-controlled by ICs Long-term risk reduction outweighs habitat disruption, and/or habitat improvements are provided by the cap	Expected human exposure is substantial and not well-controlled by ICs Long-term risk reduction of sediment removal outweighs sediment disturbance and habitat disruption

Characteristics	Monitored Natural Recovery	In-situ Capping	Dredging Excavation
Hydrodynamic Conditions	Deposition of sediment is occurring in the areas of contamination Hydrodynamic conditions (e.g., floods, ice scour) are not likely to compromise natural recovery	Hydrodynamic conditions (e.g., floods, ice scour) are not likely to compromise cap or can be accommodated in design Rates of ground water flow in cap area are low and not likely to create unacceptable contaminant releases	Water diversion is practical, or current velocity is low or can be minimized to reduce resuspension and downstream transport during dredging
Sediment Characteristics	Sediment is resistant to resuspension (e.g., cohesive or well-armored sediment)	Sediment has sufficient strength to support cap (e.g., has high density/low water content)	Contaminated sediment is underlain by clean sediment (so that over-dredging is feasible) Sediment contains low incidence of debris (e.g., logs, boulders, scrap material) or is amenable to effective debris removal prior to dredging or excavation
Contaminant Characteristics	Contaminant concentrations in biota and in the biologically active zone of sediment are moving towards risk-based goals Contaminants readily biodegrade or transform to lower toxicity forms Contaminant concentrations are low and cover diffuse areas Contaminants have low ability to bioaccumulate	Contaminants have low rates of flux through cap Contamination covers contiguous areas (e.g., to simplify capping)	Higher contaminant concentrations cover discrete areas Contaminants are highly correlated with sediment grain size (i.e., to facilitate separation and minimize disposal costs)

Highlight 7-3 may assist project managers in evaluating cleanup options. For convenience, these comparisons are organized around the NCP s nine remedy selection criteria. This highlight is intended only to identify some of the general differences between these three remedy types, not as an example of an actual comparative alternatives analysis for a site. An actual site alternatives analysis would typically include more complex alternatives and many site-specific details, as described in the ROD Guidance (U.S. EPA 1999a) and EPA s *Guidance for Conducting Remedial Investigations and Feasibility Studies under CERCLA* (U.S. EPA 1988a, commonly referred to as the RI/FS Guidance). The example criterion components column used in Highlight 7-3 below are adapted from the RI/FS Guidance and are

intended only as examples of some of the components that may be considered when evaluating each remedy selection criterion.

Highlight 7-3: Examples of Some Key Differences Between Remedial Approaches for Contaminated Sediment

NCP Remedy Selection Criteria	Example Criterion Components	Monitored Natural Recovery	In-Situ Capping	Dredging Excavation
Overall Protective-ness		Generally relies upon natural processes for protection May provide low level of short-term protection, but may provide potentially acceptable long-term protection	Generally, relies upon adequate cap placement and maintenance for protection May provide moderate to high level of protection, depending upon areal extent, design of cap, and long-term maintenance	Generally, relies upon effective removal and low residual levels for protection May provide moderate to high level of protection, depending on residual, or where remedy is combined with backfilling, capping, or MNR
Compliance with Applicable or Relevant and Appropriate Require-ments (ARARs)		Generally, only chemical-specific ARARs apply (these would also apply to other approaches)	Generally, the Clean Water Act (CWA) §404 (regulates discharge of dredged or fill materials into waters of the U.S.) and the Rivers and Harbors Act (prohibits obstruction or alteration of a navigable waterway) are ARARs See Chapter 3, Section 3.3, for additional examples of ARARs	Generally, CWA §404 and the Rivers and Harbors Act are ARARs. Generally, treatment facilities and in-water disposal sites should meet substantive requirements of the CWA §§404 and 401 for discharge of effluents into waters of the U.S. Generally, state solid hazardous waste rules and RCRA is an ARAR for disposal in solid or hazardous waste landfills See Chapter 3, Section 3.3, for additional examples of ARARs
Long-Term Effective-ness and Permanence	**Magnitude of Risk Reduction and Residual Risks**	May provide low to high level of risk reduction and residual risk, depending on processes being relied upon and site-specific characteristics that might enhance or prevent long-term isolation or destruction of contaminants	May provide moderate to high level of risk reduction and low to moderate residual risk, depending on cap design, placement, construction, and maintenance to address site characteristics that might otherwise prevent long-term isolation of contaminants	May provide moderate to high level of risk reduction and low to moderate residual risk, depending on effectiveness of dredging and use of backfill material May provide low (upland) to moderate (in-water) residual risk for sediments and treatment residuals contained at controlled disposal sites

NCP Remedy Selection Criteria	Example Criterion Components	Monitored Natural Recovery	In-Situ Capping	Dredging Excavation
Long-Term Effective-ness and Permanence (cont.)	**Adequacy and Reliability of Controls for Residual Risk**	May provide low control, but potentially acceptable, depending on processes being relied upon and site-specific conditions May provide moderate ability to control physical disturbance due to human activity via institutional controls; may provide little ability to control physical disturbance due to natural forces May provide no ability to control advection and diffusion of contaminants through overlying cleaner sediment, where this is of concern	May provide moderate to high control, depending on cap stability and contaminant migration through cap May provide low to moderate ability to control physical disturbance due to human and natural forces and to control effects of advective flow and diffusion through cap design and moderate ability to control disruption through institutional controls	May provide high control due to removal of contaminants, if residual contamination is below cleanup levels or addressed through backfilling, or capping May leave residual risks at upland disposal sites that are easily controlled; at in-water sites control can be more complex
	Need for Five-Year Reviews	Five-year reviews generally would be required for most sites due to waste left in place and possible continuing need for use restrictions	Five-year reviews generally would be required for most sites due to waste left in place and possible continuing need for use restrictions	Five-year review may be generally required until remedial action objectives are met Reviews generally required for on-site disposal facilities
Reduction of Toxicity, Mobility, and Volume (TMV) Through Treatment		No treatment is involved	Typically, no treatment is involved Research is ongoing concerning the combination of innovative in-situ treatment components within a cap	Sediment is treated in some cases if practical and cost-effective; stabilization is most common form Potential exists for beneficial reuse of dredged sediment Water treatment can reduce TMV of contaminants where significant quantities of toxics are removed from the water

NCP Remedy Selection Criteria	Example Criterion Components	Monitored Natural Recovery	In-Situ Capping	Dredging Excavation
Short-Term Effective-ness	**Environ-mental Impacts During Remedy Implemen-tation**	There should be no additional impact to bottom-dwelling ecological community from the remedy itself, but impacts of contaminated sediment on environment continue until protection is achieved	May provide high impact to bottom habitat in area of cap. Cap design can facilitate recolonization in some cases May provide low potential for impacts from releases to the environment during cap placement and initial consolidation	May provide high impact to bottom habitat in dredged area. Backfill design can facilitate recolonization in some cases May provide moderate potential for impacts to biota from release during dredging; releases partially controllable by physical barriers and by selection and operation of dredging equipment
	Community and Worker Protection During Remedy Implementa-tion	There should be no additional health impacts to community from the remedy itself; any pre-existing impacts would continue until protection is achieved May provide moderate ability to control community impacts from fish/shellfish ingestion and, where applicable, direct contact with contaminated sediment, through consumption advisories and use restrictions There should be minimal impacts on workers and community from monitoring activities	There should be low potential for health impacts to community and workers from contaminant releases during cap placement. Engineering controls may minimize these releases; worker protection generally available Increased truck or rail traffic for transport of cap material may impact workers and the community Staging needs for cap placement may disrupt local community during placement	There should be low to moderate potential for health impacts to community and workers from contaminant release during dredging, staging, transport, and disposal. Engineering controls may minimize these releases; worker protection generally available Increased truck or rail traffic for transport of dredged material may impact workers and the community Dredged materials and water handling or treatment needs may disrupt local community during dredging

NCP Remedy Selection Criteria	Example Criterion Components	Monitored Natural Recovery	In-Situ Capping	Dredging Excavation
Short-Term Effective-ness (cont.)	**Time Until Protection is Achieved**	Generally, longest time to achieve protection, depending on rates of natural processes and bioavailability of the contaminants Time to achieve protection is frequently highly uncertain	Generally, shortest time to achieve protection Complete biota recovery could take several years Generally, most certainty concerning time to achieve protection	Time to achieve protection varies depending on the size and complexity of the project Complete biota recovery could take several years Time frame generally more uncertain than for capping due to difficulty of predicting residual contamination
Implement-ability	**Technical Feasibility**	Generally, no construction is required Reliability can be uncertain in some environments due to uncertain rates of natural processes and uncertainties concerning sediment stability Where site-specific conditions allow, should be relatively easy to implement a different remedy if MNR is not effective Methods for monitoring sediment cleanup levels are relatively well established	Cap placement methods are generally well-established; ability to construct a cap depends on a number of factors including water depth and currents, slope and geotechnical stability of underlying materials, and stability of the cap itself during and after construction Reliability generally high, depending on site-specific conditions, and degree of monitoring and maintenance Relatively easy to repair cap in case of localized erosion or disruption, but can be difficult or costly to implement sediment removal if cap is not effective Methods for monitoring cap integrity and contaminant migration within cap are relatively well established	Dredging and excavation methods are generally well-established; technical feasibility of dredging depends on a number of factors including accessibility, extent of debris, and the ability to over-dredge Disposal in upland landfills is a well-established technique; in-water disposal methods are less well-established and may require greater monitoring; technical feasibility generally depends on distance to the disposal site, ease of dewatering, and slope and geotechnical stability of disposal site May be necessary to re-dredge, cap or implement MNR if dredging alone does not meet cleanup standards Monitoring methods for sediment cleanup levels and short-term releases from dredging are relatively well established

NCP Remedy Selection Criteria	Example Criterion Components	Monitored Natural Recovery	In-Situ Capping	Dredging Excavation
Implement-ability (cont.)	**Administra-tive Feasibility**	State-regulated ICs, including fish consumption advisories where contaminants are bioaccumulative, may be needed for a longer period than for other remedies	Containment in public waters can require long-term coordination with state and local regulators due to potential need for long-term controls on waterway use Where contaminants are bioaccumulative, fish consumption advisories frequently needed for a period of years. Length of time generally depends on residual contamination outside of capped area	Dredging and excavation plan should be coordinated with other agencies to ensure compatibility with other waterway uses and habitat concerns during the removal operation Where contaminants are bioaccumulative, fish consumption advisories frequently needed for a period of years. Length of time generally depends on residual contamination within and outside of dredged area Disposal siting often requires extensive coordination with several government agencies and the public
	Availability of Services, Materials, Capacities, and Equipment	Monitoring and analytical services are generally readily available	Location and suitability of capping material source is critical and can be problematic if not available locally Specialized cap placement equipment may be needed in some environments, but are generally available Availability of suitable cap material staging areas is critical and can be problematic for some sites (e.g., some urban areas)	Environmental dredging and excavation equipment is generally available, although availability may be a problem for large projects. Specialized equipment may need to be constructed for special situations Availability of suitable dredged material staging, separation, and, where required, water treatment capacity is critical and can be problematic for some sites (e.g., some urban areas) Availability of a suitable disposal facility is critical and can be problematic for some sites (e.g., where local disposal is infeasible or high volumes are involved)

NCP Remedy Selection Criteria	Example Criterion Components	Monitored Natural Recovery	In-Situ Capping	Dredging Excavation
Cost		Generally, no capital cost Long-term monitoring costs typically continue until cleanup levels and remedial action objectives are met. Length of long-term monitoring is generally dependent on assurance of sediment stability	Capital costs generally higher than MNR and lower than dredging/ excavation Long-term maintenance and monitoring costs generally higher than MNR and dredging/ excavation Long-term monitoring costs typically continue until cleanup levels and remedial action objectives are met. Length of long-term operation and maintenance (O&M) period dependent on time necessary to verify long-term stability of cap and lack of significant contaminant fluxes through cap	Capital costs generally higher than MNR or capping Long-term monitoring costs generally lower than MNR and capping Long-term monitoring costs typically continue until cleanup levels and remedial action objectives are met. Length of long-term O&M period dependent on extent of residual contamination and use of on-site disposal
State Acceptance and Community Acceptance		Commonly identified benefits include lack of disruption to local residents, lack of disruption to aquatic and terrestrial animal and plant life, and low cost	Commonly identified benefits include use of an active remedy with no disposal issues, generally moderate cost, and potentially faster biota recovery than MNR or dredging due to rapid placement of exposure barrier	Commonly identified benefits include removing contaminants from waterway, possible treatment of contaminants, faster biota recovery than MNR, increased/restored navigational depth, decreased flooding, and lack of use limitations after completion

NCP Remedy Selection Criteria	Example Criterion Components	Monitored Natural Recovery	In-Situ Capping	Dredging Excavation
State Acceptance and Community Acceptance (cont.)		Commonly identified concerns include objections to a "do nothing" remedy, leaving contamination in place, possible spread of contaminants during flooding or other disruption; uncertainties of predicting rates of natural burial; and a potentially lengthy period of fish consumption advisories	Commonly identified concerns include leaving contamination in place, temporary disruption to local residents and businesses, increased truck, rail or barge traffic during capping; temporarily reduced recreational access; potentially long-term reduction of navigational waterway access; reduced access to buried utilities, possible long-term anchoring or other waterway use restrictions, and costs to potentially responsible parties (PRPs) and/or state during O&M	Commonly identified concerns include temporary disruption to local residents and businesses, contaminant releases during dredging, temporary reduction of recreational and navigational waterway access during dredging; siting of and risks from local disposal facilities; and increased truck, rail, or barge traffic during dredging

7.4 COMPARING NET RISK REDUCTION

Each approach to managing contaminated sediment has its own uncertainties and potential relative risks. The concept of comparative net risk reduction was discussed by the NRC as a method to ensure that all positive and negative aspects of each sediment management approach were appropriately considered at contaminated sediment sites. The Committee on Remediation of PCB-Contaminated Sediments states that (NRC 2001):

> All remediation technologies have advantages and disadvantages when applied at a particular site, and it is critical to the risk management that these be identified individually and as completely as possible for each site. For example, managing risks from contaminated sediment in the aqueous environment might result in the creation of additional risks in both aquatic and terrestrial environments... Removal of contaminated materials can adversely impact existing ecosystems and can remobilize contaminants, resulting in additional risks to humans and the environment. Thus, management decisions at a contaminated sediment site should be based on the relative risks of each alternative management action... For a site, it is important to consider overall or net risk in addition to specific risks.

Project managers are encouraged to use the concept of comparing net risk reduction between alternatives as part of their decision-making process for contaminated sediment sites, within the overall framework of the NCP remedy selection criteria. Consideration should be given not only to risk reduction associated with reduced human and ecological exposure to contaminants, but also to risks

introduced by implementing the alternatives. The magnitude of implementation risks associated with each alternative generally is extremely site-specific, as is the time frame over which these risks may apply to the site. Evaluation of both implementation risk and residual risk are existing important parts of the NCP remedy selection process. By evaluating these two concepts in tandem, additional information may be gained to help in the remedy selection process. Highlight 7-4 provides examples of elements that could be evaluated by project managers in this comparative evaluation.

Highlight 7-4: Sample Elements for Comparative Evaluation of Net Risk Reduction

Elements Potentially Reducing Risk

- Reduced exposure to bioavailable/bioaccessible contaminants
- Removal of bioavailable/bioaccessible contaminants
- Removal or containment of buried contaminants that are likely to become bioaccessible

Elements Potentially Continuing or Increasing Risk

For MNR:

- Continued exposure to contaminants already at sediment surface and in food chain
- Potential for undesirable changes in the site's natural processes (e.g., lower sedimentation rate)
- Potential for contaminant exposure due to erosion or human disturbance

For In-Situ Capping:

- Contaminant releases during capping
- Continued exposure to contaminants currently in the food chain
- Other community impacts (e.g., accidents, noise, residential or commercial disruption)
- Worker risk during transport of cap materials and cap placement
- Releases from contaminants remaining outside of capped area
- Potential contaminant movement through cap
- Disruption of benthic community

For Dredging or Excavation:

- Contaminant releases during sediment removal, transport, or disposal
- Continued exposure to contaminants currently in the food chain
- Other community impacts (e.g., accidents, noise, residential or commercial disruption)
- Worker risk during sediment removal and handling
- Residual contamination following sediment removal
- Releases from contaminants remaining outside dredged/excavated area
- Disruption of benthic community

7.5 CONSIDERING INSTITUTIONAL CONTROLS (ICs)

Institutional controls (ICs) such as fish consumption advisories, fishing bans, or ship draft/anchoring/wake controls are common parts of sediment remedies (see Chapter 3, Section 3.6, Institutional Controls). Structural maintenance agreements are another legal mechanism that may be important for protecting some remedies. 40 CFR §300.430(a)(1)(iii)(D) contains the following general EPA expectations with respect to ICs. These expectations generally apply to all Superfund sites, including sediment sites:

- EPA expects to use institutional controls such as water use and deed restrictions to supplement engineering controls as appropriate for short- and long-term management to prevent or limit exposure to hazardous substances, pollutants, or contaminants;

- Institutional controls may be used during the conduct of the RI/FS and implementation of the remedial action and, where necessary, as a component of the completed remedy; and

- The use of institutional controls shall not be substituted for active response measures (e.g., treatment and/or containment of source material, restoration of ground waters to their beneficial uses) as the sole remedy unless such active measures are determined not to be practicable, based on the balancing of trade-offs among alternatives that is conducted during the selection of remedy.

EPA policies concerning ICs are explained in *Institutional Controls: A Site Manager's Guide to Identifying, Evaluating, and Selecting Institutional Controls at Superfund and RCRA Corrective Action Cleanups* (U.S. EPA 2000f). In addition to considering the NCP expectations concerning ICs, the project manager should determine what entities possess the legal authority, capability and willingness to implement, and where applicable, monitor, enforce, and report on the status of the IC. An evaluation should also be made of the durability and effectiveness of any proposed IC. The objectives of any ICs contained in the selected alternative should be clearly stated in the ROD or other decision document together with any relevant performance standards. While the specific IC mechanism need not be identified, the types of ICs envisioned should be discussed in sufficient detail to support a conclusion that effective implementation of the ICs can be reasonably expected. For some federal facilities in the CERCLA program, the IC implementation details (i.e., the specific IC mechanism) should be placed in the ROD. The program manager should refer to EPA s *Guidance on the Resolution of the Post-ROD Dispute* (U.S. EPA 2003d) for guidelines describing and documenting ICs in Federal Facility RODs, Remedial Designs, Remedial Action Workplans, and Federal Facility Agreements/Interagency Agreements.

Reliability and effectiveness of ICs are of particular concern with sediment alternatives, whether they are used alone or in combination with MNR, in-situ capping, or sediment removal. Project managers should recognize that, generally, ICs cannot protect ecological receptors or prevent disruption of an in-situ cap by bottom-dwelling organisms. In addition, in many cases ICs have been only partially effective in modifying human behavior, especially in the case of voluntary or advisory controls. Although fish consumption advisories can be an important component of a sediment remedy, it should be recognized that they are unlikely to be entirely effective in eliminating exposures. Where advisories or bans are relied upon to reduce human health risk for long periods, public education, and where applicable, enforcement by the appropriate agency, are critical. This point is emphasized in EPA s risk management Principle 9, Maximize the Effectiveness of Institutional Controls and Recognize Their Limitations (U.S. EPA 2002a; see Appendix A).

Implementing and overseeing ICs can often be more difficult at sediment sites where control of the water body may involve multiple entities and a single landowner is not present to provide oversight and enforcement. As for other types of sites, at sediment sites, project managers should review ICs during the five-year review. Where a water body is owned or controlled by local, state, or federal

government entities, their regulations and guidance should be consulted to determine what governmental controls can be used to restrict the use of the water body, and the regulatory or administrative process to enforce such a restriction. In complex situations, it may be useful to layer a number of different ICs as discussed in the ICs site manager s guide (U.S. EPA 2000f). Additional guidance on other aspects of ICs is under development by EPA.

7.6 CONSIDERING NO-ACTION

As presented in Section 8.1 of the ROD Guidance, a no-action decision may be appropriate in the following situations:

- When the site or operable unit poses no current or potential threat to human health or the environment;
- When CERCLA does not provide the authority to take remedial action; or
- When a previous response(s) has eliminated the need for further remedial response [often called a no-further-action alternative].

Generally, if ICs are necessary to control risks caused by a contaminant of concern at a site, a no-action decision is not appropriate. For example, if fish consumption advisories or fishing bans are necessary to control risks from contaminants of concern at a site, a no-action decision for sediment is not appropriate, even if the advisories or bans are already in place. Instead, a remedy should be considered that includes at least the institutional control (e.g., advisories or bans), and, if appropriate, other actions for sediment or other media.

A no-action decision; however, may include monitoring. For example, sediment may pose no unacceptable risk to human health or the environment; however, uncertainties concerning that evaluation may make it wise to continue some level of monitoring. In this case, a no-action decision that includes monitoring may be appropriate. It is important to note that this is different from a MNR remedy where current or expected future risk is unacceptable and natural processes are being relied upon to reduce that risk to an acceptable level within a reasonable time frame. Although a no-action decision may require long-term monitoring, a MNR remedy generally needs more intensive monitoring to show that contaminant concentrations are being reduced by anticipated mechanisms at the predicted rates.

7.7 CONCLUSIONS

The focus of remedy selection should be on selecting the alternative best representing the overall risk reduction strategy for the site according to the NCP nine remedy selection criteria. As discussed in the OSWER Directive 9285.6-08, *Principles for Managing Contaminated Sediment Risks at Hazardous Waste Sites* (U.S. EPA 2002a), EPA s policy has been and continues to be that there is no presumptive remedy for any contaminated sediment site, regardless of the contaminant or level of risk. Generally, as discussed in Chapter 3, Feasibility Study Considerations, project managers should evaluate each of the three potential remedy approaches (i.e., MNR, in-situ capping, and removal through dredging or excavation) at every sediment site. Project managers should develop a conceptual site model that considers key site uncertainties. Such a model can be used within an adaptive management approach to

control sources and to implement a cost-effective remedy that will achieve long-term protection while minimizing short-term impacts (refer to Chapter 2, Section 2.2 on conceptual site models).

Controlling any continuing sources of contaminants is an important factor for any sediment remedy (U.S. EPA 2002a). Where source control is uncertain, cannot be achieved, or is outside the scope of the remedial action, project managers should consider the potential for recontamination and factor that potential into the remedy selection process and into the long-term monitoring plan for the site. However, project managers should note that delaying an action to complete source control may not always be wise. Early actions in some areas may be appropriate as part of a phased approach to address site-wide contamination even if sources are not fully controlled initially; in such situations, careful consideration should be given as to whether the uncontrolled sources will cause the early action to be ineffective.

At many sites, but especially at large sites, the project manager should consider a combination of sediment approaches as the most effective way to manage the risk. This is because the characteristics of the contaminated sediment and the settings in which it exists are not usually homogeneous throughout a water body (NRC 2001). As discussed in the remedy-specific chapters of this document, when evaluating alternatives, project managers should include realistic assumptions concerning residuals and contaminant releases from in-situ and ex-situ remedies, the potential effects of those residuals and releases, and the length of time a risk may persist.

The project manager should include a scientific analysis of sediment stability in the remedy selection process for all sites where sediment erosion or contaminant transport is a potential concern. Typically, it is not sufficient to assume that a site as a whole is depositional or erosional. Generally, as discussed in Chapter 2, Remedial Investigation Considerations, project managers should make use of available empirical and modeling methods for evaluating sediment stability and fate and transport, especially when there are significant differences between alternatives.

The project manager should include in the remedy selection process a clear analysis of the uncertainties involved, including uncertainties concerning the predicted effectiveness of various alternatives and the time frames for achieving cleanup levels and remedial action objectives. Project managers should quantify, as far as possible, the uncertainty of the factors that are most important to the remedy decision. Where it is not possible to quantify uncertainty, the project manager should use a sensitivity analysis to determine which apparent differences between alternatives are most likely to be significant.

The project manager should monitor all sediment remedies during and after implementation to determine if the actions are effective and if all cleanup levels and remedial action objectives are met. Sediment remedies should not only include monitoring of surficial sediment immediately following implementation of the action, but also long-term monitoring of sediment to assess changes in residual contamination and possible recontamination, as well as monitoring of fish or other relevant biota recovery data. Without these data, an assessment of the long-term effectiveness of the remedy is difficult, and five-year reviews may be difficult to perform accurately. Additional monitoring data may help not only to assess the site but to help build a body of knowledge that will decrease uncertainties in decision making at future sites. Chapter 8, Remedial Action and Long-Term Monitoring, discusses these and other general monitoring considerations for contaminated sediment sites.

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8.0 REMEDIAL ACTION AND LONG-TERM MONITORING

This chapter provides a recommended approach to developing an effective monitoring plan at contaminated sediment sites. A monitoring plan is recommended for all types of sediment remedies, both during and after remedial action. Monitoring should be conducted at most contaminated sediment sites for a variety of reasons, including: 1) to assess compliance with design and performance standards; 2) to assess short-term remedy performance and effectiveness in meeting sediment cleanup levels; and/or 3) to evaluate long-term remedy effectiveness in achieving remedial action objectives (RAOs) and in reducing human health and/or environmental risk. In addition, monitoring data are usually needed to complete the five-year review process where a review is conducted.

A fully successful sediment remedy typically is one where the selected sediment chemical or biological cleanup levels have been met and maintained over time, and where all relevant risks have been reduced to acceptable levels based on the anticipated future uses of the water body and the goals and objectives stated in the record of decision (ROD). Due to the significant post-remedial residual contamination at some sites, or the inability to control all sources of contamination to the water body, reaching sediment or biota levels resulting in unlimited exposure and unrestricted use may take many years if not decades. Where appropriate, several interim measures of remedy effectiveness should be evaluated at most sites in addition to the key measure of long-term risk reduction. Highlight 8-1 presents four measures that should be considered for all Superfund sediment sites where the remedy includes active remediation such as dredging, excavation, and/or capping. At sites where achieving protection relies upon institutional controls (ICs) such as fish consumption advisories and/or on monitored natural recovery (MNR), only measures 2 and 4would typically apply. A monitoring plan that addresses the appropriate measures generally should be developed and implemented at every sediment site. The term remedy effectiveness as used in Highlight 8-1 of this guidance addresses the potential role of monitoring in measuring progress, not as one of the nine criteria provided in National Oil and Hazardous Substances Pollution Contingency Plan (NCP) to evaluate alternatives.

Highlight 8-1: Sample Measures of Sediment Remedy Effectiveness

Interim Measures:

1 - Short-term remedy performance (e.g., Have the sediment cleanup levels been achieved? Was the cap placed as intended?)

2 - Long-term remedy performance (e.g., Have the sediment cleanup levels been reached and maintained for at least five years, and thereafter as appropriate? Has the cap withstood significant erosion?)

3 - Short-term risk reduction (e.g., Do data demonstrate or at least suggest a reduction in fish tissue levels, a decrease in benthic toxicity, or an increase in species diversity or other community indices after five years?)

Key Measure:

4 - Long-term risk reduction (e.g, Have the remediation goals in fish tissue been reached or has ecological recovery been accomplished?)

For Fund-lead sites subject to a state cost share, it may be necessary to distinguish monitoring that is part of the remedial action phase of the remedy from monitoring that is associated with the

operation and maintenance (O&M) phase of the remedy. Distinguishing these two monitoring activities is a site-specific decision. Project managers may find it useful to refer to Chapter 3, Section 3.5.2, Operation and Maintenance Costs, for suggestions about what types of activities are frequently associated with long-term O&M as compared to similar activities typically conducted during the remedial action.

This chapter is based in part on the framework presented in the U.S. Environmental Protection Agency s (EPA s) new Monitoring Guidance, Office of Solid Waste and Emergency Response (OSWER) Directive 9355.4-28, *Guidance for Monitoring at Hazardous Waste Sites: Framework for Monitoring Plan Development and Implementation* (U.S. EPA 2004c). This chapter presents more specific guidance for monitoring of sediment sites; however, many technical details are outside the scope of this chapter. More specific guidance on particular monitoring topics is under development by EPA to assist project managers. In addition, the triad approach to systematic planning, dynamic work plans and real-time measurement technologies may have strategies that can be fruitfully applied to sediment site monitoring (see http://www.epa.gov/tio/triad).

8.1 INTRODUCTION

As described in EPA s Monitoring Guidance (U.S. EPA 2004c), monitoring may be viewed as the collection and analysis of repeated observations or measurements to evaluate changes in condition and progress toward meeting a management objective. Monitoring should include the collection of field data (i.e., chemical, physical, and/or biological) over a sufficient period of time and frequency to determine the status at a particular point in time and/or trend over a period of time in a particular environmental parameter or characteristic, relative to clearly defined management objectives. The data, methods, and endpoints should be directly related to the RAOs and cleanup levels or remediation goals for the site.

Environmental sampling and analysis is typically conducted during all phases of the Superfund process to address various questions. By the time a project manager is implementing a remedial action or writing a monitoring plan, a considerable amount of baseline site data should have been collected during the remedial investigation or site characterization phase. In the site characterization phase, sampling is performed to determine the nature and extent of contamination, to develop the information necessary to assess risks to human health and the environment, and to assess the feasibility of remedial alternatives. During site characterization, the project manager should anticipate expected post-remedy monitoring needs to ensure that adequate baseline data are collected to allow comparisons to future data sets. Monitoring plans should also be designed to allow comparison of results with model predictions that supported remedy selection.

Project managers should ensure that agreements with contractors or responsible parties concerning remedial design and remedial action include requirements for development of an appropriate monitoring plan. The need for environmental monitoring and how the data will be used to measure performance against cleanup levels and RAOs should be considered in the ROD and discussed further early in the remedial design process. Where ICs are part of the remedy, this discussion should also include implementation and, where appropriate, monitoring plans for those controls. Having an early discussion of the monitoring needs as they relate to any engineering performance standards for the particular remedies should allow the project manager sufficient time to resolve logistical or other implementation issues long before the monitoring program is put in place. This discussion during remedial design is also important to determine whether sufficient baseline data have been collected so that both the remedial action and long-term monitoring data can be easily compared to pre-remedy conditions.

At sediment sites, it is also frequently necessary to continue collecting background data from upstream or other reference areas away from the direct influence of the site. This can be especially important where there are uncertainties or potentially changing conditions in background areas, for example, where upstream urban storm water runoff or other possible continuing sources of contamination could impact a remedy.

During the remedial design phase, it is also important to develop a clear understanding of how the monitoring data will be used in the post-remediation decision process, and to ensure that reviews of the monitoring results are conducted in a timely fashion so additional actions can be taken when necessary. In this way, the monitoring data should become a key element of the decision process both in terms of whether the cleanup levels and RAOs are being met and whether additional management actions are warranted.

Highlight 8-2 lists some key questions the project manager should answer before developing a monitoring plan.

Highlight 8-2: Key Questions For Environmental Monitoring

- What is the purpose of the monitoring?
- Are detection limits adequate to meet the purpose of the monitoring?
- Are there likely to be other factors, such as non site-related releases, besides the cleanup that will influence the monitoring results, and are these well understood?
- How often should monitoring take place, and how long should it continue?
- Can the monitoring results be readily placed into searchable, electronic databases and made available to the project team and others?
- Is it clear who is responsible for reviewing the monitoring data and what the triggers are for identifying important trends (positive or negative) in the results?
- What are the most appropriate methods for analyzing the monitoring data? Should these be based on statistical tests or other quantitative analysis? Will there be sufficient data to support these statistical measures?
- Is there agreement on what actions will be taken based on the results of the monitoring data?
- How will the results be communicated to the public, and who is responsible for doing this?

Although sediment sites vary widely in size and complexity, monitoring typically requires a higher degree of planning than at some other types of sites for the following reasons:

- Sediment sites often involve more than one affected medium (e.g., sediment, surface water, biota, floodplain soils, and ground water) and multiple contaminants of concern;
- Contaminants at sediment sites are often from a variety of sources, some of which may be outside of the site in question;

- Sediment sites may require monitoring over large areas and in a variety of physical and ecological settings;

- Spatial and temporal variabilities of aquatic sediment and biota can be great; and

- Risk goals, for sites with bioaccumulative contaminants, generally relate to contaminants in biota and the relationship between contaminant levels in sediment and biota is frequently complex.

An especially important issue for project managers at large sites with more than one response action is the need to monitor both the effectiveness of individual sediment actions and the ability of achieving overall site RAOs. Frequently, the monitoring parameters at large sites are different. For example, where contaminants from multiple sources are indistinguishable, it may be necessary to use unique parameters for monitoring effectiveness of individual actions. However, it also may be very important to monitor parameters (i.e., some fish species), which may be responding to multiple sources or areas of a site.

8.2 SIX RECOMMENDED STEPS FOR SITE MONITORING

When developing a monitoring plan, it is important to review the ROD and supporting documents for the site. The ROD generally should contain numerical cleanup levels and/or action levels for sediment and sometimes for other media, and narrative RAOs that relate more directly to reducing risk. Generally, these form the basis of the monitoring plan. RODs or other site documents may also contain specific performance criteria or objectives for the short-term and long-term performance of the remedy that should be incorporated into the monitoring plan.

EPA s Monitoring Guidance (U.S. EPA 2004c) describes six key steps that are recommended in developing and implementing a monitoring plan. These steps are listed in Highlight 8-3 and explained briefly along with sediment site examples in the following text. This guidance was developed for use at all hazardous waste sites, not just Superfund sites, and therefore, uses the term site activity to apply to implementation of removal actions, remedial actions, ICs, or habitat mitigation.

Step 1. Identify Monitoring Plan Objectives

Generally, the most important element in developing an effective monitoring plan is for the project manager to identify clear and specific monitoring objectives. Identifying appropriate monitoring objectives normally includes examining the intended outcomes of the action and the methods used to achieve that outcome at the site. Inadequate or vague monitoring objectives can lead to uncertainty about why the monitoring is being conducted and how the data will be used. Furthermore, funding for monitoring is often limited. Specifying objectives can help to focus the experimental design and ensure that the most useful information is collected. When identifying monitoring objectives other than those already established in decision or enforcement documents, the project manager should involve participants from all concerned stakeholders (e.g., public, natural resource trustees, state agencies, potentially responsible parties).

Highlight 8-3: Recommended Six-Step Process for Developing and Implementing a Monitoring Plan

Step 1. Identify Monitoring Plan Objectives

- Evaluate the site activity
  - Identify the activity objectives
  - Identify the activity endpoints
  - Identify the activity mode of action
- Identify monitoring objectives
- Obtain stakeholder input

Step 2. Develop Monitoring Plan Hypotheses

- Develop monitoring conceptual models
- Develop monitoring hypotheses and questions

Step 3. Formulate Monitoring Decision Rules

Step 4. Design the Monitoring Plan

- Identify data needs
- Determine monitoring plan boundaries
- Identify data collection methods
- Identify data analysis methods
- Finalize the decision rules
- Prepare monitoring quality assurance project plans (QAPPs)

Step 5. Conduct Monitoring Analyses and Characterize Results

- Conduct data collection and analysis
- Evaluate results per the monitoring of data quality objectives (DQOs), developed in Steps 1-4, and revise data collection and analysis as necessary
- Characterize analytical results and evaluate relative to the decision rules

Step 6. Establish the Management Decision

- Monitoring results support the decision rule for site activity success
  - Conclude the site activity and monitoring
- Monitoring results do not support the decision rule for site activity success but are trending toward support
  - Continue the site activity and monitoring
- Monitoring results do not support the decision rule and are not trending toward support
  - Conduct causative factor and uncertainty analysis
  - Revise site activity and/or monitoring plan and implement

Source: U.S. EPA 2004c

Physical, chemical, and/or biological endpoints should be identified to help evaluate each monitoring objective. In general, physical and chemical endpoints are less costly and more easily measured and interpreted than biological endpoints and, therefore, may be more appropriate where quick decisions are needed. However, the ability of physical and chemical endpoints to quantify changes in ecological risk reliably may be less direct than biological measurements, for example where risk is due to direct contact with multiple contaminants. In this case, toxicity tests or bioassessments may provide an integrated measurement of the cumulative effects of all contaminants and, therefore, can be a better

assessment of ecological risks in some situations. Conversely, where the primary risk is due to humans and wildlife eating fish, chemical endpoints in fish may be most appropriate.

When identifying appropriate endpoints, it is important for the project manager to ensure that the measure employed matches the time frame established for the criteria. For example, acute toxicity tests quantify short-term effects on an organism; therefore, this type of test may be appropriate for operational monitoring (e.g., monitoring during remedial dredging), where it can be performed in a short period of time. Other biological endpoints, such as changes in species diversity, typically occur over long periods of time and may be more appropriate for use in a long-term monitoring program designed to look at ecological recovery. Although no single endpoint can quantify all possible risks, a combination of physical, chemical, and biological endpoints usually provides the best overall approach for measuring risk reduction.

> **Example:** In the ROD, EPA established a RAO of reducing polychlorinated biphenyl (PCB) concentrations in fish tissue to levels that would eliminate the need for a fish consumption advisory for PCBs (for this site, 0.05 ppm). To achieve this objective, EPA selected a cleanup level of 0.5 ppm total PCBs in sediment. The short-term objective of the monitoring program is to monitor PCB concentrations in sediment until the cleanup level is met and the long-term objective of the monitoring program is to monitor PCB concentrations in fish tissue until the RAO is met.

Step 2. Develop Monitoring Plan Hypotheses

Typically, monitoring hypotheses represent statements and/or questions about the relationship between a site activity, such as sediment remediation, and one or more expected outcomes (U.S. EPA 2004c). The development of the monitoring hypotheses is analogous to the problem formulation step (Step 1) of the DQO process (U.S. EPA 2000a). The monitoring hypothesis may be generally stated as The site activity has been successful in reaching its stated goals and objectives, or in question form, as Has the site activity reached its stated goals and objectives? As described in EPA s Monitoring Guidance (U.S. EPA 2004c), the concept of a monitoring conceptual model may be helpful in identifying and organizing appropriate hypotheses. This model, frequently a flow chart or graphical display, consists of a series of working hypotheses that identify the relationships between site activities and expected outcomes.

> **Example hypotheses:** The PCB concentration in sediment has reached the cleanup level of 0.5 ppm. The PCB concentration in fish tissue has reached the remedial goal of 0.05 ppm.

Step 3. Formulate Monitoring Decision Rules

Once monitoring objectives and hypotheses are agreed upon and stated explicitly, the next step should be to identify specific decision rules that will be used to assess whether the objectives are met. A decision rule is normally an if... then... statement that defines the conditions that would cause the decision maker to choose an action. In a monitoring plan, the decision rules should establish criteria for continuing, stopping, or modifying the monitoring or for taking an additional response action. Four main elements of a decision rule usually are: 1) the parameter of interest; 2) the expected outcome of the

remedial action; 3) an action level, the basis on which a monitoring decision will be made; and 4) alternative actions, the monitoring decision choices for the specified action (U.S. EPA 2004c).

Another factor the project manager should consider when developing decision rules is the time frame under which they will operate. For example, when dredging highly contaminated sediment, a real-time monitoring program could be established to analyze water samples before proceeding with the next day s dredging. In contrast, the time frame required to assess a long-term monitoring objective (e.g., to lower fish tissue concentrations) would be longer. In either case, the time frame should be explicitly stated and understood by all the participants.

> **Examples:** A decision rule could be established to require certain actions if suspended sediment or contaminant concentration in the surface water due to releases from dredging exceed certain criteria. A decision rule could be established to assess whether the sediment cleanup level of 0.5 ppm PCBs has been reached, defined as an average of 0.5 ppm PCBs in each of ten grids over the site. A decision rule could be established to assess whether progress is being made toward the remedial action objective of reduced PCB concentrations in fish tissue by establishing an interim goal of achieving 0.8 ppm in fish tissue within five years, after which monitoring frequency will be revisited. PCB concentrations in fish species A will be measured on a specific frequency (e.g., annually) that is commensurate with the relevant species uptake and depuration rates.

Step 4. Design the Monitoring Plan

The fourth recommended step for the project manager is to identify the monitoring design for collecting the necessary data. Design considerations include identifying data needs; determining monitoring boundaries (frequency, location, duration); identifying data collection methods; and identifying data analysis methods, including uncertainty analysis. EPA recommends that a systematic planning approach be used to develop acceptance or performance criteria for all environmental data collection and use. The Agency s DQO process is a planning approach normally appropriate for sediment sites (U.S. EPA 2000a). Quality assurance project plans (QAPPs) or their equivalent are also recommended for environmental data collection and use.

The spatial and temporal aspects of a monitoring plan typically define where and when to collect samples. In general, sampling locations should be based on the areal extent and magnitude of the contaminated sediment and the propensity for the contaminants to move, either through transport (e.g., remediation, natural events) or through the food chain. Generally, the more dynamic the conditions, the more frequently sampling is necessary to represent conditions accurately. However, a less costly alternative can be to use data endpoints which respond to cumulative, longer-term conditions, where appropriate. Additional factors that should be considered in establishing sampling locations include locations of baseline or pre-remediation sampling stations and spatial gradients in concentration. For example, generally greater sample density is needed where concentration gradients are high.

Selecting a statistical approach to use in evaluating the data is another important aspect of the monitoring program design. Data are sometimes collected in a manner that is incompatible with or insufficient for the statistical tests used to analyze the data. Although the amount of data needed to compare point-in-time data may be less than that needed to reliably establish a trend in data, both types of analyses may be needed to draw conclusions reliably. Especially for critical decisions, project managers

should seek expert advice in order to design a sampling program that will yield statistically defensible results. One potential method, power analysis, is described in *Biostatistical Analysis* (Zar 1999).

Another crucial element of developing a monitoring plan typically is cost. Generally, it is more cost-effective to collect less data, providing they are the correct or most useful data than it is to collect more of the wrong data. Following the key steps outlined in this guidance to design a monitoring plan should help project managers determine what are the correct data. Project managers may also find it useful to consider the use of indicator or surrogate parameters that correlate with those of primary interest, as a supplement to primary parameters that are especially costly or problematic to collect.

Finally, this step of monitoring plan development should ensure mechanisms are in place for modifying the plan based on new information.

> **Example:** From the remedial investigation data, we know that smallmouth bass spend most of their time in the contaminated area and spawn in late spring. The proposed sampling plan would consist of overlaying an unbiased sampling grid onto a map of the contaminated area of River X as well as in the areas upstream and downstream of the site. It is decided that 30 four-year old female bass will be collected in the early spring, before spawning, in each of these areas. A power analysis on baseline data indicated 20 fish would allow the project team to discern a 0.5 ppm or greater change in tissue concentration with 0.25 ppm confidence intervals (90 percent). However, given cost considerations, only ten samples will be analyzed immediately and the other 20 archived for further analyses pending the results.

Step 5. Conduct Monitoring Analyses and Characterize Results

The next recommended step in developing a monitoring plan includes data collection and analysis, evaluating analytical results, and addressing data deviations from the monitoring DQOs. At this point, the project manager should evaluate the data with regard to the monitoring hypotheses, the DQOs, and the monitoring decision rules developed in previous steps. At this step, the project manager should implement decision rules that may call for continuing, stopping, or modifying the monitoring or for taking additional action at the site.

In addition, the project manager should communicate data and results to the appropriate audiences. Frequently, the importance of communicating the results is underestimated. Because information is often provided to individuals with various levels of technical expertise, it should be comprehensible at multiple levels of understanding. Complex scientific data are not often easily understood by those without a technical background, and ineffective data communication often leads to skepticism about the conclusions. Therefore, it is important that the project manager consider the audience and present results in multiple formats. To those less familiar with the technical presentation of data, information can be presented in easily understood visual formats [e.g., geographic information system (GIS)]. This approach maximizes the effective dissemination of information to the greatest number of individuals, thus increasing the probability that the conclusions will be understood and believed.

> **Example:** At this point, three years of fish tissue data have been collected, analyzed, and validated. The decision criterion for this monitoring objective was to reduce the PCB

concentrations in fish tissue to 0.8 ppm within five years. The data show that after the third year, fish tissue concentrations have decreased significantly but the averages are still above 0.8 ppm; however, the higher levels are restricted to a relatively small area and most fish are below 0.8 ppm. The results are summarized and presented to the stakeholders. Due to the declining trend, the decision is made that the monitoring objective is expected to be met within five years and the fourth year monitoring effort can be skipped.

Step 6. Establish the Management Decision

The final step of a monitoring plan should be an extension of Step 5, to evaluate monitoring results and uncertainties and come to a decision regarding any changes in site activities or changes in the monitoring plans that may be appropriate at this time. Developing contingency plans in advance for actions that may need to be taken in response to monitoring results is recommended.

Example: Due to the declining trend, the decision is made that the monitoring objective is expected to be met within five years and the fourth year monitoring effort can be skipped.

An outline of the six steps and suggested subparts is shown in Highlight 8-2. It should be noted that the following outline essentially follows EPA s DQO process, with modification for ease of application to a contaminated sediment site. Project managers should refer to the DQO process guidance (U.S. EPA 2000a) to supplement this outline when preparing a sediment site monitoring program.

8.3 POTENTIAL MONITORING TECHNIQUES

This section provides a brief overview of the types of monitoring techniques and data endpoints that the project manager could consider when developing a monitoring plan. Selection of endpoints depends on the requirements in the decision and/or enforcement documents, as well as more general considerations related to the cleanup methods selected and the phase of the operation, as discussed in previous sections. For complex sites, frequently a combination of physical, chemical, and biological methods and a tiered monitoring plan (Highlight 8-3), is the best approach to determine whether a sediment remedy is meeting sediment cleanup levels, RAOs or goals, and associated performance criteria both during remedial action and in the long term. Monitoring, sampling, and analysis methods are being constantly improved based on research and increased field experience. Project managers should watch for new methods and, where they offer additional accuracy or lower cost but also allow for data to be compared to existing data, consider using them.

Generally, physical and chemical endpoints are easier to measure and interpret than biological endpoints. In the case of human health risk, chemical measurements are commonly used to assess risk. In contrast, measurement of the biological community is a direct but often complex measurement for monitoring changes in ecological risk. Caged organisms (e.g., *Macoma*, or mussels) at the site over a defined time frame can identify changes in bioavailable concentrations of many contaminants. Collection of fish and tissue analysis can address both human health and ecological response of the system, if both needs are considered during design of the sampling and analysis plan. The project manager should refer to EPA s Office of Water *Methods for Collection, Storage, and Manipulation of Sediments for Chemical*

and Toxicological Analyses (U.S. EPA 2001k) and *Managing and Sampling and Analyzing Contaminants in Fish and Shellfish* (U.S. EPA 2000h) for more detailed information.

Biological endpoints (e.g., toxicity tests) typically provide an integrated measurement of the cumulative effects of all contaminants. When using biological endpoints, it is important for the project manager to ensure the biological test employed fits the intended criteria. For example, acute toxicity tests are designed to quantify short-term effects on an organism; therefore, this type of test may be appropriate when monitoring for short-term impacts of a remedy. However, for toxicity tests to be useful, it is important to have demonstrated during site characterization a significant relationship between the contaminant and toxicity. Other biological endpoints, such as changes in species diversity, typically occur over long periods of time and may be more appropriate for use in a long-term monitoring program designed to look at ecological recovery. While no single endpoint can quantify all possible risks, project managers should consider a combination of physical, chemical, and biological endpoints to provide the best overall approach for assessing the long-term effectiveness of a remedial action in achieving the RAOs.

8.3.1 Physical Measurements

Physical testing at a site may include measurements of erosion and/or deposition of sediment, ground water advective flow, particle size, surface water flow rates, and sediment homogeneity/heterogeneity. Potential types of physical data and their uses include the following:

- *Sediment Geophysical Properties:* Uses include fate and transport modeling, determination of contaminant bioavailability, and habitat characteristics of post-cleanup sediment surface;

- *Water Column Physical Measurements (e.g., turbidity, total suspended solids):* Uses include monitoring the amount of sediment resuspended during dredging and during placement of in-situ caps;

- *Bathymetry Data:* Uses include evaluating post-capping or post-dredging bottom elevations for comparison to design specifications, and evaluating sediment stability during natural recovery;

- *Side Scan Sonar Data:* Uses include remote sensing to monitor the distribution of sediment types and bedforms;

- *Settlement Plate Data:* Uses include monitoring changes in cap thickness over time and measuring cap consolidation;

- *Sediment Profile Camera Data:* Uses include monitoring of changes in thin layering within sediment profiles, sediment grain sizes, bioturbation and oxidation depths, and the presence of gas bubbles; and

- *Subbottom Profiler Data:* Uses include remote sensing measurement of changes in sediment surface and subsurface layers, bioturbation and oxidation depths, and presence of gas bubbles.

8.3.2 Chemical Measurements

Chemical testing may include sediment chemistry (both the upper biological surficial zone and/or deeper sediment), evaluating biodegradation, contaminant partitioning to the pore water, and concentrations of total organic carbon. Potential sampling tools and environmental monitoring methods used in support of chemical measurements include the following:

- *Sediment Grab Samplers:* Uses include collection of samples for measurement of surface sediment chemistry;
- *Coring Devices (e.g., vibracore, gravity piston, or drop tube samplers):* Uses include obtaining a vertical profile of sediment chemistry, or detection of contaminant movement through a cap or through a layer of naturally deposited clean sediment;
- *Direct Water Column Measurements (probes):* Uses include measurement of parameters such as pH and dissolved oxygen in the water column;
- *Surface Water Samplers:* Uses include measurement of chemical concentrations (dissolved and particulate) in water or contaminant releases to the water column during construction;
- *Semi-Permeable Membrane Devices:* Uses include measurement of dissolved contaminants at the sediment-water interface; and
- *Seepage Meters:* Uses include measurement of contaminant flux into the water column.

8.3.3 Biological Measurements

Biological testing can include toxicity bioassays, examining changes in the biological assemblages at sites, either to document problems or evaluate restoration efforts, and/or determining toxicant bioaccumulation and food chain effects. Potential types of biological monitoring data and their uses also include the following:

- *Benthic Community Analysis:* Uses include evaluation of population size and diversity, and monitoring of recovery following remediation;
- *Toxicity Testing:* Uses include measurement of acute and long-term lethal or sublethal effects of contaminants on organisms to help establish a protective range of remediation goals;
- *Tissue Sampling:* Uses include measurement of bioaccumulation, modeling trophic transfer potential, and estimating food web effects;
- *Caged Fish/Invertebrate Studies:* Uses include monitoring change in uptake of contaminants by biota from the sediment or water column to measure the effect of the remedy on bioaccumulation rates; and

- *Sediment Profile Camera Studies:* Uses include indirect measurement of macroinvertebrate recolonization, for example, measuring population density of polychaetes by counting the number of burrow tubes per linear centimeter along the sediment-water interface.

The interpretation of fish tissue results and their relationship to sediment contaminant levels can be especially complex. Potential complications may relate to questions of home range, lipid content, age, feeding regime, contaminant excretion rates, and other factors. Especially at low contaminant concentrations, these variabilities can make understanding the relationship between trends in sediment and biota concentrations especially difficult.

Fact sheets are under development at EPA concerning biological monitoring at sediment sites, including:

- An approach for using biological measures to evaluate the short-term and long-term remedial effects at Superfund sites; and

- An approach for using bioaccumulation information from biota sediment accumulation factors (BSAFs) and food chain models to assess ecological risks and to develop sediment remediation goals.

8.4 REMEDY-SPECIFIC MONITORING APPROACHES

The following sections discuss monitoring issues particular to MNR, in-situ capping, and dredging or excavation. Many sediment remedies involve a combination of cleanup methods, and for these remedies, the monitoring plan will likely include a combination of techniques to measure short- and long-term success. At many sediment sites, monitoring of source control actions is an important first step.

8.4.1 Monitoring Natural Recovery

Monitoring of natural recovery remedies often tests the hypothesis that natural processes are continuing to operate at a rate that is expected to reduce contaminant concentrations in appropriate media such as biota to an acceptable level in a reasonable time frame. Other measures of reduced risk may also be appropriate for a site. In most cases, monitoring involves measuring natural processes indirectly or measuring the effects of those processes. As a sound strategy for monitoring natural recovery the project manager should consider the following:

- Monitoring direct or indirect measures of natural processes (e.g., sediment accumulation rates, degradation products, sediment and contaminant transport);

- Monitoring contaminant levels in surface sediment, surface water, and biota; and

- Monitoring measures of biota recovery (e.g., sediment toxicity, benthic community size and/or diversity).

When monitoring natural recovery, it is usually important to monitor sediment, surface water, and biota. The water column is typically important because it integrates the flux of contaminants from sediment and is not typically subject to as large a spatial variability as sediment. Biota monitoring is important because it is frequently directly related to risk.

Monitoring continued effectiveness of source control actions can be especially important at MNR sites. Depending on the quality of existing trend data, MNR remedies may require more intensive monitoring early in the recovery period, which may be relaxed if predicted recovery rates are being attained. Also, there may be a need to collect additional data after an intensive disturbance event.

EPA s Science Advisory Board (SAB), in its May 2001 report, *Monitored Natural Attenuation: USEPA Research Program - An EPA Science Advisory Board Review* (U.S. EPA 2001j), Section 3.4, Summary of Major Research Recommendations, indicates the need for the development of additional monitoring methods to quantify attenuation mechanisms, contaminated sediment transport processes, and bioaccumulation to support footprint documentation and analysis of permanence. EPA is aware of these research needs and plans to address some of these topics in ongoing and future work.

For areas that may be subject to sediment disruption, the project manager should conduct more extensive monitoring when specified disruptive events (e.g., storms or flow stages of a specified recurrence interval or magnitude) occur to evaluate whether buried contaminated sediment has been disturbed or transported and the extent of contaminant release contaminants and increased exposure. The project manager should design the monitoring plan to handle the relatively quick turnaround times needed to effectively monitor disruptive events. However, interpretation of these data in terms of increased risk should take into account the length of time organisms may be exposed to higher levels of contaminant concentrations.

The project manager should include periodic comparisons of monitoring data to rates of recovery expected for the site in an MNR monitoring program. Where predictions were based on modeling, the project manager should make monitoring results available to the modeling team or other researchers to conduct field validation of the model. Where contingency remedies or triggers for additional work are part of a remedy decision, the project manager should design the monitoring plan to help determine whether those triggers are met. For example, a contingency for additional evaluation or additional work may be triggered by an increasing or insufficiently decreasing trend in contaminant concentrations in sediment, surface water, or biota at specified locations. Where contingencies for additional work are triggered, the project manager may need to include measures such as additional source control, additional ICs, the placement of a thin layer of clean sediment to enhance natural recovery, or an active cleanup (i.e., dredging or capping).

Following attainment of cleanup levels and remedial action objectives, monitoring may still be needed at some MNR sites. For sites where natural recovery is based on burial with clean sediment, continued monitoring may be necessary to assess whether buried contaminants remain buried after an intensive disturbance event. This monitoring should continue until the project team has reasonable confidence in the continued effectiveness of the remedy.

8.4.2 Monitoring In-Situ Capping

Remedial action monitoring for capping generally includes monitoring of construction and placement, and of cap performance during an initial period. It may also include monitoring of broader RAOs such as recovery of the benthic community or of contaminant levels in fish. Long-term monitoring for capping generally includes continued periodic monitoring of cap performance and maintenance activities, and continued monitoring of RAOs. In some cases (e.g., Fund-lead sites) it may be necessary to distinguish monitoring that is part of remedial action from monitoring that is part of O&M. This should be a site-specific decision. Highlight 8-4 lists sample elements of monitoring an in-situ cap. It is important to note that not all of these elements may be needed for every cap. In general, cap monitoring should be designed so that elements can be phased back or eliminated if the remedy is performing as expected and there has been no large-scale disturbance of the cap.

As shown in Highlight 8-4, a variety of monitoring equipment and methods can be used for capping projects during both remedial action and long-term monitoring. The extent of any necessary monitoring should be a site-specific decision and also may depend on decision and enforcement document requirements. In general, bathymetric surveys to determine cap thickness and stability over time, sediment core chemistry (including surface sediment and upper portion of cap) to confirm physical and chemical isolation and test for recontamination, and some form of biological monitoring are useful for most capping projects. Specialized equipment, such as seepage meters, diffusion samplers (e.g., peepers and semi-permeable membrane devices), sediment profile cameras, sediment traps, or use of caged organisms, may also be useful in some cases.

Construction monitoring for capping normally is designed to measure whether design plans and specifications are followed in the placement of the cap and to monitor the extent of any contaminant releases during cap placement. During construction, monitoring results can be used to identify modifications to design or construction techniques needed to meet unavoidable field constraints. Construction monitoring frequently includes interim and post-construction cap material placement surveys. Appropriate methods for monitoring cap placement include bathymetric surveys, sediment cores, sediment profiling camera, and chemical resuspension monitoring for contaminants. For some sites, visual observation in shallow waters or surface visual aids, such as viewing tube or diver observations, can also be useful.

Biological monitoring in the initial period following cap construction may include monitoring of the benthic community that may recolonize the capped site and the bioturbation behavior of bottom-dwelling organisms. Where contaminants are bioaccumulative, fish or other biota edible tissue or whole body monitoring are also likely to be needed.

Long-term monitoring of in-situ capping sites typically is important to ensure that the cap is not being eroded or significantly compromised (e.g., penetrated by submerged aquatic vegetation, ground water recharge, or bioturbation) and that chemical contaminant fluxes that ultimately do move through the cap to surface water do so at the low projected rate and concentration. It may be also desirable to include ongoing monitoring for recontamination of the cap surface and non-capped areas from other sources.

Highlight 8-4: Sample Cap Monitoring Phases and Elements

Monitoring Phase	Element	Component	Analysis	Frequency Location
Cap Construction	Cap material quality	Cap material sampling	Physical properties	5% of loads
	Cap thickness and areal extent	Bathymetry Subbottom profile	Thickness of cap layers Areal extent of cap	Baseline Initial placement Final surveys over entire area
		Sediment profile camera	Thickness of cap layers	Baseline Initial placement Defined grid for remaining cells
		Cores	Layer thickness and physical properties Chemical properties for baseline	Defined grid
	Sediment resuspension	Plume tracking Acoustic doppler current profile (ADCP) Water column samples	Suspended sediment Water column chemistry	5% of load placements
	Sediment displacement	Sediment samples	Chemical properties of sediment	Sediment bed near cap boundaries
Cap Performance	Recolonization	Sediment profile camera Benthic community analysis	Layer thickness Re-colonization, population size, and diversity	Defined grid - frequency determined by local information about recolonization rates
	Physical isolation	Subbottom profile Bathymetry	Layer thickness	Annual checks in some cases Surveys over entire area every five years, modify as needed
	Chemical isolation	Cores Peepers, seepage meters, if needed	Physical properties Sediment chemistry, pore water chemistry	Defined grid every five years, modify as needed
Severe Event Response	Cap integrity	Subbottom profile Sediment profile camera Cores		Following major storms or earthquakes

For areas that may be subject to cap disruption, more extensive monitoring should be triggered when specified disruptive events (e.g., storms, flow stages, or earthquakes of a specified recurrence interval or magnitude) occur, to evaluate whether the cap was disturbed and whether any disturbance caused a significant release of contaminants and increased risk. Additional monitoring for the effects of tidal and wave pumping and boat propeller wash is also recommended where these are expected to be important factors. In general, the project manager should monitor cap integrity both routinely and following storm/flood events that approach the design storm magnitude envisioned by the cap s engineers. As for other types of sediment remedies, the project manager should design the monitoring plan to handle the relatively quick turnaround times needed to effectively monitor disruptive events.

Cap maintenance is generally limited to the repair and replenishment of the erosion protection layer in potentially high erosion areas where this is necessary. Project managers should consider the ability to detect and respond quickly to a loss of the erosion protection layer when evaluating a capping alternative. Seasonal limitations, such as ice formation or closure of navigation structures (locks), can affect the ability to monitor and maintain in-situ caps and should be accounted for in monitoring plans.

Capping remedies frequently include provisions for actions to be taken in the case that one or more cap functions are not being met. Options for modifying the cap design may or may not be available. If monitoring shows that the stabilization component is being eroded by events of lesser magnitude than planned, or the erosive energy at the capping site was underestimated, then eroded material can be replaced with more erosion-resistant cap material. If monitoring indicates that bottom-dwelling organisms are penetrating the cap and causing unacceptable releases of contaminants, then project managers should consider placing additional cap material on top of the cap to maintain isolation of the contaminated sediment. These types of management options are usually feasible where additional cap thickness, and the resulting decrease in water depths at the site, does not conflict with other waterway uses. Where a cap has been closely designed to a thickness that will not limit waterway use (i.e., recreational or commercial navigation), the options for modifying a cap design after construction can be limited.

8.4.3 Monitoring Dredging or Excavation

Monitoring for dredging or excavation remedies generally includes construction and operational monitoring of the dredging or excavation, transport, dewatering, any treatment, transport, and any on-site disposal placement. Following dredging or excavation, the residual sediment contamination should also be monitored. Additional monitoring following sediment removal may include monitoring of sediment toxicity or benthic community recovery or, for bioaccumulative contaminants, tissue concentrations in fish or shellfish, as well as continued monitoring of any on-site disposal facilities and monitoring sediment and/or biota for recontamination.

Depending on the levels of contamination and the selected methods of dredging/excavation, transport, treatment or disposal, potential construction and operational monitoring may include the following:

- Surface water monitoring at the dredging site and any in-water disposal sites (e.g., total suspended solids, total and dissolved contaminant concentrations, caged fish toxicity, caged mussel intake);

- Dredging/excavation residual monitoring at the sediment surface to determine whether cleanup levels are met;
- Effluent quality monitoring after sediment dewatering and/or treatment;
- Air monitoring at the dredge, transport, on-site disposal, and treatment sites; and
- On-site disposal monitoring of dredged sediment or treatment residuals.

A thorough monitoring plan will normally enable project managers to make design or construction changes to ensure that the spread of contamination to uncontaminated areas of the water body, sensitive habitats, or adjacent human populations is minimized during dredging, transport, treatment, or disposal. Depending on the contaminants present and their tendency to volatilize or bioaccumulate, the project manager should consider water, air, and biological sampling in the monitoring plan.

Generally, a monitoring plan for dredging should include collecting data to test the effectiveness of silt curtains, dredge operating practices, and any other measures used to control sediment resuspension or sediment or contaminant transport. In most cases the project manager should include sampling upgradient of the dredging operation and both inside and outside of any containment structures. Generally this sampling should also include dissolved compounds in the water column, although in some cases it may be a appropriate to use a tiered approach with analysis of dissolved compounds triggered by exceedances of threshold criteria for total compounds or for suspended solids. Also, where contaminants may be volatile, project managers should consider the need for air sampling. At highly contaminated sites, it may be necessary for the project manager to conduct a pilot study on a small area to determine if the sediment can be removed without causing unacceptable risks to adjacent human populations or adjacent benthic habitat. This information can help to determine what containment barriers or dredging methods work best and what performance standards are achievable at the site. The project manager should compare monitoring results with baseline data for contaminant concentrations in water and, where appropriate, in air. This should ensure that effects due to dredging may be separated and evaluated from natural perturbations caused by tides and storms. The project manager should develop contingency plans to guide changes in operation where performance standards are not met.

Following dredging, it is usually essential for project managers to conduct monitoring to determine whether cleanup levels in sediment are achieved. Initial sampling should be analyzed rapidly, so that contingency actions, such as additional dredging, excavation, or backfilling, can be implemented quickly if cleanup levels have not been met.

Following sediment removal, it is usually necessary for the project manager to conduct long-term monitoring to ensure that the dredged or excavated area is not recontaminated by additional sources or by disturbance of any residuals that remain above cleanup levels. Long-term monitoring is usually necessary to provide data to determine whether RAOs are met, and may be necessary for a period of time following remedial action to provide confidence that the objectives will remain met.

If an in-water or upland disposal facility is constructed on site as part of the remedy, it should also be monitored to ensure that it remains intact and that there are no unacceptable contaminant releases in the long term. Monitoring is recommended to determine whether contaminants are leaking through the bottom or walls of the on-site confined disposal facility (CDF) or landfill, and to determine if any surface

cap remains intact to ensure protection from infiltration. Depending on the type of disposal site and the nature of the contamination, long-term disposal site monitoring may include the following:

- Seepage from the CDF containment cells to surrounding surface water;
- Ground water monitoring;
- Surface water runoff monitoring;
- Disposal area cap integrity monitoring; and
- Revegetation or recolonization by plant and animal communities monitoring, and their potential uptake of contaminants.

Highlight 8-5 lists important points to remember related to monitoring sediment sites.

Highlight 8-5: Some Key Points to Remember About Monitoring Sediment Sites

- Presentation of a monitoring plan is important for all types of sediment remedies, both during and following any physical construction, to ensure that exposure pathways and risks have been adequately managed
- Development of monitoring plans should follow a systematic planning process that identifies monitoring objectives, decision criteria, endpoints, and data collection, and data interpretation methods
- Before implementing a remedial action, project managers should determine if data adequate baseline data exists for comparison to future monitoring data and, if not, collect additional data
- Where background conditions may be changing or where uncertainty exists concerning continuing off-site contaminant contributions to a site, it may be necessary to continue collecting data from upstream or other reference areas for comparison to site monitoring data
- Monitoring needs include both monitoring of construction and operation and monitoring intended to measure whether cleanup levels in sediment and remedial action objectives for biota or other media have been met
- Monitoring plans should be designed to evaluate whether performance standards of the remedial action are being met and should be flexible enough to allow revision if operating procedures are revised
- Field measurement methods and quick turnaround analysis methods with real-time feedback are especially useful during capping and dredging operations to identify potential problems which may be corrected as the work progresses
- After completion of remedial action, long-term monitoring should be used to identify recontamination, to assess continued containment of buried or capped contaminants, and to monitor dredging residuals and on-site disposal facilities

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CONTAMINATED SEDIMENT REMEDIATION GUIDANCE FOR HAZARDOUS WASTE SITES:

APPENDIX A: PRINCIPLES FOR MANAGING CONTAMINATED SEDIMENT RISKS AT HAZARDOUS WASTE SITES

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460
Feb. 12, 2002

OFFICE OF
SOLID WASTE AND EMERGENCY
RESPONSE

OSWER Directive 9285.6-08

MEMORANDUM

SUBJECT: Principles for Managing Contaminated Sediment Risks at Hazardous Waste Sites

FROM: Marianne Lamont Horinko */s/ Marianne Lamont Horinko*
Assistant Administrator

TO: Superfund National Policy Managers, Regions 1 - 10
RCRA Senior Policy Advisors, Regions 1 - 10

I. PURPOSE

This guidance will help EPA site managers make scientifically sound and nationally consistent risk management decisions at contaminated sediment sites. It presents 11 risk management principles that Remedial Project Managers (RPMs), On-Scene Coordinators (OSCs), and RCRA Corrective Action project managers should carefully consider when planning and conducting site investigations, involving the affected parties, and selecting and implementing a response.

This guidance recommends that EPA site managers make risk-based site decisions using an iterative decision process, as appropriate, that evaluates the short-term and long-term risks of all potential cleanup alternatives consistent with the National Oil and Hazardous Substances Pollution Contingency Plan s (NCP s) nine remedy selection criteria (40 CFR Part 300.430). EPA site managers are also encouraged to consider the societal and cultural impacts of existing sediment contamination and of potential remedies through meaningful involvement of affected stakeholders.

This guidance also responds in part to the recommendations contained in the National Research Council (NRC) report discussed below.

II. BACKGROUND

On March 26, 2001, the NRC published a report entitled *A Risk Management Strategy for PCB-Contaminated Sediments*. Although the NRC report focuses primarily on assessment and remediation of PCB-contaminated sediments, much of the information in that report is applicable to other contaminants. Site managers are encouraged to read the NRC report, which may be found at http://www.nrc.edu.

In addition to developing these principles, OSWER, in coordination with other EPA offices (Office of Research and Development, Office of Water, and others) and other federal agencies (Department of Defense/U.S. Army Corps of Engineers, Department of Commerce/National Oceanic and Atmospheric Administration, Department of the Interior/U.S. Fish and Wildlife Service, and others) is developing a separate guidance, *Contaminated Sediment Remediation Guidance for Hazardous Waste Sites* (Sediment Guidance). The Sediment Guidance will provide more detailed technical guidance on the process that Superfund and RCRA project managers should use to evaluate cleanup alternatives at contaminated sediment sites.

While this directive applies to all contaminants at sediment sites addressed under CERCLA or RCRA, its implementation at particular sites should be tailored to the size and complexity of the site, to the magnitude of site risks, and to the type of action contemplated. These principles can be applied within the framework of EPA s existing statutory and regulatory requirements.

III. RISK MANAGEMENT PRINCIPLES

1. Control Sources Early.

As early in the process as possible, site managers should try to identify all direct and indirect continuing sources of significant contamination to the sediments under investigation. These sources might include discharges from industries or sewage treatment plants, spills, precipitation runoff, erosion of contaminated soil from stream banks or adjacent land, contaminated groundwater and non-aqueous phase liquid contributions, discharges from storm water and combined sewer outfalls, upstream contributions, and air deposition.

Next, site managers should assess which continuing sources can be controlled and by what mechanisms. It may be helpful to prioritize sources according to their relative contributions to site risks. In the identification and assessment process, site managers should solicit assistance from those with relevant information, including regional Water, Air, and PCB Programs (where applicable); state agencies (especially those responsible for setting Total Maximum Daily Loads (TMDLs) and those that issue National Pollutant Discharge Elimination

System (NPDES) permits); and all Natural Resource Trustees. Local agencies and stakeholders may also be of assistance in assessing which sources can be controlled.

Site managers should evaluate the potential for future recontamination of sediments when selecting a response action. If a site includes a source that could result in significant recontamination, source control measures will likely be necessary as part of that response action. However, where EPA believes that the source can be controlled, or where sediment remediation will have benefits to human health and/or the environment after considering the risks caused by the ongoing source, it may be appropriate for the Agency to select a response action for the sediments prior to completing all source control actions. This is consistent with principle 5 below, which indicates that it may be necessary to take phased or interim actions (e.g., removal of a hot spot that is highly susceptible to downstream movement or dispersion of contaminants) to prevent or address environmental impacts or to control human exposures, even if source control actions have not been undertaken or completed.

2. Involve the Community Early and Often.

Contaminated sediment sites often involve difficult technical and social issues. As such, it is especially important that a project manager ensure early and meaningful community involvement by providing community members with the technical information needed for their informed participation. Meaningful community involvement is a critical component of the site characterization, risk assessment, remedy evaluation, remedy selection, and remedy implementation processes. Community involvement enables EPA to obtain site information that may be important in identifying potential human and ecological exposures, as well as in understanding the societal and cultural impacts of the contamination and of the potential response options. The NRC report (p. 249) recommends that increased efforts be made to provide the affected parties with the same information that is to be used by the decision-makers and to include, to the extent possible, all affected parties in the entire decision-making process at a contaminated site. In addition, such information should be made available in such a manner that allows adequate time for evaluation and comment on the information by all parties. Through Technical Assistance Grants and other mechanisms, project managers can provide the community with the tools and information necessary for meaningful participation, ensuring their early and continued involvement in the cleanup process.

Although the Agency has the responsibility to make the final cleanup decision at CERCLA and RCRA sites, early and frequent community involvement facilitates acceptance of Agency decisions, even at sites where there may be disagreement among members of the community on the most appropriate remedy.

Site managers and community involvement coordinators should take into consideration the following six practices, which were recently presented in OSWER Directive 9230.0-99 *Early*

and Meaningful Community Involvement (October 12, 2001). This directive also includes a list of other useful resources and is available at http://www.epa.gov/superfund/pubs.htm.

(1) Energize the community involvement plan.
(2) Provide early, proactive community support.
(3) Get the community more involved in the risk assessment.
(4) Seek early community input on the scope of the remedial investigation/feasibility study (RI/FS).
(5) Encourage community involvement in identification of future land use.
(6) Do more to involve communities during removals.

3. Coordinate with States, Local Governments, Tribes, and Natural Resource Trustees.

Site managers should communicate and coordinate early with states, local governments, tribes, and all Natural Resource Trustees. By doing so, they will help ensure that the most relevant information is considered in designing site studies, and that state, local, tribal, and trustee viewpoints are considered in the remedy selection process. For sites that include waterbodies where TMDLs are being or have been developed, it is especially important to coordinate site investigations and monitoring or modeling studies with the state and with EPA s water program. In addition, sharing information early with all interested parties often leads to quicker and more efficient protection of human health and the environment through a coordinated cleanup approach.

Superfund s statutory mandate is to ensure that response actions will be protective of human health and the environment. EPA recognizes, however, that in addition to EPA s response action(s), restoration activities by the Natural Resource Trustees may be needed. It is important that Superfund site managers and the Trustees coordinate both the EPA investigations of risk and the Trustee investigations of resource injuries in order to most efficiently use federal and state resources and to avoid duplicative efforts.

Additional information on coordinating with Trustees may be found in OSWER Directive 9200.4-22A *CERCLA Coordination with Natural Resource Trustee*s (July 1997), in the 1992 ECO Update *The Role of Natural Resource Trustees in the Superfund Process* http://www.epa.gov/superfund/programs/risk/tooleco.htm), and in the 1999 OSWER Directive 9285.7-28 P *Ecological Risk Assessment and Risk Management Principles for Superfund Sites* (also available at the above web site). Additional information on coordinating with states and tribes can be found in OSWER Directive 9375.3-03P *The Plan to Enhance the Role of States and Tribes in the Superfund Program* (http://www.epa.gov/superfund/states/strole/index.htm).

4. Develop and Refine a Conceptual Site Model that Considers Sediment Stability.

A conceptual site model should identify all known and suspected sources of contamination, the types of contaminants and affected media, existing and potential exposure pathways, and the known or potential human and ecological receptors that may be threatened. This information is frequently summarized in pictorial or graphical form, backed up by site-specific data. The conceptual site model should be prepared early and used to guide site investigations and decision-making. However, it should be updated periodically whenever new information becomes available, and EPA s understanding of the site problems increases. In addition, it frequently can serve as the centerpiece for communication among all stakeholders.

A conceptual site model is especially important at sediment sites because the interrelationship of soil, surface and groundwater, sediment, and ecological and human receptors is often complex. In addition, sediments may be subject to erosion or transport by natural or man-made disturbances such as floods or engineering changes in a waterway. Because sediments may experience temporal, physical, and chemical changes, it is especially important to understand what contaminants are currently available to humans and wildlife, and whether this is likely to change in the future under various scenarios. The risk assessor and project manager, as well as other members of the site team, should communicate early and often to ensure that they share a common understanding of the site and the basis for the present and future risks. The May 1998 EPA *Guidelines for Ecological Risk Assessment* (Federal Register 63(93) 26846-26924, http://www.epa.gov/superfund/programs/risk/tooleco.htm), the 1997 Superfund Guidance *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments* (EPA 540-R-97-006, also available at the above web site), and the 1989 *Risk Assessment Guidance for Superfund (RAGS), Volume 1, Part A* (EPA 540-1-89-002, http://www.epa.gov/superfund/programs/risk/ragsa) provide guidance on developing conceptual site models.

5. Use an Iterative Approach in a Risk-Based Framework.

The NRC report (p. 52) recommends the use of a risk-based framework based on the one developed by the Presidential/Congressional Commission on Risk Assessment and Risk Management (PCCRARM, 1997, *Framework for Environmental Health Risk Management*, Vol. 1, as cited by NRC 2001). However, as recognized by the NRC (p. 60): The framework is intended to supplement, not supplant, the CERCLA remedial process mandated by law for Superfund sites.

Although there is no universally accepted, well-defined risk-based framework or strategy for remedy evaluation at sediment sites, there is wide-spread agreement that risk assessment should play a critical role in evaluating options for sediment remediation. The Superfund program uses a flexible, risk-based framework as part of the CERCLA and NCP process to adequately characterize ecological and human health site risks. The guidances used by the

RCRA Corrective Action program (http://www.epa.gov/correctiveaction/resource/guidance) also recommend a flexible risk-based approach to selecting response actions appropriate for the site.

EPA encourages the use of an iterative approach, especially at complex contaminated sediment sites. As used here, an iterative approach is defined broadly to include approaches which incorporate testing of hypotheses and conclusions and foster re-evaluation of site assumptions as new information is gathered. For example, an iterative approach might include pilot testing to determine the effectiveness of various remedial technologies at a site. As noted in the NRC report (p. 66): "Each iteration might provide additional certainty and information to support further risk-management decisions, or it might require a course correction."

An iterative approach may also incorporate the use of phased, early, or interim actions. At complex sediment sites, site managers should consider the benefits of phasing the remediation. At some sites, an early action may be needed to quickly reduce risks or to control the ongoing spread of contamination. In some cases, it may be appropriate to take an interim action to control a source, or remove or cap a hot spot, followed by a period of monitoring in order to evaluate the effectiveness of these interim actions before addressing less contaminated areas.

The NRC report makes an important point when it notes (p. 256): The committee cautions that the use of the framework or other risk-management approach should not be used to delay a decision at a site if sufficient information is available to make an informed decision. Particularly in situations in which there are immediate risks to human health or the ecosystem, waiting until more information is gathered might result in more harm than making a preliminary decision in the absence of a complete set of information. The committee emphasizes that a wait-and-see or do-nothing approach might result in additional or different risks at a site.

6. Carefully Evaluate the Assumptions and Uncertainties Associated with Site Characterization Data and Site Models.

The uncertainties and limitations of site characterization data, and qualitative or quantitative models (e.g., hydrodynamic, sediment stability, contaminant fate and transport, or food-chain models) used to extrapolate site data to future conditions should be carefully evaluated and described. Due to the complex nature of many large sediment sites, a quantitative model is often used to help estimate and understand the current and future risks at the site and to predict the efficacy of various remedial alternatives. The amount of site-specific data required and the complexity of models used to support site decisions should depend on the complexity of the site and the significance of the decision (e.g., level of risk, response cost, community interest). All new models and the calibration of models at large or complex sites should be peer-reviewed consistent with the Agency s peer review process as described in its Peer Review Handbook (EPA 100-B-00-001, http://www.epa.gov/ORD/spc/2peerrev.htm).

Site managers should clearly describe the basis for all models used and their uncertainties when using the predicted results to make a site decision. As recognized by the NRC report (p. 65), however, Management decisions must be made, even when information is imperfect. There are uncertainties associated with every decision that need to be weighed, evaluated, and communicated to affected parties. Imperfect knowledge must not become an excuse for not making a decision.

7. Select Site-specific, Project-specific, and Sediment-specific Risk Management Approaches that will Achieve Risk-based Goals.

EPA s policy has been and continues to be that there is no presumptive remedy for any contaminated sediment site, regardless of the contaminant or level of risk. This is consistent with the NRC report s statement (p. 243) that There is no presumption of a preferred or default risk-management option that is applicable to all PCB-contaminated-sediment sites. At Superfund sites, for example, the most appropriate remedy should be chosen after considering site-specific data and the NCP s nine remedy selection criteria. All remedies that may potentially meet the removal or remedial action objectives (e.g., dredging or excavation, in-situ capping, in-situ treatment, monitored natural recovery) should be evaluated prior to selecting the remedy. This evaluation should be conducted on a comparable basis, considering all components of the remedies, the temporal and spatial aspects of the sites, and the overall risk reduction potentially achieved under each option.

At many sites, a combination of options will be the most effective way to manage the risk. For example, at some sites, the most appropriate remedy may be to dredge high concentrations of persistent and bioaccumulative contaminants such as PCBs or DDT, to cap areas where dredging is not practicable or cost-effective, and then to allow natural recovery processes to achieve further recovery in net depositional areas that are less contaminated.

8. Ensure that Sediment Cleanup Levels are Clearly Tied to Risk Management Goals.

Sediment cleanup levels have often been used as surrogates for actual remediation goals (e.g., fish tissue concentrations or other measurable indicators of exposure relating to levels of acceptable risk). While it is generally more practical to use measures such as contaminant concentrations in sediment to identify areas to be remediated, other measures should be used to ensure that human health and/or ecological risk reduction goals are being met. Such measures may include direct measurements of indigenous fish tissue concentrations, estimates of wildlife reproduction, benthic macroinvertebrate indices, or other effects endpoints as identified in the baseline risk assessment.

As noted in the NRC report (p. 123), The use of measured concentrations of PCBs in fish is suggested as the most relevant means of measuring exposures of receptors to PCBs in contaminated sediments. For other contaminants, other measures may be more appropriate.

For many sites, achieving remediation goals, especially for bioaccumulative contaminants in biota, may take many years. Site monitoring data and new scientific information should be considered in future reviews of the site (e.g., the Superfund five-year review) to ensure that the remedy remains protective of human health and the environment.

9. Maximize the Effectiveness of Institutional Controls and Recognize their Limitations.

Institutional controls, such as fish consumption advisories and waterway use restrictions, are often used as a component of remedial decisions at sediment sites to limit human exposures and to prevent further spreading of contamination until remedial action objectives are met. While these controls can be an important component of a sediment remedy, site managers should recognize that they may not be very effective in eliminating or significantly reducing all exposures. If fish consumption advisories are relied upon to limit human exposures, it is very important to have public education programs in place. For other types of institutional controls, other types of compliance assistance programs may also be needed (e.g., state/local government coordination). Site managers should also recognize that institutional controls seldom limit ecological exposures. If monitoring data or other site information indicates that institutional controls are not effective, additional actions may be necessary.

10. Design Remedies to Minimize Short-term Risks while Achieving Long-term Protection.

The NRC report notes (p. 53) that: Any decision regarding the specific choice of a risk management strategy for a contaminated sediment site must be based on careful consideration of the advantages and disadvantages of available options and a balancing of the various risks, costs, and benefits associated with each option. Sediment cleanups should be designed to minimize short-term impacts to the extent practicable, even though some increases in short-term risk may be necessary in order to achieve a long-lasting solution that is protective. For example, the long-term benefits of removing or capping sediments containing persistent and bioaccumulative contaminants often outweigh the additional short-term impacts on the already-affected biota.

In addition to considering the impacts of each alternative on human health and ecological risks, the short-term and long-term impacts of each alternative on societal and cultural practices should be identified and considered, as appropriate. For example, these impacts might include effects on recreational uses of the waterbody, road traffic, noise and air pollution, commercial fishing, or disruption of way of life for tribes. At some sites, a comparative analysis of impacts such as these may be useful in order to fully assess and balance the tradeoffs associated with each alternative.

11. Monitor During and After Sediment Remediation to Assess and Document Remedy Effectiveness.

A physical, chemical, and/or biological monitoring program should be established for sediment sites in order to determine if short-term and long-term health and ecological risks are being adequately mitigated at the site and to evaluate how well all remedial action objectives are being met. Monitoring should normally be conducted during remedy implementation and as long as necessary thereafter to ensure that all sediment risks have been adequately managed. Baseline data needed for interpretation of the monitoring data should be collected during the remedial investigation.

Depending on the risk management approach selected, monitoring should be conducted during implementation in order to determine whether the action meets design requirements and sediment cleanup levels, and to assess the nature and extent of any short-term impacts of remedy implementation. This information can also be used to modify construction activities to assure that remediation is proceeding in a safe and effective manner. Long-term monitoring of indicators such as contaminant concentration reductions in fish tissue should be designed to determine the success of a remedy in meeting broader remedial action objectives. Monitoring is generally needed to verify the continued long-term effectiveness of any remedy in protecting human health and the environment and, at some sites, to verify the continuing performance and structural integrity of barriers to contaminant transport.

IV. IMPLEMENTATION

EPA RPMs, OSCs, and RCRA Corrective Action project managers should immediately begin to use this guidance at all sites where the risks from contaminated sediment are being investigated. EPA expects that Federal facility responses conducted under CERCLA or RCRA will also be consistent with this directive. This consultation process does not apply to Time-Critical or emergency removal actions or to sites with only sediment-like materials in wastewater lagoons, tanks, storage or containment facilities, or drainage ditches.

Consultation Process for CERCLA Sites

To help ensure that Regional site managers appropriately consider these principles *before* site-specific risk management decisions are made, this directive establishes a two-tiered consultation procedure that will apply to most contaminated sediment sites. The consultation process applies to all proposed or listed NPL sites where EPA will sign or concur on the ROD, all Non-Time-Critical removal actions where EPA will sign or concur on the Action Memorandum, and all NPL-equivalent sites where there is or will be an EPA-enforceable agreement in place.

Tier 1 Process

Where the sediment action(s) for the entire site will address more than 10,000 cubic yards or five acres of contaminated sediment, Superfund RPMs and OSCs should consult with their appropriate Office of Emergency and Remedial Response (OERR) Regional Coordinator at least 30 days before issuing for public comment a Proposed Plan for a remedial action or an Engineering Evaluation/Cost Analysis (EE/CA) for a Non-Time-Critical removal action.

This consultation entails the submission of the draft proposed plan or draft EE/CA, a written discussion of how the above 11 principles were considered, and basic site information that will assist OERR in tracking significant sediment sites. If the project manager has not received a response from OERR within two weeks, he or she may assume no further information is needed at this time. EPA believes that this process will help promote nationally consistent approaches to evaluate, select and implement protective, scientifically sound, and cost-effective remedies.

Tier 2 Process

This directive also establishes a new technical advisory group (Contaminated Sediments Technical Advisory Group–CSTAG) that will monitor the progress of and provide advice regarding a small number of large, complex, or controversial contaminated sediment Superfund sites. The group will be comprised of ten Regional staff and approximately five staff from OSWER, OW, and ORD. For most sites, the group will meet with the site manager and the site team several times throughout the site investigation, response selection, and action implementation processes. For new NPL sites, the group will normally meet within one year after proposed listing. It is anticipated that for most sites, the group will meet annually until the ROD is signed and thereafter as needed until all remedial action objectives have been met. The specific areas of assistance or specific documents to be reviewed will be decided by the group on a case-by-case basis in consultation with the site team. For selected sites with an on-going RI/FS or EE/CA, the group will be briefed by the site manager some time in 2002 or 2003. Reviews at sites with remedies also subject to National Remedy Review Board (NRRB) review will be coordinated with the NRRB in order to eliminate the need for a separate sediment group review at this stage in the process.

Consultation Process for RCRA Corrective Action Facilities

Generally, for EPA-lead RCRA Corrective Action facilities where a sediment response action is planned, a two-tiered consultation process will also be used. Where the sediment action(s) for the entire site will address more than 10,000 cubic yards or five acres of contaminated sediment, project managers should consult with the Office of Solid Waste s Corrective Action Branch at least 30 days before issuing a proposed action for public comment. This consultation entails the submission of a written discussion of how the above 11 principles

were considered, and basic site information that will assist OSW in tracking significant sediment sites.

If the project manager has not received a response from OSW within two weeks, he or she may assume no further information is needed. States are also encouraged to follow these procedures. For particularly large, complex, or controversial sites, OSW will likely call on the technical advisory group discussed above.

EPA also recommends that both state and EPA project managers working on sediment contamination associated with Corrective Action facilities consult with their colleagues in both RCRA and Superfund to promote consistent and effective cleanups. EPA believes this consultation would be particularly important for the larger-scale sediment cleanups mentioned above.

EPA may update this guidance as more information becomes available on topics such as: the effectiveness of various sediment response alternatives, new methods to evaluate risks, or new methods for characterizing sediment contamination. For additional information on this guidance, please contact the OERR Sediments Team Leader (Stephen Ells at 703 603-8822) or the OSW Corrective Action Programs Branch Chief (Tricia Buzzell at 703 308-8632).

NOTICE: This document provides guidance to EPA Regions concerning how the Agency intends to exercise its discretion in implementing one aspect of the CERCLA and RCRA remedy selection process. This guidance is designed to implement national policy on these issues. Some of the statutory provisions described in this document contain legally binding requirements. However, this document does not substitute for those provisions or regulations, nor is it a regulation itself. Thus it cannot impose legally binding requirements on EPA, states, or the regulated community, and may not apply to a particular situation based upon the circumstances. Any decisions regarding a particular situation will be made based on the statutes and regulations, and EPA decision-makers retain the discretion to adopt approaches on a case-by-case basis that differ from this guidance where appropriate. Interested parties are free to raise questions and objections about the substance of this guidance and the appropriateness of the application of this guidance to a particular situation, and the Agency welcomes public input on this document at any time. EPA may change this guidance in the future.

cc: Michael H. Shapiro
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NARPM Co-Chairs
OERR Records Manager, IMC 5202G
OERR Documents Coordinator, HOSC 5202G
RCRA Key Contacts, Regions 1 - 10

Attachment K

Letter from Judith Enck,
U.S. Envtl. Prot. Agency
Region 2 Administrator to
Basil Seggos
Dec 16, 2016

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
290 BROADWAY
NEW YORK, NY 10007-1866

DEC 16 2016

Honorable Basil Seggos
Commissioner
New York State Department of Environmental Conservation
625 Broadway, 14th Floor
Albany, NY 12233-1010

Re: Hudson River PCBs Superfund Site Operation, Maintenance and Monitoring Program

Dear Commissioner Seggos:

Your November 14, 2016 letter regarding the Hudson River PCBs Superfund Site raises several issues concerning the Operation, Maintenance and Monitoring (OM&M) sediment sampling program that will help assess the effectiveness of the Hudson River dredging that was completed in 2015. While EPA shares the New York State Department of Environmental Conservation's interest in ensuring that EPA has data sufficient to monitor the effectiveness of the dredging, we disagree that the OM&M sediment sampling program is inadequate for that purpose. EPA has discussed our reasoning in detail with your staff in several meetings and phone calls this year.

It may be useful to reiterate here the purpose of the sediment monitoring component of the OM&M program. The data quality objectives of that component were developed in coordination with NYSDEC and are set forth in Section 2.3.1 of the 2010 Operation, Maintenance, and Monitoring Scope for Phase 2 of the Remedial Action (OM&M Scope), which is incorporated into the consent decree between EPA and General Electric Company. Those objectives are:

- Determine post-remediation PCB levels in sediments in non-dredge areas of the Upper Hudson River.
- Provide data on Select Areas that exceeded the mass per unit area removal criteria that were not targeted for removal because they were buried by cleaner sediments to assess whether the deposits have experienced erosion.[1]
- Determine sediment recovery rates in non-dredge areas of the Upper Hudson River.
- Examine the changes to surface PCB concentrations in backfill areas.

The OM&M Scope calls for surface sediment samples to be collected from "[a]pproximately 350 sampling locations" in order to track the recovery of surface sediments in non-dredge areas.[2] The Scope also calls for

[1] The OM&M Scope indicates that this particular objective will be addressed through bathymetric surveys rather than sediment sampling. See Section 2.3.3 of the OM&M Scope. This work will be performed in 2017 and is separate from the OM&M sediment sampling program.

[2] 2010 OM&M Scope, § 2.3.2.1.

sediment sampling in a minimum of 50 backfill areas in each of the three river sections.[3] The number of samples was based on the variability seen in sediment PCB data that was collected in 2010-2013. The number of sampling locations identified in the 2010 OM&M Scope is presented as an estimate because at the time the Scope was written, EPA anticipated that the actual number of samples would be determined during development of a work plan for the sediment sampling.

After considering the post-dredging variability in surface sediment PCB concentrations and in consultation with EPA's statistician, EPA determined that a total of 226 locations in non-dredge areas and 149 locations in dredge areas should be sampled in order to have a statistically sufficient number of samples to track surface sediment PCB concentrations in the Upper Hudson River over time. This fall GE collected samples from each of the 226 non-dredge areas, but due to safety concerns related to deteriorating weather conditions GE needed to demobilize from the river before collecting samples from the dredge areas. In the spring of 2017 GE will return to the river and collect samples from the 149 dredge locations that GE was unable to sample in 2016. If, prior to the collection of samples from the 149 dredge locations, EPA decides to calculate average surface sediment PCB concentrations, the analytical results from 275 samples that GE previously collected from the dredge areas immediately after the placement of backfill can be used to represent the dredge areas for purposes of those calculations.

The density of surface sediment samples is consistent with similar dredging projects where sediment samples are being collected to assess temporal changes in contaminant levels. If, after review of the fall 2016 sediment sampling results, EPA determines that additional sample locations in the non-dredge areas are needed to evaluate changes in sediment PCB concentrations over time, such additional sampling would be performed in the spring of 2017. Even if EPA decides that such additional sampling is needed, however, we do not expect the number of such samples to be anywhere near the 1800 additional locations requested by NYSDEC. While an effort of that magnitude, increasing the number of sampling locations by a factor of more than four, would allow for a more detailed delineation across the areal extent of the sediment, it is not necessary in order to achieve the data quality objectives quoted above. EPA has identified a statistically appropriate number of sampling locations and will require GE to sample the same locations in the same manner over an extended period of time, at appropriate (five-year) intervals.

The OM&M sediment sampling program was designed to assess sediment recovery rates in non-dredge areas in the three river sections, and not on a pool-by-pool basis.[4] Nevertheless, because that program includes sample locations in each of the Upper Hudson River dam pools, which GE will sample using an unbiased approach for each river mile, the OM&M sediment sampling data will allow EPA to infer average sediment concentrations over time on a pool-by-pool basis. In addition, and as EPA discussed at the December 8 meeting of the Community Advisory Group, the ongoing fish monitoring program will provide localized information that is representative of post-dredging conditions in the 40 miles of the Upper Hudson River. If in the future EPA determines that fish PCB levels in a particular pool do not appear to be declining at an acceptable rate, then at that time EPA will consider whether to collect additional sediment samples from that area in order to better understand any delay in fish recovery.

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[3] Id.

[4] See, e.g., OM&M Scope § 2.3.2.1 (surface sediments from non-dredge areas "will be sampled upon completion of dredging in each river section...") and "[t]he backfill sampling program will entail collection of samples from a minimum of 50 locations from backfilled areas in each river section." In addition, samples from "about 30 locations per river section" will be analyzed for beryllium-7. OM&M Scope § 2.3.2.3.

As EPA has explained to NYSDEC staff, if EPA were to require anything like the major increase in sediment sampling sought by NYSDEC, it is unlikely that the sampling could be completed before the summer of 2017. There are a number of reasons for this, including the fact that it would take considerable time and effort to redesign the sampling grid (including performing new statistical analyses), and to consult with NYSDEC, the federal trustees and other stakeholders. It would also take time to try and reach agreement with GE for its performance of the work, which would be far more extensive than the program GE agreed to perform in the consent decree. In addition, a significantly larger program would take longer to perform.

Your letter states that "much more sampling is necessary in order to answer the questions many stakeholders have raised about what has been left behind by the remedy." The letter also states that additional sampling is needed in order to "identify specific areas of the Upper Hudson River that may require further active remediation in the future..." and it has been reported in the press that your Department's goal for the additional sampling is to prove that additional dredging is needed.[5] More than 10,000 sediment locations were sampled to delineate PCBs in the Upper Hudson River as part of the remedial design, and therefore the PCB distribution in the river was known at the time EPA developed the 2010 OM&M Scope. The identification of potentially missed PCB inventory is not a purpose of either the OM&M program or the five-year review and would be outside the scope of the data quality objectives established for the sediment sampling program.

EPA believes that the sediment sampling program provided in the 2010 OM&M Scope and the 2016 Sediment Sampling Work Plan (and potentially supplemented by some additional sampling in the spring of 2017, if that is determined to be necessary) meets the objective of providing information for evaluating the change in PCB concentrations in the sediments over time. We do not believe that the additional 1800 samples requested by NYSDEC are needed either for the OM&M program or the five-year review, and do not believe that conditions in the river have changed since 2010 in a manner that warrants the significant changes requested by NYSDEC.

Your letter also suggests that the substantially greater number of sediment samples that NYSDEC is seeking is needed "to understand the ability of the project to meet its remedial action objectives (RAOs) in the timeframes predicted by the Record of Decision (ROD) (i.e., 5 and 16 years, respectively, after dredging)." We assume that you are alluding to the fish fillet target concentrations of 0.4 mg/kg and 0.2 mg/kg PCBs, respectively, that are included in Section 9.1 of the ROD. However, as EPA explained to your staff, these target concentrations are not RAOs under the ROD. Rather, they are interim milestones that, once achieved, might allow fish advisories to be relaxed somewhat. PCB levels in Hudson River water have declined since the dredging was completed, and we expect that PCB levels in fish also will continue to follow a downward trend. As we have also discussed with NYSDEC staff, the model forecasts used for the ROD were not intended to predict the specific years in which specified PCB levels would be achieved in the fish, but rather, were used to help EPA compare the remedial alternatives. The RAOs do not include specific years in which specified PCB levels need to be achieved in fish in order for EPA to deem the remedy protective.[6]

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[5] Moore, Kathleen, "DEC using new way to push for more dredging." *Glens Falls Post-Star*, 15 Nov. 2016

[6] In this connection it is important to note that -- as has always been understood by your staff -- models cannot be used to predict specific dates by which such a milestone will be reached. Models are used to compare remedial alternatives, and they can provide a general timeframe within which such a milestone is expected to be met. Real world occurrences -- such as river flows that differ from the assumed flows in the model and adjustments to remedial operations over the course of the remedial work -- will impact recovery rates in ways that were not captured in the previously developed model forecasts. For example, adjustments to the dredging operations that provided an overall benefit to the project also likely increased the short-term exposures of fish to PCBs and will result in some delay (likely several years) to the forecasted years for achieving the 0.4 mg/kg PCB target level.

If NYSDEC wishes to conduct additional sediment sampling, it is free to do so. With respect to your request for EPA to defer issuing the Certification of Completion of the Remedial Action until after such additional sampling occurs, EPA will make a decision about issuing the Certification in accordance with the applicable consent decree requirements, as EPA has explained to your staff. Neither the schedule for NYSDEC's sediment sampling nor the schedule for the OM&M sediment sampling are factors that will affect the issuance of the Certification.

If you wish to discuss these issues further, please let me know or ask your staff to contact Walter Mugdan at 212-637-4390 or mugdan.walter@epa.gov, or Gary Klawinski at 518-407-0400 or klawinski.gary@epa.gov. Thank you.

Sincerely,

Judith A. Enck

Judith A. Enck
Regional Administrator

Attachment L

Clarifying the Use of Protectiveness Determinations for Comprehensive Environmental Response, Compensation, and Liability Act Five-Year Reviews,

September 13, 2012

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
SOLID WASTE AND
EMERGENCY RESPONSE

OSWER 9200.2-111

SEP 13 2012

MEMORANDUM

SUBJECT: Clarifying the Use of Protectiveness Determinations for Comprehensive Environmental Response, Compensation, and Liability Act Five-Year Reviews

FROM: James E. Woolford, Director
Office of Superfund Remediation and Technology Innovation

Reggie Cheatham, Director
Federal Facilities Restoration and Reuse Office

TO: National Superfund Program Managers, Region 1-10

PURPOSE

The purpose of this memorandum is to clarify the use of protectiveness determinations in Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) Five-Year Reviews (FYR). It provides general guidance for the use of specific protectiveness determinations and recommends language to be used when drafting a protectiveness statement. The information provided in this memorandum supplements, but does not supersede, the language in the *"Comprehensive Five-Year Review Guidance," OSWER No. 9355.7-03B-P (June 2001).*

BACKGROUND

An audit by the Office of Inspector General (OIG) entitled "*Stronger Management Controls Will Improve EPA Five-Year Reviews of Superfund Sites*" issued February 6, 2012 identified situations where data provided in a FYR report did not fully support the region's protectiveness determination. Specifically, the OIG identified situations where the regions did not follow agency guidance for making protectiveness determinations for remedies under construction and concluded that short-term protectiveness was not adequately defined in Agency guidance. As a result, the OIG recommended that the Office of Solid Waste and Emergency Response (OSWER) clearly define the protectiveness categories used in Agency guidance and ensure that protectiveness definitions are consistently applied across the Agency.

The purpose of a FYR is to evaluate the implementation and performance of a remedy in order to determine if the remedy is or will be protective of human health and the environment. Protectiveness is generally defined in the National Contingency Plan (NCP) by the risk range for carcinogens and the hazard index (HI) for non-cancer effects. Evaluation of the remedy and the determination of protectiveness should be based on and sufficiently supported by data and observations. Consistent with the "*Comprehensive Five-Year Review Guidance,*" a discussion of this evaluation should be described and presented in the FYR report, along with the protectiveness determination.

IMPLEMENTATION

To assess the protectiveness of the remedy, it is important to evaluate human health risks, ecological risks, and the general performance of the selected remedy. To facilitate this evaluation, a technical assessment of a remedy is conducted to answer the following questions. The answers to these questions provide a framework for organizing and evaluating the FYR data and information:

Question A –Is the remedy functioning as intended by the decision documents?

Question B – Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?

Question C – Has any other information come to light that could call into question the protectiveness of the remedy?

Evaluating Remedy Protectiveness

For CERCLA sites that require a FYR, a separate protectiveness statement is required for each operable unit (OU) where the remedial action is currently underway or remedial construction is complete. If the site is construction complete, a site-wide protectiveness determination is also required and will generally be the same protectiveness determination as the least protective OU at the site.

The OSWER *"Comprehensive Five-Year Review Guidance"* defines five protectiveness categories: protective, short-term protective, will be protective, protectiveness deferred, and not protective. The following discussion provides general guidance for the use of the specific protectiveness determinations and recommends language to be used when drafting the protectiveness statement for the FYR report.

Protective

A protectiveness determination of "protective" may be appropriate for remedies where:

- Construction activities are complete and remedy is operating; or
- Construction activities are complete, remedial action objectives (RAOs) have been achieved, and operation and maintenance activities are occurring.

A protectiveness determination of "protective" is typically used when the answers to Questions A, B and C provide sufficient data and documentation to conclude that the remedy is functioning as intended and all human and ecological risks are currently under control and are anticipated to be under control in the

future.

Recommended Language for a Protectiveness Determination of "Protective"

"The remedy at OUX is protective of human health and the environment."

The Remedial Project Manager should briefly describe in a separate paragraph below the protectiveness statement the elements of the remedy that protect human health and the environment and how the RAOs have been met or are being met.

Short-Term Protective

A protectiveness determination of "short-term protective" may be appropriate for remedies where:

- Construction activities are complete and remedy is operating; or
- Construction activities are complete, remedial action objectives have been achieved, and operation and maintenance activities are occurring.

A protective determination of "short-term protective" is typically used when the answers to Questions A, B and C provide sufficient data and documentation to conclude that the human and ecological exposures are currently under control and no unacceptable risks are occurring. However, the data and/or documentation review also raise issues that could impact future protectiveness or remedy performance but not current protectiveness. Examples of scenarios that may result in a short-term protectiveness determination may include:

- No exposure is occurring but institutional controls have not been fully implemented;
- Future land use assumptions may have changed;
- Engineering performance issues related to the operation of the remedy; or
- Monitoring data indicates that remedy will not achieve goals in the anticipated time frame

Recommended Language for a Protectiveness Determination of "Short-Term Protective"

"The remedy at OU X currently protects human health and the environment because (describe the elements of the remedy that protect human health and the environment in the short-term). However, in order for the remedy to be protective in the long-term, the following actions need to be taken (describe the actions needed) to ensure protectiveness.

Will be Protective

A protectiveness determination of "will be protective" may be appropriate for remedies where:

- Construction activities are ongoing

A protective determination of "will be protective" is typically used when the answers to Questions A, B and C provide sufficient data and documentation to conclude that the human and ecological exposures are currently under control and no unacceptable risks are occurring in those areas. In addition, answers

to Questions A, B and C also indicate that the remedy under construction is anticipated to be protective upon completion and no remedy implementation or performance issues have been identified.

Recommended Language for a Protectiveness Determination of "Will Be Protective"

"The remedy at OUX is expected to be protective of human health and the environment upon completion. In the interim, remedial activities completed to date have adequately addressed all exposure pathways that could result in unacceptable risks in these areas."

Protectiveness Deferred

A protectiveness determination of "protectiveness deferred" may be appropriate for remedies where:

- Construction activities are ongoing;
- Construction activities are complete and remedy is operating; or
- Construction activities are complete, remedial action objectives have been achieved, and operation and maintenance activities are occurring.

This protective determination is generally used when the available information to answer Questions A, B and C does not provide sufficient data and documentation to conclude that all human and ecological risks are currently under control and no unacceptable exposures are occurring. Examples of scenarios that may result in a "protectiveness deferred" determination include:

- A new exposure pathway (e.g., vapor intrusion) has been identified and additional data are required to determine if an unacceptable risk is occurring;
- An emerging contaminant is present and the current risk has not been evaluated;
- An ecological risk assessment has never been adequately addressed at the site; or
- The toxicity value has changed and it unclear whether the current remedy at a site is protective or whether the selected remedy can achieve the new risk-based cleanup level.

When a protectiveness deferred determination is made, the protectiveness statement generally discusses the actions needed to collect the missing information and the timeframe anticipated to complete these actions. Once the necessary data and/or information are obtained, a Five-Year Review addendum is typically completed that documents the protectiveness determination for the OU(s) where the protectiveness had been deferred.

Recommended Language for a Protectiveness Determination of "Protectiveness Deferred"

"A protectiveness determination of the remedy at OU X cannot be made at this time until further information is obtained. Further information will be obtained by taking the following actions (describe the actions). It is expected that these actions will take approximately (insert time frame) to complete, at which time a protectiveness determination will be made."

Not Protective

A protectiveness determination of "not protective" may be appropriate for remedies where:

- Construction activities are ongoing;
- Construction activities are complete and remedy is operating; or
- Construction activities are complete, remedial action objectives have been achieved, and operation and maintenance activities are occurring.

A protectiveness determination of "not protective" is generally used when the answers to Questions A, B and C provide adequate data and documentation to conclude that the human and/or ecological risks are not currently under control. Examples of scenarios that may result in a "not protective" determination include:

- An immediate threat is present (ex. new exposure pathway identified and it is reasonably likely to assume that unacceptable exposures are occurring)
- Migration of contaminants is uncontrolled and poses an unacceptable risk to human health and the environment; or
- Potential or actual exposure is clearly present or there is evidence of exposure

Recommended Language for a Protectiveness Determination of "Not Protective"

"The remedy at OU X is not protective because of the following issues(s) (describe each issue). The following actions need to be taken (describe the actions needed) to ensure protectiveness."

CONCLUSION

A five-year review should determine whether the remedy at a site is or upon completion will be protective of human health and the environment. The level of effort necessary to conduct a five-year review is site-specific and should be tailored appropriately for the remedial action and its stage of implementation.

If you have any questions, please contact David Cooper at (703) 603-8763 or at cooper.davide@epa.gov.

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NARPM Co-Chairs

Attachment M

Recommendations to EPA for the “Five Year Review Report” for Hudson River PCBs Site

Recommendations to EPA for the Five Year Review Report for Hudson River PCBs Site

Executive Summary

The Hudson River is one of the highest priority natural resources for the Department of Environmental Conservation (DEC) in New York State. Since the 1970s, DEC has been at the forefront in requiring General Electric (GE) to address the PCB contamination of the Hudson River. With over forty years of effort involved in confronting this major environmental issue, DEC has a unique historical perspective to offer to the Environmental Protection Agency (EPA). DEC scientists and engineers have conducted an independent evaluation of the site history and current conditions, utilizing EPA's own guidance and criteria for performing five year remedy reviews. DEC also has a point of view different from EPA, in that the Hudson River is primarily a natural resource of the State; the people of the State will be making use of this precious resource long into the future. As a result, DEC is providing the State's positions on the upcoming 2017 Five-Year Review (FYR) for the Hudson River PCBs Site before EPA finalizes its report.

DEC's position has been informed by an independent evaluation of the information and data available for the site in an effort to provide EPA with an objective analysis regarding whether or not the remedy is protective of human health and the environment. When deciding on the remedy for the Hudson River, EPA considered that cancer and non-cancer health risks were well above the acceptable risk range for people who ate fish from both the upper Hudson River (between Hudson Falls and Troy) and the lower Hudson River (from Troy south to Manhattan). Risks to ecological receptors, particularly fish-eating animals, were also above EPA's acceptable range. The primary purpose of the remedy was to address this risk. In turn, the primary goal of the FYR is to ensure this risk has been adequately addressed by the remedy.

DEC also considered the rationale relied upon by EPA in the Record of Decision (ROD), which describes in detail why the implemented remedy was selected. EPA chose an active remedy, under which significant amounts of PCBs would be removed from the sediments of the upper Hudson by sediment dredging. EPA selected this remedy primarily based upon the time it would take to achieve targeted fish PCB concentrations after dredging. This was necessary, according to EPA, to protect the human and ecological receptors exposed to PCBs by eating fish. EPA understood the advisories for people to stop eating fish were not completely effective, and could never apply to ecological receptors, and thus the remedy selection needed to be based primarily upon the time to meet the targeted reductions in fish PCB concentrations. Specifically, EPA stated in the ROD that a delay of ten years in reaching target fish concentrations, of 0.4 mg/kg within 5 years of the completion of dredging and 0.2 mg/kg within 16 years of the completion of dredging, was unacceptable. This ten year delay was used as a basis for rejecting the Monitored Natural Attenuation (MNA) remedial alternative.

The most important point made in the rationale provided by EPA in the ROD for the selected remedy is that EPA concluded the dredging was needed to accelerate the time it would take to reach the remedial targets for fish flesh in order to quickly reduce human health and ecological risk compared to other alternatives that were evaluated. Additional delays of ten or more years to reach the target fish PCB concentrations were unacceptable to EPA. Otherwise, EPA would have selected the MNA only remedy. Institutional controls were understood to not be completely effective, and the acceleration of the time frame was necessary to protect people who eat fish as well as ecological receptors, both of which are subject to unacceptable levels of risk from consuming PCB-contaminated fish from the Hudson River. The State s concurrence with the ROD was based on these very same principles and the understanding that delays to reach the target fish PCB concentrations were not acceptable.

EPA admitted in its first five year review that, based on the fact that portions of the upper Hudson River, particularly in River Section 2, are much more contaminated than previously thought, fish flesh PCB targets will not be met within the time frames anticipated in the ROD.

As the time to reach targeted fish PCB concentrations was the primary basis for the selected remedy, DEC has recommended that EPA perform the sampling work necessary to complete a detailed evaluation of the performance of the remedy, including increasing the sampling of sediment and fish tissue to the scale and frequency necessary to optimize the remedy through further remedial work as necessary to achieve the targeted fish PCB reductions identified in the ROD.

DEC also recommends that EPA expand the investigation of the site to include performance of a Remedial Investigation and Feasibility Study for the portion of the site between the Federal Dam at Troy and the Battery in New York City. This work is necessary to determine the nature and extent of PCB contamination in the sediments, water, and biota of the lower Hudson, and to evaluate remedial alternatives to address the currently uncontrolled unacceptable risks to human health and the environment. Until these recommendations are acted upon, EPA must not conclude that the remedy is protective of human health and the environment.

Taking into account these recommendations, EPA s basis for selecting the remedy, and all data and information that has been gathered from implementation of the dredging project, DEC has determined the following:

1) that the dredging remedy is currently not protective of human health and the environment, as there are known exposures to both human and ecological receptors which have not been controlled and which remain in excess of EPA s acceptable risk range; and

2) that an issue raised in the previous Five Year Review, the fact that sediment concentrations higher than anticipated will remain after dredging, indicates that the

targeted fish PCB concentrations will not be reached in the time frames identified in the ROD.

Therefore, EPA should carefully consider the Department□s recommendations and incorporate them into the FYR, EPA should determine that the remedy is not protective of human health and the environment based on uncontrolled risks, and EPA should undertake all necessary actions to ensure that the remedy becomes fully protective to the benefit of the people of New York State.

Section 1 Purpose of Document

This document is intended to provide EPA, and the people of the State of New York, the position of DEC as it relates to the ongoing Five-Year Review for the Hudson River PCBs Site currently being conducted by EPA.

According to the EPA Guidance ("Comprehensive Five Year Review Guidance" OSWER No. 9355.7-03B-P, 2001) the purpose of an FYR is to:

> *"…evaluate the implementation and performance of a remedy in order to determine if the remedy is or will be protective of human health and the environment. Protectiveness is generally defined in the National Contingency Plan (NCP) by the risk range and the Hazard Index (HI). Evaluation of the remedy should be based upon and sufficiently supported by data and evaluations."* (Section 1.1, page. 1-1)

While this document is not intended to replace or represent the EPA's Five-Year Review Report, the same format for report sections will be followed to allow for readers of both documents to understand the State's positions on the outcome of the process in a stepwise manner. This document tracks the FYR reporting process step-by-step, and concludes with the State's recommended protectiveness determinations, and recommendations for future action.

Section 2 Site Chronology

This section summarizes the Site Chronology to provide the reader with a basis to understand the history of PCB contamination in the Hudson River and the government's response. For more detail, please refer to the project documents, including the previous EPA Five Year Review Report site chronology.

1947-1977: Direct discharges of PCBs occur from two GE capacitor manufacturing facilities in Hudson Falls and Fort Edward

1983: Hudson River PCBs Site listed on the EPA National Priorities List

1984: EPA issues the first Record of Decision for the site, selecting Interim No Action for the PCB contaminated sediments in the upper Hudson.

1989: At the request of New York State, EPA begins Five Year Review of 1984 remedy

1990-91: Remnant Sites are capped as an Interim Remedial Measure by GE

1990: EPA starts the Reassessment of the 1984 remedy

2000: EPA issues Proposed Plan, identifying "Rem 3/10/Select", an active sediment removal remedy, as the preferred remedial alternative.

2002: EPA issues Record of Decision selecting "Rem 3/10/Select" as the remedy for the contaminated sediments of the upper Hudson between Fort Edward and Troy.

2003-09: GE, under a series of EPA administrative Orders, performs remedial design and baseline monitoring.

2006: EPA issues Remedial Action Consent Decree under which GE will perform the remedy.

2009: GE performs Phase 1, the first year of the dredging remedy.

2010: EPA performs a peer review of the remedy and issues modified scope of work for Phase 2

2011-2016: GE performs Phase 2, the remaining portion of the remedy.

2012: EPA issues first Five-Year Review Report

Section 3 Background

Section 3.1: Site Location, Physical Characteristics, Land and Resource Uses

(The following is taken largely from the 2012 EPA Five Year Review Report, and is included to give the reader the same perspective on these site characteristics.)

Site Location

The Site includes a nearly 200 river-mile stretch of the Hudson River in eastern New York State from the Village of Hudson Falls to the Battery in New York City. The Site is divided into the Upper Hudson River (the length of river between Hudson Falls and the Federal Dam at Troy, New York) and the Lower Hudson River (the length of river between the Federal Dam at Troy and the Battery). For purposes of the project, EPA further divided the Upper Hudson River area into three main sections known as River Section 1, River Section 2, and River Section 3. River Section 1 is the most upstream section, extending approximately 6 miles from Fort Edward to the Thompson Island Dam; River Section 2 extends from the Thompson Island Dam to the Northumberland Dam near Schuylerville, an extent of approximately 5 miles; and River Section 3 extends from below the Northumberland Dam to the Federal Dam at Troy, an extent of approximately 29 miles.

The Site also includes five Remnant Deposits located upriver from River Section 1. The Remnant Deposits are areas of PCB-contaminated sediments that became exposed after the river water level dropped following the removal of the Fort Edward Dam in 1973. Remnant Deposit 1 originally appeared as an island, but due to flooding in 1976 and 1983 most of the exposed sediment associated with this deposit site was scoured. Remnant Deposit 2 is approximately 3.5 acres and is located on the west bank of the Hudson River, in the town of Moreau. Remnant Deposit 3 is approximately 17 acres and is located on the east bank of the Hudson River, in the town of Fort Edward. Remnant Deposit 4 is approximately 24 acres and is located on the west bank of the Hudson River in the town of Moreau. Remnant Deposit 5 is approximately 3.5 acres and is located on the east bank of the Hudson River in the town of Fort Edward. The site has been broken up into □Operable Units□or □OUs□by EPA for administrative purposes. OU1 is the remedial work done under the 1984 ROD, including the work at the Remnant Sites. OU2 is the dredging remedy selected in the 2002 ROD. OU3 is a removal action taken on Rogers Island by EPA in 1999 to address soil contamination with PCBs and metals. OU4 is the floodplains, currently the subject of an ongoing remedial investigation.

Physical Characteristics

The Upper Hudson River is freshwater and non-tidal. Downstream of Fort Edward, the river is joined by several tributaries, the largest of which are the Mohawk River, Batten Kill, Fish Creek, and the Hoosic River. The flow in the Upper Hudson River is primarily controlled by several reservoirs above Glens Falls, including the Great Sacandaga

Lake. The Upper Hudson River has an average depth of less than 8 feet in the shoal areas and approximately 18 feet in the channel, with a maximum depth of more than 45 feet. The New York State Canal Corporation (NYSCC) navigation channel is generally identified as being a minimum of 12 feet deep by design in the project area.

The Champlain Canal is coincident with portions of the Hudson River, extending from Waterford on the Hudson to Whitehall at the southern end of Lake Champlain. Bedrock, cut away to form the Champlain Canal, is exposed in some areas of the river, while lacustrine silts and clays of glacial age are exposed in other areas. Coarser-grained sediments are often observed in the river channel, while finer-grained sediments are more common in shallow water. Areas adjacent to the Upper Hudson River include forested shoreline wetlands, transitional uplands, and vegetated backwater such as emergent marsh and scrub-shrub wetlands.

Land and Resource Use

In the Upper Hudson River, land use is primarily residential and agricultural with some commercial and industrial activities. Such uses of the river and lands surrounding the river are projected to remain the same. The Site passes through 14 different counties as the river flows to its final discharge point in New York Harbor. Four counties (Albany, Washington, Rensselaer and Saratoga) lie adjacent to the Upper Hudson River. Within these four counties, forest and farmlands surround urban centers and historic villages. In addition to the General Electric (GE) Hudson Falls and Fort Edwards plants, the area is home to other businesses including technology companies, oil service companies, and food companies.

The City of Poughkeepsie, the Dutchess County Water and Wastewater Authority, the Village of Rhinebeck, the Castle Point Medical Center, as well as the Highland and Port Ewen Water Districts obtain at least a portion of their potable water supplies directly from the Hudson River. The Towns of Waterford and Halfmoon also have intakes for obtaining Hudson River water, although both towns currently obtain their water from the City of Troy via an EPA-constructed water line. The river has been utilized for hydroelectric and thermal power generation, as well as for manufacturing processes, cooling and fire protection. The river is also used for irrigating agricultural lands and watering domestic lawns and gardens.

The river supports a variety of water-based recreational activities including sport fishing, waterfowl hunting, swimming and boating; however, at the current time, there is a New York State Department of Health □eat none□fish consumption advisory for the entire Upper Hudson River between the Corinth Dam and the Federal Dam at Troy.

Section 3.2 Initial Problem Identification and Responses

During an approximate 30-year period ending in 1977, GE used PCBs in its capacitor manufacturing operations at its Hudson Falls and Fort Edward, New York facilities. PCB

oils were discharged both directly and indirectly from these plants into the Hudson River.

In the early 1970s, in response to the discovery of PCBs in fish caught in the Hudson River, New York State began an enforcement action against General Electric. This enforcement action resulted first in an interim Order and Opinion in February 1976, and a final Agreement and Order in September 1976, under which GE implemented abatement actions to limit the direct discharges of PCBs from the capacitor plants in Hudson Falls and Fort Edward, NY. These actions included the limitation of direct PCB discharges from the capacitor plants, as well as construction and operation of a new wastewater treatment plant at the Fort Edward capacitor plant.

In 1973, the owner of the Fort Edward Dam removed the dam. As the dam was a short distance downstream of the GE Fort Edward capacitor plant (the Hudson Falls plant being located further upstream as well, above the Bakers Falls dam in the Village of Hudson Falls) some PCBs had contaminated the sediments in the pool impounded by the Fort Edward Dam. When the dam was removed, some of the PCBs still upstream of the dam were remobilized along with the sediments. These sediments were redeposited primarily in the vicinity of Rogers Island, a short distance downstream.

In August 1975, the New York State Department of Health issued the first advisories against consumption of fish from the Hudson River. These advisories exist, modified as appropriate, to the present day. NYSDOH continues to recommend that people eat none of the fish from the Upper Hudson River, that children under the age of 15 and that women of child-bearing age eat none of the fish from the river for the entire 200 mile length of the Superfund site, and that the general population limit their consumption of most species of fish caught south of the Federal Dam at Troy.

In February 1976, the New York State Department of Environmental Conservation implemented restrictions on fishing in the upper Hudson from Hudson Falls to the Federal Dam at Troy/Green Island. These restrictions were modified in 1995 to allow for catch and release fishing only in this reach of the upper Hudson.

Section 3.3 Superfund Listing and Initial Remedy Selection

The Site was proposed for inclusion on the National Priorities List (NPL) in September 1983 and formally listed in September 1984.

In 1984, EPA completed a Feasibility Study (FS) and issued a ROD for the Site. EPA identified PCB contamination in the Upper Hudson River sediments as a threat to human health and the environment, but selected an interim No Action remedy for the contaminated sediments because (as believed by EPA at the time) the reliability and effectiveness of remedial technologies available at that time were uncertain, and there were apparent downward trends of PCBs in fish, sediment, and water at the time (which did not continue after 1984).

The 1984 ROD included the following components:

• An interim No Action decision with regard to PCBs in the sediments of the Upper Hudson River;

• In-place capping, containment and monitoring of exposed Remnant Deposits (areas of former river bottom exposed by removal of the Fort Edward Dam), stabilization of the associated river banks and revegetation of the areas; and

• A detailed evaluation of the Waterford Water Works treatment facilities, including sampling and analysis of treatment operations to determine if modifications of the facilities were needed.

GE, as an Interim Remedial Measure under a 1990 Consent Decree with EPA, conducted the in-place capping of four Remnant Deposits located along the river banks upstream of the former Fort Edward Dam. The in place capping of these Remnant Deposits included grading, placement of a two-foot layer of soil and a manufactured geosynthetic clay liner, followed by revegetation to minimize erosion. This prevented direct contact with, and potential volatilization of, PCBs. The river banks were stabilized with rock to prevent scouring. Cap construction and the erection of gates to limit access were completed in 1991.

NYSDEC, with funding provided by EPA, conducted a treatability study at the Waterford Water Works. The study was released in 1990, and found that PCB concentrations were below current analytical detection limits after treatment and met current standards applicable to public water supplies.

Section 3.4 Reassessment and Remedy Selection Leading to the 2002 Record of Decision

In December 1989, EPA announced its decision to initiate a detailed Reassessment of the interim No Action decision for the Upper Hudson River sediments. This was prompted by the five-year review required by CERCLA, technical advances in sediment dredging and treatment / destruction technologies, as well as a request by NYSDEC for a re-examination of the 1984 decision.

EPA completed the Reassessment in December 2000, with the release of the Feasibility Study and Proposed Plan in late 2000. The Reassessement work is documented in several reports, including:

- Phase 1 Report (summary of existing conditions) – 1991
- Database Report – 1995
- Data Evaluation and Interpretation Report – 1997
- Low Resolution Sediment Coring Report – 1998
- Human Health Risk Assessment – Mid Hudson – 1999
- Revised Baseline Ecological Risk Assessment – 2000
- Revised Human Health Risk Assessment – 2000
- Revised Baseline Monitoring Report - 2000
- Feasibility Study Report - 2000

EPA issued the proposed plan in December 2000.

Following numerous public meetings and after extensive public comment, EPA issued a Record of Decision in February 2002.

The major components of the remedy in the 2002 ROD are:

- Upstream Source Control at the two GE capacitor plants in Hudson Falls and Fort Edward to achieve a target PCB surface water load at Rogers Island equal to an average surface water PCB concentration of 2 nanograms per liter.
- Targeted Environmental Dredging to remove PCB contaminated sediment from the Upper Hudson to meet specific removal criteria for PCB surface sediment concentration and PCB mass per unit area. This was done to achieve several objectives.
- Operation, Maintenance and Monitoring (OMM), including monitoring to evaluate the effectiveness of the remedy as well as to ensure that the remedy is protective of human health and the environment. Maintenance of any long term structures (such as caps) is also included.
- Monitored Natural Attenuation (now referred to as Monitored Natural Recovery, or MNR) , a reliance on natural processes after the dredging work to continue to result in a decrease in surface sediment PCB concentrations until the ultimate remedial goal is reached.
- Institutional Controls to reduce the potential for human consumption of fish from the Hudson River. These controls are the fish consumption advisories (FCAs), and the current catch and release fishery regulations in the upper Hudson.

(For a detailed listing of all remedy elements, see the 2002 ROD.)

Section 3.5 Summary of the Basis for the Need to Take Action

A good basic summary of the need to take action can be found on EPA□s Hudson River web page (https://www3.epa.gov/hudson/cleanup.html#quest1). There EPA states:

Polychlorinated biphenyls, or PCBs, were widely used as a fire preventive and insulator in the manufacture of electrical devices, like transformers and capacitors, because of their ability to withstand exceptionally high temperatures. During a 30-year period ending in 1977, when EPA banned the production of PCBs, is estimated that approximately 1.3 million pounds of PCBs were discharged into the Hudson River from two General Electric (GE) capacitor manufacturing plants located in the towns of Fort Edward and Hudson Falls, New York. Once PCBs entered the river, they were deposited and mixed with the sediments at many locations on the river bottom and at some locations along the shoreline in the floodplain.

PCBs build up in the environment (bioaccumulate), increasing in concentration as you move up the food chain. The primary health risk associated with the site is the accumulation of PCBs in the human body through eating contaminated fish. Since 1976, high levels of PCBs in fish have led New York State to close various recreational and

commercial fisheries and to issue advisories restricting the consumption of fish caught in the Hudson River. PCBs are considered probable human carcinogens and are linked to other adverse health effects such as low birth weight, thyroid disease, and learning, memory, and immune system disorders. PCBs in the river sediment also affect fish and wildlife.

In 1984, 200 miles of river, between Hudson Falls and the Battery in New York City, was placed on EPA's National Priorities List of the country's most contaminated hazardous waste sites.
Today the Hudson River exists as one of the most extensively studied rivers in the country, having been monitored almost continuously for a period of more than 25 years. Ongoing evaluations of water quality, sediment, air quality, fish, and wildlife by the Federal Government and the State of New York demonstrated that the river was not cleaning itself and PCBs in the sediment posed a serious risk to human health and the environment. Studies conducted to evaluate the extent of the problem revealed that most of the contaminated sediments were in "hot spots" situated in a 40-mile stretch of the river between the town of Fort Edward and the Troy Dam.

In EPA's 2002 ROD, there is also a good summary of the human health and environmental risks posed by the disposal of PCB in the Hudson River by GE. In the "Risk Characterization" section of the ROD, on page 38, EPA describes the cancer risk for a reasonably maximum exposed human fish consumer (one fish meal per week) of fish from the upper Hudson as one in a thousand. The hazard index (HI) a way of describing how much greater of an exposure is present as compared to an exposure which is not expected to cause non-cancer health impacts. The HI for adults consuming one fish meal per week from the upper Hudson, according to EPA, was 65; for adolescents, 71; for children, 104.

In the mid-Hudson area, EPA calculated the cancer risk to adult fish consumers at four in ten thousand, and one in ten thousand for children. The Hazard Index for adult fish consumers was 30; for children, 10.

EPA also calculated ecological risks posed by the PCBs disposed in the river. EPA's summary of the ecological risks included:

- Birds and mammals that eat PCB-contaminated fish from the Hudson River, such as the bald eagle, belted kingfisher, great blue heron, mink and river otter are at risk at the population level. PCBs may adversely affect the survival, growth, and reproduction of these species.
- Piscivorous (fish eating) mammals, represented by the river otter, are at the greatest risk due to their feeding patterns.
- Fragile populations of threatened and endangered species, represented by the bald eagle, are particularly susceptible to adverse effects from PCB exposure.
- Piscivorous fish (e.g., largemouth bass and striped bass) and omnivorous fish (e.g., brown bullhead and shortnose sturgeon) in the Hudson River may be

adversely affected (i.e., reduced survival, growth and/or reproduction) from exposure to PCBs.

- Omnivorous animals, such as the raccoon, that derive a large portion of their food from the Hudson River may be adversely affected (i.e., reduced survival, growth, and/or reproduction) from exposure to PCBs.
- Birds and mammals that feed on insects with an aquatic stage spent in the Hudson River, such as the tree swallow and little brown bat, may be adversely affected (i.e., reduced survival, growth and/or reproduction), particularly insectivorous mammals living in the Thompson Island Pool area.

Overall, EPA stated in the ROD (p. 49) that:

> Basis for Action: The excess cancer risk and non-cancer health hazards associated with human ingestion of fish, as well as the ecological risks associated with ingestion of fish by birds, fish and mammals, are above acceptable levels under baseline conditions. The response action selected in this ROD is necessary to protect the public health or welfare and the environment from actual releases of hazardous substances into the environment.

Section 4 Remedial Actions

Section 4.1 Remedy Selection

EPA evaluated five final remedial alternatives in the Feasibility Study. Those five alternative can be grouped into two types of alternatives – those which involve active remediation of the PCB contaminated sediments of the upper Hudson River (capping and/or dredging), and those which do not (No Action and Monitored Natural Attenuation. The five alternatives in the Proposed Plan and ROD were:

- No Action;
- Monitored Natural Attenuation – reliance on source control and natural recovery processes only;
- Cap 3/10/Select – capping of targeted areas of river bottom, with different criteria by River Section, along with source control and natural recovery processes;
- Removal 3/10/Select – environmental dredging of contaminated sediments from targeted areas of river bottom, with different criteria by River Section, along with source control and natural recovery processes;
- Removal 0/0/3 - capping of targeted areas of river bottom utilizing a more stringent set of criteria by River Section, along with source control and natural recovery processes.

In the ROD, EPA weighed the alternatives according to the remedy selection criteria in the National Contingency Plan, and made several determinations, resulting in the selection of the Removal 3/10/Select remedy for Operable Unit 2 of the site. The rationale is articulated in section 13.4 of the ROD, Rationale for Selection of the Selected Remedy, on pages 102-105.

A summary of the determinations by EPA in this section of the ROD are as follows:

1) An active remedial approach is necessary, because the unacceptable risks to human health and the environment would persist throughout the Hudson River for an unacceptable period of time.
    - There is an unacceptable risk to human health and the environment from the consumption of fish from the Hudson River.
    - The unacceptable risk will continue for many decades without active remediation of the PCB-contaminated sediments and control of the upstream sources.
    - The No Action alternative is not protective of human health and the environment and therefore could not be selected for the Site.
2) A delay of twenty years in reaching target fish concentrations is unacceptable.
    - The Monitored Natural Attenuation (MNA) alternative, which does not include any active remediation of the sediments but does account for future upstream source control, will reduce risks from consumption of fish,

but it is predicted to take at least twenty years longer than the selected remedy to reach target levels in fish tissue in River Sections 1 and 2.

3) The selected remedy is protective because it results in significant reductions in risk, and is cost effective.
    - All of the three active remediation alternatives, REM- 3/10/Select, CAP-3/10/Select, and REM-0/0/3, would be protective of human health and the environment as they permanently remove large volumes of PCBs from the river, which will result in significant reductions in risk from consumption of fish from the Hudson.
    - The lesser cost, and similar reduction in risk, associated with REM-3/10/Select makes REM-3/10/Select more cost effective.
4) A delay of ten years or more in reaching targets is unacceptable.
    - EPA projected that that the target concentration of 0.4 mg/kg PCB in fish fillet (wet weight), which is protective of the average adult who consumes one fish meal from the Upper Hudson every two months, will be attained within 5 years of completion of dredging for the three active remediation alternatives.
    - The target of 0.2 mg/kg PCB, protective of an adult who consumes one fish meal from the Upper Hudson per month, is projected to be attained within 16 years of completion of dredging for the three active remediation alternatives.
    - It is projected to take at least 10 additional years for MNA to reach the 0.2 mg/kg and 0.4 mg/kg PCB target levels, and up to decades longer compared to the active remediation alternatives.
5) The time to reach the ultimate remedial goal of 0.05 ppm PCB in fish was not a factor in remedy selection.
    - The Remediation Goal of 0.05 mg/kg PCB for human consumption of fish, which is protective of an adult who consumes one fish meal from the Upper Hudson per week, will not be attained by any of the alternatives within the modeling time frame (67 years after dredging) in the Upper Hudson River as a whole.
6) The remedy is expected to result in meeting the ultimate remedial goal in the lower river.
    - The Remediation Goal of 0.05 mg/kg also is expected to be attained in the majority of the Lower Hudson River.
7) Institutional controls are an element of the remedy, and represent the sole controls on human health risk after dredging.
    - The selected remedy relies on institutional controls (fish consumption advisories and fishing restrictions) to protect human health until target PCB concentrations in fish are achieved.
    - Institutional controls do not protect ecological receptors.

8) The institutional controls are not completely effective, and the shorter time to reach target fish PCB concentrations to protect fish consumers is a basis for the selected remedy.
    - Human health risk reduction relies on knowledge of and voluntary compliance with the consumption advisories and fishing restrictions.
    - The active remedial alternatives are substantially more protective of people who do not follow the fish consumption advisories, because of the residual risk in consuming fish and the shorter time required to reach fish PCB target levels under those alternatives.

The most important point made in the rationale provided by EPA in the ROD for the selected remedy is that EPA concluded the dredging was needed to accelerate the time it would take to reach the remedial targets for fish flesh in order to quickly reduce human health and ecological risk compared to other alternatives that were evaluated. The targets to protect human health, 0.4 ppm and 0.2 ppm PCB in river-reach and species averaged fish in the upper Hudson, were to be met five and sixteen years, respectively, after the completion of dredging. Additional delays of ten or more years to reach the target fish PCB concentrations were deemed to be not acceptable, or EPA would have selected the "MNA only" remedy. Institutional controls were understood to not be completely effective; the acceleration of the time frame was necessary to protect people who eat fish as well as ecological receptors, both of which are subject to unacceptable levels of risk from consuming PCB contaminated fish from the Hudson River.

Section 4.2 Remedy Implementation after the 2002 ROD

After the ROD was issued in 2002, EPA issued Orders on Consent to GE for the design of the remedy selected in the ROD. These agreements also called for the gathering of baseline water quality and fish data before the start of dredging.

In October 2005, GE and EPA executed an agreement under which GE agreed to perform the first year of dredging work. This agreement also called for a peer review of the results of the first year of work, an opportunity for EPA to revise the scope of work and performance standards set for the work, and provisions for GE to agree to perform the remaining remedial work.

During project design, there was also efforts to protect downstream water supplies from potential impact during the dredging work. There was a Public Water Supply Monitoring Program undertaken by the New York State Department of Health, and construction of a new water transmission pipeline from the City of Troy to serve the Towns of Waterford and Halfmoon.

GE completed construction of the site dewatering facility in Fort Edward between 2007 and 2009. The first year of dredging work ("Phase 1") was performed in 2009.

During Phase 1, approximately 286,000 cubic yards of contaminated sediment was removed from approximately 48 acres of river bottom, dewatered and rail transported to permitted offsite disposal facilities. Initial plans were for 265,000 cubic yards to be removed from 90 acres of river bottom, but it was found that the PCB contamination in several Phase 1 areas extended deeper than anticipated, as a result of a sampling technique applied during design, which resulted in the depth of contamination being underestimated. As a result the volume dredged increased.

EPA performed the Peer Review in 2010, and issued modifications to the scope of work and performance standards. GE agreed in late 2010 to perform the remaining portion of the remedy based on these modifications.

Between 2011 and 2015, GE removed approximately an additional 2.4 million cubic yards of contaminated sediment, which was dewatered and rail transported to permitted offsite disposal facilities. In the areas dredged each year, the replanting of wetland and aquatic vegetation was completed the following year. The last work required as part of project construction scope, habitat planting in the final dredge areas, was completed in 2016. DEC has concerns that the habitat reconstruction work has not been sufficient to address the ecological impacts of the dredging work and that the habitat construction is likely to fail. DEC will continue to work with EPA to seek the needed additional habitat reconstruction.

EPA is currently in the process of finalizing the plans for the monitoring programs to be undertaken as part of the Operation, Maintenance and Monitoring element of the remedy. Initial sediment sampling work was performed in the fall of 2016, but the data are not expected to be available for the FYR; plans for water and fish monitoring have yet to be finalized. This monitoring work out into the future is critical in understanding the performance of the remedy and identifying any potential need for future action to meet the remedial goals. However, monitoring alone is not a substitute for ensuring that the remedy is protective of human health and the environment.

Section 4.3 Operation, Maintenance and Monitoring

EPA has not yet approved work plans for long term monitoring for water quality or fish tissue; currently, the work specified for off season monitoring as part of the Remedial Action Monitoring Plan is being performed by GE. This work includes annual spring sport fish and fall forage fish sampling, and water sampling at Bakers Falls, Rogers Island, Thompson Island, Schuylerville, Waterford, Albany, and Poughkeepsie.

At the time of the preparation of this document, no post remedial fish data for 2016 were available.

No post remedial sediment data are yet available; however, EPA directed GE to begin performing surface sediment sampling in late October 2016, and such data may become available to EPA prior to the writing of the FYR report. Such data may modify the conclusions of this document. The Department has already identified, in a letter to

EPA on November 12, 2016, the need for substantially more sediment data in order to understand the performance of the remedy on both a more highly resolved spatial scale and in a time frame commensurate with the times to reach remedial goals identified in the ROD. There is water data available for 2016. It appears that the water column concentrations and loads are lower in 2016 than in the years before dredging. The decrease is most significant upstream, with the most improvement at Thompson Island. The degree of improvement declines with distance downstream, with lesser improvement at Schuylerville than at Thompson Island, and even less improvement at Waterford. As there are significant year to year variations in flows, and these flow variations can impact both concentration and mass loading of PCBs in water, it is difficult to draw detailed conclusions from the available data other than what is described above.

Section 5 Progress Since Last Review

In this Section, the protectiveness statements from the previous Five-Year Review in 2012 will be reviewed. The status of any recommendations and follow up actions will be provided, along with the results of any implemented actions. The status of any prior issues from the previous Five Year Review will also be provided.

Section 5.1 Protectiveness Determinations in 2012 Five-Year Review Report

The following protectiveness determinations were made by EPA in the previous Five-Year Review report issued in 2012.

Section 5.1.1 Protectiveness Determination for Operable Unit 1

In the 2012 Five Year Review Report, EPA identified that the appropriate protectiveness determination for the Remnant Site remedy, completed as an Interim Remedial Measure in the early 1990s, was "Short Term Protective", stating that:

> "The remedy at the formerly exposed Remnant Deposits at the Hudson River PCBs Superfund Site currently protects human health and the environment as the in-place containment and cap system prevents human exposure, and as perimeter fencing and signage continue to be maintained. However, in order for the remedy to be protective in the long-term, an institutional control needs be implemented to ensure that future use of the Remnant Deposits does not compromise the integrity of the cap system or result in unsafe exposures."

Section 5.1.2 Protectiveness Determination for Operable Unit 2

In the 2012 Five Year Review report, the protectiveness determination for Operable Unit 2 was "Will Be Protective", stating that

> "Based on data collected and reviewed to date, EPA expects that the remedy at OU2 will be protective of human health and the environment upon completion. In the interim, human exposure pathways that could result in unacceptable risks are being controlled."

However, this determination may not have been in compliance with EPA guidance. According to EPA's guidance clarifying the use of protectiveness determinations for Five Year Reviews (OSWER 9200.2-111), "Will Be Protective" is intended for remedies where sufficient data and documentation exists to conclude that human and ecological risks are under control, and no unacceptable risks are occurring in those areas. In addition, the guidance states that to make the "Will Be Protective" determination, the available information must also indicate that the remedy under construction is anticipated to be protective upon completion, and no remedy implementation or performance issues have been identified.

EPA identified in the 2012 Five Year Review report (on page 33) that there would likely be a delay in reaching the ROD targets for reductions in fish PCB concentrations due to

the remedy leaving behind more PCBs, primarily in River Section 2, than anticipated during remedy selection:

> The notable difference between the ROD-anticipated reduction based on the HUDTOX modeling conducted at the time of the ROD and that predicted from the remedial design Sediment Sampling and Analysis Plan (SSAP) core data occurs in River Section 2. The reduction anticipated by the ROD modeling (64 percent) is about twice as much of an improvement for River Section 2 as predicted from the remedial design (36 percent). This indicates that it will likely take River Section 2 longer to reach its ultimate remedial goals than the original forecast in the ROD.

EPA also stated, on page 33, that:

> Nevertheless, EPA believes that the remedial goals could be achieved more quickly, and with a reduced time and extent of injury to ecological receptors, if additional dredging (beyond the ROD requirements) were to be carried out, particularly in River Section 2.

It is also pertinent to note that nowhere in the 2012 Five Year Review Report, or elsewhere in the available record, does EPA conclude that the remedy will be protective upon completion of construction. Rather, EPA stated that only after some period of MNA will the remedy be protective.

Section 5.1.3 Site-wide Protectiveness Determination

For the entire site, EPA also determined that the remedy "Will Be Protective", stating that

> EPA anticipates that once the institutional control has been implemented at OU1 and the dredging and MNA remedy have been completed at OU2, the remedies at the Hudson River PCBs Superfund Site will be protective of human health and the environment. In the interim, exposure pathways that could result in unacceptable risks are being controlled.

EPA relied upon the eventual reaching of the RAOs, at some future date, due to MNA as a basis for stating that the remedy "Will Be Protective"; however, this is also inappropriate, as the remedy would only be protective at the end of the MNA period, several decades into the future, and contradicts the basis upon which EPA selected the remedy, that a delay in abating the uncontrolled ecological and human health exposures was not acceptable.

It is inappropriate under EPA guidance for EPA to state that the remedy "Will Be Protective" in the 2012 Five Year Review, as the exposures at the time were (which remain to the present day) result in human health and environmental risks above the acceptable risk ranges. Also the institutional controls are known to be, as were expected in the ROD, not completely effective controls on the risks.

Construction is now complete for this site. For remedies where construction is complete, EPA classifies them as "Operating Remedial Actions":

> "Operating remedial actions are those actions that are ongoing, but where cleanup levels have not yet been achieved. Such actions typically have remedial components requiring several years to reach cleanup levels (e.g., groundwater and surface water restoration, monitored natural attenuation, soil vapor extraction, and bioremediation)." ("Comprehensive Five Year Review Guidance", OSWER 9355.7-03B-P, page 4-2)

Clearly, Operable Unit 2 of the Hudson River site is now an "Operating Remedial Action", and "Will Be Protective" no longer applies.

Section 6 Five Year Review Process

In undertaking this evaluation, DEC has considered and followed the applicable EPA Guidance on performing Five Year Reviews, including:

- OSWER 9355.7-03B-P: "Comprehensive Five Year Review Guidance" (July 2001)
- OSWER 9200.2-111: "Clarifying the Use of Protectiveness Determinations for Comprehensive Environmental Response, Compensation, and Liability Act Five-Year Reviews" (September 2012)
- OSWER 9355.7-18: "Recommended Evaluation of Institutional Controls: Supplement to the "Comprehensive Five-Year Review Guidance"" (September 2011)

DEC has also reviewed the environmental quality data for the site available through the date of this report, including:

- The data presented in the Reassessment RI/FS leading to the Record of Decision in 2002;
- The data contained in the DEC fish PCB database;
- The data generated during project design after 2002;
- The data generated in the Baseline Monitoring Program before dredging began;
- The data generated in the Remedial Action Monitoring Plan since the start of dredging.

Section 7 Technical Assessment

In the evaluation of the remedial action undertaken by GE in the upper Hudson between 2007 and 2015, the first question to be answered (in accordance with EPA guidance) is Question A:

Section 7.1 First Question in Five-Year Review guidance

Question A: Is the remedy functioning as intended by the decision documents?

Section 7.1.1 Intended Function of the Remedy in the Record of Decision

To answer this question, it is important to first clearly lay out what the decision documents portrayed as the intent of the remedy. The primary decision document is the Record of Decision text, supported by the responsiveness summary and the Feasibility Study.

- The ROD clearly selected a remedy for only the "upper Hudson River" portion of the Hudson River PCBs Site. The Hudson River NPL site extends from Bakers Falls to the Battery in New York City. The "Lower Hudson River" specifically did not have any remedial actions evaluated or identified. However, EPA stated that the remedial actions to be undertaken in the upper Hudson would result in reduced PCB concentrations in the lower Hudson.
- The remediation of the two GE capacitor plants, in Fort Edward and Hudson Falls, was an important element of the overall remedy, but was not part of the EPA lead dredging project. EPA relied upon the State of New York to achieve control over these two historic sources of PCBs to the river.
- The specific expectation in the ROD for the upstream "source control" efforts was that the surface water PCB mass loading at Roger Island, downstream of the plant sites but upstream of the area to be dredged, would decrease to a loading equivalent to an annual average surface water concentration of 2 ng/l of Tri+ PCB.
- The ROD stated that the selected remedy will greatly reduce the mass of PCBs in sediments and lower the average surface sediment PCB concentration, which will in turn reduce PCB levels in the surface water and fish tissue, thereby reducing the level of risk to human and ecological receptors.
- The expectation in the ROD for the decrease in fish PCB concentrations was that the remedy would result in large, rapid declines in fish PCB concentrations in the upper Hudson, such that the reach and species weighted average fish PCB concentration would reach 0.4 parts per million (ppm) five years after dredging was completed. EPA also anticipated that a second target concentration of 0.2 ppm would be reached sixteen years after dredging.
- EPA did not expect to reach the ultimate remedial goal of 0.05 ppm in the average fish PCB concentration in the upper Hudson, but did expect that the remedial work in the upper Hudson to have an impact on the lower Hudson such

that the ultimate remedial goal of 0.05 ppm in average fish PCB concentration would be achieved in the lower Hudson River.

- EPA expected that natural recovery processes after dredging would result in continuing reductions in fish, water, and sediment PCB concentrations, and that these processes would be monitored and the results compared to the anticipated conditions at the time of remedy selection.
- EPA expected that the Institutional Controls (Fish Consumption Advisories and Fishing Regulations) would be maintained and/or modified until the ultimate remedial goal is met.

Section 7.1.2 Elements of the Selected Remedy

EPA, on pages 94-96 of the ROD, articulated the specific elements of the remedy. The primary elements of the remedy are summarized as follows:

- Upstream Source Control at the two GE capacitor plants in Hudson Falls and Fort Edward to achieve a target PCB surface water load at Rogers Island equal to an average surface water PCB concentration of 2 nanograms per liter.
- Targeted Environmental Dredging to remove PCB contaminated sediment from the Upper Hudson to meet specific removal criteria for PCB surface sediment concentration and PCB mass per unit area. This was required to achieve several objectives, including reductions in PCB mass and surface sediment PCB concentrations, targeted reductions in fish PCB concentrations in the time frame identified in the ROD, and reductions in PCB mass transport from the upper Hudson to the lower Hudson.
- Operation, Maintenance and Monitoring (OMM), including monitoring to evaluate the effectiveness of the remedy as well as to ensure that the remedy is protective of human health and the environment. Maintenance of any long term structures (such as caps) is also included.
- Monitored Natural Attenuation (now referred to as Monitored Natural Recovery, or MNR), a reliance on natural processes after the dredging work to continue to result in a decrease in surface sediment PCB concentrations until the ultimate remedial goal is reached.
- Institutional Controls to reduce the potential for human consumption of fish from the Hudson River. These controls are the fish consumption advisories (FCAs), and the current catch and release fishery regulations in the upper Hudson.

Section 7.1.3 Assessment of the Current Status vs. Remedy Intent

The table below lists the remedy elements described above, the intent expressed in the ROD for how the remedy element was to perform, and DEC's evaluation of current conditions and an assessment of whether the remedy is performing as intended.

Table 1: Performance of Remedy Elements as compared to Stated Intent in the Record of Decision (Page 1 of 4)

Remedy Element	ROD Intent	Current Status	Performance as intended by ROD?
Upstream Source Control (Reduction in PCB Mass Load from upstream sources, including the two GE Capacitor plants in Hudson Falls and Fort Edward)	Source control at the GE Hudson Falls plant was projected to decrease the current concentration of PCBs in the water - column of approximately 13 ng/L Tri+ PCB to 2 ng/L Tri+ PCB, by January 1, 2005.	Several years of monitoring data are available for the period after completion of the primary source control measures at the two GE plant sites. The data indicate that the load from the upstream source areas (above Rogers Island) meet or exceed the reductions projected in the ROD.	Yes, performing as intended.
Targeted Removal of Contaminated Sediment in the Upper Hudson (Reduction in PCB Mass Load over the Federal Dam to the Lower Hudson due to sediment removal in the Upper Hudson)	The reduced PCB load over the Federal Dam projected by the selected remedy will ultimately result in reduced concentrations of PCBs in fish, sediment and water. This in turn will result in reduced risks to humans and ecological receptors living in and near the Lower Hudson River from PCB contamination originating in the Upper Hudson River.	Less than one year of post dredging monitoring data available; limited available data suggests that there has been a reduction in PCB load over the Federal Dam as compared to baseline monitoring.	Unknown. Insufficient water, sediment, and/or fish data is available to document any significant trends.; Further monitoring is required to determine if the remedy is performing as intended in reducing PCB loading, resulting in a reduction in sediment, water and fish PCB concentrations in the lower Hudson.

Table 1 (p. 2 of 4)

Remedy Element	ROD Expectation	Current Status	Performing as intended by ROD?
Targeted Removal of Contaminated Sediment in the Upper Hudson (Reduction in PCB Mass and average Surface Sediment PCB Concentrations	Implementation of the selected remedy will greatly reduce the mass of PCBs in the sediments in the Upper Hudson and lower the average PCB concentration in surface sediments, which in turn will reduce PCB levels in the water column and fish and other biota, thereby reducing the level of risk to human and ecological receptors.	EPA and GE are currently engaged in a process of measuring surface sediment PCB concentrations. DEC has demanded a more rigorous sampling program than currently planned. Estimate of average surface sediment PCB concentrations to be left behind after dredging, made in the previous Five Year Review, indicates that higher PCB concentrations in surface sediment were to be left behind than anticipated at the time of the ROD.	Unknown and unlikely. Insufficient surface sediment PCB data is available. It is unlikely the remedy will achieve the reduction in surface sediment PCB concentrations in River Section 2 intended by the remedy at the end of dredging.
Targeted Removal of Contaminated Sediment in the Upper Hudson (Reduction in Fish PCB concentrations in upper Hudson fish in the specified time frames)	The target concentration of 0.4 mg/kg PCB in fish fillet (wet weight), which is protective of the average adult who consumes one fish meal from the Upper Hudson every two months, will be attained within 5 years of completion of the dredging (before or by 2013) for the three active remediation alternatives. The target of 0.2 mg/kg PCB, protective of an adult who consumes one fish meal from the Upper Hudson per month, is projected to be attained within 16 years of completion of dredging	There is not sufficient post dredging fish PCB sampling results from the upper Hudson to compare to the target concentrations to be met in five and sixteen years, respectively. Currently available fish PCB concentrations are well above the targets, but these do not represent post remedial conditions in the upper Hudson.	Unknown. Insufficient data are available in the upper Hudson to quantify the magnitude of the delay in reaching the target concentrations. Currently available fish PCB concentrations indicate ongoing exposures which present unacceptable human health and ecological risks. The elevated average surface sediment PCB concentrations remaining after dredging will delay the time to reach the ROD-specified targets for fish PCB concentrations to be met five and sixteen years after dredging.

Table 1 (Page 3 of 4)

Remedy Element	ROD Expectation	Current Status	Performing as intended by ROD?
Targeted Removal of Contaminated Sediment in the Upper Hudson Reduction in Fish PCB concentrations in Lower Hudson fish as a result of the remedy	The Remediation Goal of 0.05 mg/kg also is expected to be attained in the majority of the Lower Hudson River	There is not sufficient post dredging fish PCB sampling results from the Lower Hudson to compare to the Remediation Goal of 0.05 ppm in fish PCB. Currently available fish PCB concentrations are well above the targets, but these do not represent post remedial conditions in the lower Hudson. PCB concentrations in the Lower Hudson (particularly fish PCB concentrations in the area below Albany) did not change in response to increased PCB load during dredging.	Unknown. Insufficient data are available in the lower Hudson to answer the question as to the magnitude of the delay in reaching the Remediation Goal of 0.05 ppm PCB in fish. However, given the limited impact of the remedy to date on fish in the Lower Hudson below Albany it is not anticipated that there will be further improvements in fish PCB in this area as a result of the dredging. Currently available fish PCB concentrations indicate ongoing exposures present unacceptable human health and ecological risk.
Monitored Natural Recovery (MNR, previously referred to as MNA) and **Operation, Maintenance and Monitoring**	Long-term monitoring would be conducted in sediments, in the water column, and in fish to confirm that contaminant reduction is occurring and that the reduction is achieving Remedial Action Objectives. The monitoring data would also be used as input parameters in the mathematical models to evaluate progress of the natural attenuation processes against the original predictions. (ROD, p. 61)	EPA and GE have not yet finalized the OMM monitoring program to gather the sediment, fish and water data. Initial surface sediment data gathering is ongoing. To date, EPA has not yet begun updating the mathematical models or inputting new data to compare to original predictions.	Unknown. No comparisons of post dredging recovery rates are possible as very limited post remedial data is available.

Table 1 (Page 4 of 4)

Remedy Element	ROD Expectation	Current Status	Performing as intended by ROD?
Institutional Controls	The selected remedy relies on institutional controls (fish consumption advisories and fishing restrictions) to protect human health until target PCB concentrations in fish are achieved.	The institutional controls are in place as envisioned in the ROD. DOH provides annual updates to the Health Advice on Eating Sportfish and Game which pertain to the entire site (both Lower and Upper Hudson), and perform outreach activities in accordance with the established plan, and level of funding set in the Remedial Action Consent Decree.	Unknown and Unlikely. The ROD does not establish a quantitative target, only an expectation that the controls will not be completely effective. Available information indicates that people continue to eat fish despite the institutional controls, and that these exposures represent human health risk beyond the EPA acceptable risk range.

Summary of Evaluation – Question A

It does not appear that the data is available to quantify the degree to which the remedy is or is not performing as intended by the ROD. The currently available fish PCB concentrations throughout the entire site are well above the target concentrations, the first of which is to be met five years after remediation. These current fish PCB concentrations also continue to result in exposures to both human and ecological receptors which are above EPA's acceptable risk range, and the institutional controls are understood to not be completely effective.

The degree to which the remedy has achieved the intended reductions in surface sediment PCB concentrations is unclear, as the data gathering necessary to answer that question has not yet been completed and is insufficient in scope. A more rigorous sampling program than currently planned is necessary, as identified by DEC, in order to provide the data necessary to determine if the current surface sediment PCB concentrations are capable of meeting the intent of the ROD; the current EPA approved sampling plan is not designed to answer that question with the appropriate degree of statistical certainty. For example, the analysis of surface sediment data in the previous Five Year Review report indicated that the intended reductions in surface sediment PCB concentrations were not achieved in River Section 2.

As a result, the remedy will not have achieved the anticipated surface sediment PCB concentrations, making it equally unlikely that the fish PCB concentrations will achieve EPA's ROD targets in the time frames identified in the ROD, within five and sixteen years of remedy completion.

It also appears that the anticipated reductions in fish PCB concentrations in the lower Hudson, as a result of the remedial work in the upper Hudson, will likely not occur as anticipated in the ROD.

The available 2016 surface water PCB data provides an early indication of the performance of the remedy. Surface water PCB concentrations at Rogers Island indicate that in 2016 the goal for upstream source control has been exceeded; concentrations have typically been lower than 2 nanograms per liter. Surface water concentrations at Thompson Island, Schuylerville, and Waterford are lower than those measured during the Baseline Monitoring Program before dredging; however, the degree of improvement appears to decline with distance downstream. The cleanup criteria in the ROD for dredging in River Sections 2 and 3 were approximately 3 times less stringent than River Section 1. The greatest improvement is at Thompson Island, downstream of River Section 1 where the most stringent cleanup criteria were used for dredging. At Schuylerville, downstream of River Section 2, the improvement appears to be more modest; and at Waterford, the downstream end of River Section 3, the improvements are minimal.

It appears, based upon the limited amount of available data, that the degree of improvement in water PCB concentrations diminishes with distance downstream, likely

the result of the less stringent sediment cleanup standards applied below the Thompson Island Dam. It is unclear, due to limited data, if the ROD targets for PCB mass transport reductions will be achieved.

Section 7.2 Second Question in Five-Year Review guidance

Question B: Are the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection still valid?

The exposure assumptions and toxicity data are summarized in Section 8 of the ROD, "Summary of Site Risks", starting on p. 31. According to the EPA's Five Year Review guidance, to answer Question B, the following should be considered:

- Standards and TBCs ("to be considered")
- Cleanup levels, including the basis for the cleanup levels (risk based or Applicable or Relevant and Appropriate Requirements (ARARs))
- Exposure Pathways, including new routes of exposure or new receptor populations
- Toxicity and other contaminant characteristics

Section 7.2.1 Standards and TBCs

It does not appear that any new ARARs, either standards or TBCs, have been identified since the ROD was issued which would impact the understanding of how the remedy is performing.

Section 7.2.2 Cleanup Levels

The cleanup levels set in the ROD for the sediment dredging element of the remedy were risk based; that is, EPA established a cleanup level based upon the anticipated risk reduction associated with the selected remedy. For this site, the reductions in risk to be achieved in the specified time frames through application of the sediment cleanup levels were a function of the anticipated reductions in fish PCB concentrations to be achieved as a direct result of the sediment removal, followed by natural recovery.

For River Section 1, the cleanup level was a Tri Plus PCB mass per unit area (MPA) of 3 grams per square meter, and a surface Tri Plus PCB concentration of ten parts per million. For River Sections 2 and 3, the cleanup level was a Tri Plus PCB MPA of 10 grams per square meter, and a surface sediment concentration of 30 parts per million Tri Plus PCB, a threefold increase over Section 1.

EPA anticipated that the use of these cleanup criteria for sediment would achieve the reductions in fish PCBs in the time frames defined to achieve the risk reduction goals. It is not possible, however, to determine at this time if the basis used to establish these cleanup levels (the understanding of the relationship between sediment, water, and fish PCBs at the time of remedy selection) is still valid today. Only through the interpretation of the sediment, water, and fish PCB concentrations to come out of the post-remedial

monitoring can this understanding be confirmed, or the need to modify this understanding be identified.

At the present time, the available sediment data can be used to extrapolate fish PCB concentrations based upon the existing understanding. In the previous five year review, EPA identified that the surface sediment PCB concentrations remaining in River Section 2 after remediation would be higher than anticipated at the time of the ROD. If the understanding of the relationship between sediment and fish PCB at the time of the ROD is applied, then the expected result is that the reductions in fish PCB concentrations in River Section 2 would be less than anticipated in the ROD, likely resulting in a greater time to achieve the ROD specified fish tissue concentrations. The impact of this on the expected rates of decline associated with natural recovery is unknown.

Summary Cleanup Levels

It appears that the data are not yet available to quantify the degree to which the sediment cleanup levels may need to be modified to achieve the targeted reductions in fish PCB concentrations in the time frames identified in the ROD. . A review of the surface sediment data from the previous Five Year Review report indicates that the cleanup levels would not reach the post remedial risk reduction goals in the specified time frame for River Section 2. Post remedial monitoring is required in water, sediment, and fish to confirm or refute the goal set forth in the ROD that the specified sediment cleanup levels would achieve the intended reductions in water, sediment, and fish concentrations such that the risk reduction targets would be met in the intended time frames. As stated in the ROD on page 66, The time to reach target PCB concentrations in fish was a primary factor in comparing remedial alternatives.

Section 7.2.3 Exposure Pathways, including new routes of exposure or new receptor populations

Air - The most significant route of exposure is still the consumption of fish and other wildlife from the Hudson River. However, some published research suggests the possibility that the air route of exposure may be a significant one. DEC has evaluated the available data from the baseline study completed in the upper Hudson by DEC before the dredging project, from the dredging project air monitoring program, and from published research from the lower Hudson. It appears that the exposure point concentrations are within the DEC standards; however, EPA should verify this hypothesis and gather representative air data to confirm that the air route of exposure is not a significant route of exposure requiring remedial action, particularly in the Lower Hudson.

Walleye – Since the risk assessment work was completed in the mid to late 1990s, it appears that there has been a change in the species mix among sport fish in the Hudson River. Walleye are now much more prevalent than during the 1990s and are now commonly found throughout the Lower Hudson and in the southern portion of the

Upper Hudson. As a sought-after food fish, walleye may represent a significant portion of the overall take of fish for human consumption, particularly in the Lower Hudson. Available data indicate that the PCB concentrations in walleye are 1.5 to 2 times higher than in bass, another commonly sought after game fish, which was the species used in EPA's risk assessment. EPA needs to update the current understanding of risks posed by fish consumption given the change in fish species available for consumption. Surveys of people taking fish from the Hudson would help inform this issue.

Differing Receptor Populations – During the process of implementing the Fish Consumption Advisories, the Department of Health (DOH) has been conducting outreach efforts in both the Upper and Lower Hudson. As a part of these efforts, DOH has been working to identify and reach out to the various ethnic groups, often immigrant, who live in the communities along the Hudson River. Since the risk assessment work was done in the late 1990s, different ethnic groups have moved into the area and have potentially different rates of fish consumption, different preferences for fish species to eat, and different preparation methods. A change in these parameters could result in a different set of assumptions which should be incorporated in the risk assessment process.

Summary - Exposure Pathways

The data may not be available to evaluate whether or not the assumptions made for exposure pathways are still valid.

Two issues related to routes of exposure should be evaluated by EPA; the hypothesis that the exposures via the air route are acceptable and do not require further remediation should be evaluated through the gathering of representative air data. EPA should also evaluate the degree to which the risk assessment assumptions would be modified by the inclusion of walleye as a species available for consumption, particularly in the lower Hudson and the southern portion of the upper Hudson.

Section 7.2.4 Toxicity and other contaminant characteristics

In the 2012 Five Year Review report, EPA stated that:

> "However, the Integrated Risk Information System, or IRIS, EPA's consensus database, is currently re-evaluating the non-cancer toxicity value for PCBs and this value will need to be reassessed at the time of the next five-year review."

The State's understanding of this statement is that the IRIS update had not yet been completed at the time; as a result, EPA was not able to use the updated information on PCB non-cancer toxicity for this review. The State encourages EPA to complete the IRIS evaluation and update as soon as possible, so that the necessary evaluations can be made about the protectiveness of the remedy utilizing the most up to date understanding of PCB toxicity, if possible in this review.

Table 2: Current Validity of Standards, Cleanup Levels, Exposure Pathways, and Toxicity used in Remedy Selection

Question B Element	Still Valid?	Discussion
Standards and TBCs	Yes	No New ARARs Identified
Cleanup Levels, including basis	Unknown	Data are not available to evaluate if the cleanup levels in sediment will achieve the needed reductions in fish PCB concentrations and thus human health and environmental risk.
Exposure Pathways	Unknown	Data are not available to determine if the changes in species availability, and changes in demographics, result in a significant change to the risk assessment inputs and results.
Toxicity and other Contaminant Characteristics	Unknown	EPA has not yet completed the Agency s update to the IRIS database.
Overall	Unknown	

Section 7.3 Third Question in Five-Year Review guidance

Question 3: Has any other information come to light which could call into question the protectiveness of the remedy?

In answering Question 3, DEC has evaluated the available data and site conditions, and has identified two areas where information has come to light which could call into question the protectiveness of the remedy: (1) the Sediment Sampling and Analysis Plan sediment data, which informed EPA that the selected remedy as implemented would result in greater surface sediment PCB concentrations than anticipated in the FS and ROD, and (2) the water and fish monitoring completed during dredging, which showed that the downstream PCB mass flux was not a significant factor in downstream fish PCB concentrations, indicating that the local sediment PCB concentrations were a much more important factor in controlling fish PCB concentrations than thought at the time of remedy selection.

Section 7.3.1 Sediment Sampling and Analysis Plan (SSAP) Data

After the ROD was issued in 2002, EPA issued an administrative Order on Consent to GE, under which GE performed a significant sediment sampling program, the intent of which was to closely define the distribution of PCB concentrations both laterally and with depth. This sediment sampling program included thousands of sampling locations throughout the upper Hudson, and provided the data to allow for an updating of the understanding of the average surface sediment PCB concentrations.

In the 2012 Five Year Review Report, EPA presented a table which summarized the difference in the area-weighted surface sediment PCB concentrations in the upper Hudson between those used in remedy selection, and an updated average taking into account the data gathered in the SSAP after the ROD was issued. This also allows estimates to be made of both the pre-remedial average, and post-remedial average, surface sediment PCB concentrations.

At the time of remedy selection, EPA estimated the average surface sediment concentrations (in parts per million, or ppm) before and after remediation as follows:

Table 3: EPA estimated surface sediment concentrations from 2012 Five Year Review Report

River Section (RS)	ROD Estimate Before Remedy	ROD Estimate After Dredging	ROD Estimated Percent Reduction	SSAP Revised Estimate Before Dredging	SSAP Revised Estimate After Dredging	SSAP Revised Estimated Percent Reduction
RS 1	4.6	0.96	79%	14.2	1.5	87%
RS 2	2.26	0.8	64%	11	7.1	36%
RS 3	0.53	0.51	4.4%	3.3	3.1	4.9%

After the SSAP data is taken into account, it became apparent that the surface sediment PCB concentrations in the upper Hudson were higher than anticipated; a factor of 3.1X in River Section 1, 4.9X in River Section 2, and 6.2X in River Section 3. The remedial approach, to take out PCB contaminated sediment based upon a removal criteria based primarily upon Mass Per Unit Area, did not change. As a result, the average surface sediment PCB concentration after dredging was quite different than anticipated in the ROD, particularly for River Sections 2 and 3.

As can be seen in Table 3 above, the average surface sediment PCB concentration after dredging was anticipated by EPA in 2012 to be about 50% higher in River Section 1 compared to the ROD estimate, a factor of about 9X higher in River Section 2 compared to the ROD estimate, and a factor of about 6X higher in River Section 3 compared to the ROD estimate.

It is also informative to look at the data in terms of the anticipated percent reduction in surface sediment PCB concentrations. In general, the expected reduction in fish PCB concentrations should be proportional to the reduction in surface sediment PCB concentrations. In the table above, one can see that the ROD anticipated percent reduction for River Sections 1 and 3 are similar to the updated anticipated percent reduction using the SSAP data. However, the updated anticipated percent reduction in River Section 2 is little more than half (36% vs. 64%) what was anticipated in the ROD.

It is EPA□s expectation, based on standard geochemical and biochemical theory, that the rates of decay in Tri+ PCB concentrations in water column and fish tissue should

parallel the rate of decay in surface sediment concentrations. This means that the lesser degree of improvement in sediment PCB concentrations should be reflected in a lesser degree of improvement in fish PCB concentrations. However, the data needs to be gathered to determine if this is the case. It is also pertinent to point out that if EPA's expectation above is demonstrated by data, then the fish in River Section 3 should only immediately improve ~ 4% as a direct result of the dredging, and the fish in the lower River, where no sediment remediation was done, should show little additional improvement as a result of the remedy.

Summary – Post ROD SSAP Surface Sediment Data

DEC has reviewed the sediment data made available since the ROD was issued. Two hypotheses are available; the post remedial fish PCB concentrations should be expected to be higher than anticipated at the time of remedy selection, but will the amount of increase be driven by the increase in the absolute concentration, or by the decrease in the amount of improvement from before to after remediation? It is clear that there will be more PCB in fish tissue than what was expected at the time of remedy selection. However the data is not currently available to allow for a quantitative conclusion to be drawn at this time, as to how much higher the fish PCB concentrations will be than reflected in the ROD, nor how much the increased PCB concentrations in sediments, compared to the post dredging conditions assumed in the ROD, will impact the post remedial declines in sediment, water and fish PCB concentrations now that the project is in the Monitored Natural Recovery phase.

Section 7.3.2 Remedial Action Monitoring Plan (RAMP) Fish and Water data

After the ROD was issued, EPA issued a several Orders on Consent to GE to implement various activities, including water, sediment, and fish sampling programs. The water and fish monitoring programs were modified and continued through the period when the dredging work was done. These monitoring programs, which continue to the present time, allow for an understanding of the relationship between water, sediment, and fish PCB concentrations over time. In particular, one can evaluate the relationship between the water column PCB concentrations and mass load (the mass of PCB carried by the river on a temporal, typically daily, basis) and the fish PCB concentrations. In the 2016 White Paper put out by EPA in response to a NOAA publication, EPA stated that fish tissue concentrations south of Albany did not increase during the dredging period when loads from the Upper Hudson increased temporarily, and that these observations suggest that Lower Hudson fish tissue levels may be additionally influenced by local factors that are unrelated to current Upper Hudson conditions such as local PCB sources and inventory of PCB in sediments from past releases.

An evaluation of PCB concentrations in fish in the upper Hudson during dredging also shows a similar pattern. In the vicinity of dredging, the PCB concentrations in fish typically increased in response to the exposures from the work; however, farther

downstream, there was much less or no response, even though the increased PCB concentrations and load from the dredging were noted throughout the upper Hudson and into the lower Hudson. This contrasts with the previously understood conceptual site model, under which the fish PCB concentrations downstream of the dredging should have increased by an amount commensurate with the increase in water column PCB concentrations. This was not observed; rather, as described above, fish PCB concentrations away from the vicinity of the dredging work and immediately downstream had little or no significant reaction to the water column PCBs concentrations during dredging. This indicates to DEC that the exposures to local sediment PCBs were much more relevant to fish PCB concentrations than water column exposures.

The available RAMP fish and water data indicate to DEC that the understanding of relative importance between exposure to PCBs from local sediments, and exposure to PCBs in the water column, should be updated for this ecosystem. In the modeling and assessment work done in the late 1990s to support remedy selection by EPA, a set of estimates were developed to define this relationship. At the time of remedy selection, DEC informed EPA that the modelling and assessment work may have underestimated the relative importance of the sediments; it appears, based upon the fish and water data gathered during dredging, that there is further reason to believe that local sediments play a larger factor in influencing fish PCB concentrations. As a result, the estimates of the relationships between fish, water, and sediment need to be updated.

Summary RAMP Fish and Water Data

The water and fish data gathered during remediation indicate that the local sediments play a larger role in influencing fish PCB concentrations than thought at the time of remedy selection. In the context of the Five Year Review, this means that EPA needs to re-evaluate and re-quantify the relationships between media (sediment, water and fish) which formed the basis upon which cleanup level was determined. Once this is done, EPA can evaluate whether further remedial work is necessary to reach the ROD goals for time to achieve the targeted reductions in fish PCB concentrations.

This means that the scale upon which the remedial program is managed should be modified from a solely River Section based approach to one which more closely reflects the exposures which now are understood to much more important in controlling fish PCB concentrations. As the fish do not travel between pools to any extent due to the locks and dams between pools, fish can only be exposed to the sediments in the pool where they live. Averaging sediment PCB concentration from multiple pools, and comparing these averages with fish PCB concentrations averaged across multiple pools, will dilute out any relationships between the sediment and fish PCB concentrations. It will be very difficult if not impossible to understand if the sediment remedy is functioning as intended in the ROD.

Section 8 Issues

In this portion of the document, DEC will list the issues identified in the assessments above, and how these issues may impact the protectiveness of the remedy.

Section 8.1 Issues related to Question 1

DEC finds that, with one exception, the data are not yet available to determine if the remedy is functioning as intended by the decision documents. It appears that the upstream source control goal is being met, but further surface water data is needed to confirm that the goal is met over the long term. For the remaining remedy elements, additional post remedial monitoring data needs to be gathered, as recommended by the State earlier this year, to compare against the intended function of the remedy as expressed in the ROD and identified above. However, it is clear that under current conditions, the remedy is not protective, as there are ongoing uncontrolled exposures, to both human and ecological receptors, in both the upper and lower Hudson, which are in excess of the EPA acceptable risk range. It is also clear, as EPA stated in the previous Five Year Review report, that the remaining PCB concentrations in sediment, particularly in River Section 2, will result in a delay in reaching the targeted reductions in fish PCB concentrations identified in the ROD. The current lack of data makes it difficult to know how much the delay will be, and to know the degree to which further remediation is needed to achieve the ROD goals.

Section 8.2 Issues related to Question 2

DEC also finds that, with one exception, the data are not yet available to determine if the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives (RAOs) used at the time of the remedy selection are still valid. It appears that no new ARARs or TBCs have been identified to take into account in the remedial program for this site. However, DEC finds that there is not sufficient data available to evaluate if the cleanup levels in the ROD are still valid; to determine if the exposure pathways used in the risk assessments are still valid (due to changes in fish species distribution, and in population demographics among human fish consumers); and to determine if the toxicity assumptions are still valid, as EPA has not yet completed the anticipated update to the IRIS database for PCBs.

Section 8.3 Issues related to Question 3

DEC finds that two important data sets have become available since remedy selection which call into question the protectiveness of the remedy.

First, the SSAP data gathered during project design indicates that higher surface sediment PCB concentrations were left behind after dredging than anticipated during remedy selection. The degree to which this will impact the remedy is unknown without further data gathering; however, it is clear that fish PCB concentrations will be higher than anticipated after dredging, and the rate of decline after dredging may also be impacted as well.

Second, the fish and water data gathered during the dredging work indicate that the degree to which local sediments influence fish PCB concentrations is greater than thought at the time of remedy selection. As a result, there will be little additional improvement in fish PCB concentrations in the lower Hudson, particularly south of Albany, as a result of the dredging. The degree to which there will be improvements in upper Hudson fish will also be impacted; however, this impact is unclear and will require further monitoring, at a spatial scale representative of the exposures from sediments to fish.

Table 4: Issues which prevent the remedy from being protective or may do so in the future (p. 1 of 2)

Issues	Affects Protectiveness (Y N)	
	Current	Future
Insufficient data are available to determine if the targeted sediment removals have resulted in the anticipated reductions in surface water total PCB load to the Lower Hudson.	Y	Y
Insufficient data are available to determine if the targeted sediment removals have lowered the surface sediment PCB concentrations sufficiently to achieve the risk reduction goals by reducing fish PCB concentrations.	Y	Y
Insufficient data are available to determine if the targeted sediment removals will result in meeting the target average fish PCB concentrations in the upper Hudson in five and sixteen years after dredging.	N	Y
Insufficient data are available to determine if the targeted sediment removals will result in meeting the remediation goal of 0.05 ppm PCB in fish in the majority of the lower Hudson River.	N	Y
Insufficient data are available to determine the rates of post-remedial decline in water, sediment, and fish PCB concentrations due to Monitored Natural Recovery are occurring at the rates anticipated in the ROD. EPA has not yet begun to update the mathematical models or inputting new data to compare to original predictions.	N	Y
Available information indicates that, while the Institutional Controls are in place and performing as intended by the ROD, some people continue to eat fish, and these exposures represent human health risk beyond the EPA acceptable risk range.	Y	Y
Insufficient data are available to determine if the cleanup levels are still valid.	Y	Y
Insufficient data are available to determine if the exposure pathway assumptions are still valid.	N	Y
EPA has yet to complete the IRIS update for PCBs	N	Y
SSAP data, available after remedy selection, indicates that higher surface sediment PCB concentrations will be left behind after dredging, leading to higher fish PCB concentrations than anticipated.	Y	Y
RAMP data, gathered during the dredging work, indicates that local sediments are more important than thought at the time of remedy selection, indicating that there will be little improvement in fish PCB concentrations south of Albany, and the monitoring program needs to account for finer spatial resolution which more closely reflects actual fish exposures to sediments.	N	Y

Section 9 Recommendations and Follow-up Actions

Section 9.1 Recommendation 1 - Conduct additional studies improve OMM activities

As described above, DEC finds that there are insufficient post remedial data available to evaluate if the remedy is functioning as intended by the decision documents. Several questions need to be answered now, and in the future, including:

- To what degree have the targeted sediment removals achieved or not achieved the intended reduction in surface water PCB load to the lower Hudson?
- To what degree have the targeted sediment removals achieved or not achieved the intended reductions in surface sediment PCBs?
- Will the targeted sediment removals done during the dredging program result in achieving the targeted average fish PCB concentrations in the time frame identified in the ROD (0.4 ppm in five years after dredging, 0.2 ppm in sixteen years after dredging)? If not, what further removals are necessary to reach these goals?
- When will the targeted sediment removals result in achieving the Remediation Goal of 0.05 ppm PCB in fish in the majority of the Lower Hudson?
- Are the post-remedial declines in sediment, water, and fish PCB concentrations due to MNR occurring at the rates anticipated in the ROD? How do the predictions from updated models, using new data, compare to original predictions?
- Do the exposures, to both human and ecological receptors, continue to result in risks beyond EPA□s acceptable risk range?
- Given the post remedial PCB distribution in sediment, how long will it take to reach the targeted reductions in fish PCB concentrations? Should EPA continue to expect that the targeted sediment removals implemented under the ROD will achieve the expected reductions in risk in the time frames expressed in the ROD?
- Are the assumptions used for the exposure pathways still valid? Are the assumptions for fish species availability, and human demographics and behaviors, still representative?
- Is the current understanding of PCB toxicity up to date?
- What will the overall impact be on the remedy of the finding after remedy selection that higher PCB concentrations in surface sediment will be left behind after remediation?
- What will the impact be on the remedy of the finding after remedy selection that local sediments appear to have a higher influence on fish PCB concentrations? How does this affect the overall conceptual site model?

These questions should form the basis for the Data Quality Objectives to guide the needed additional studies. In many cases, the questions can be answered by monitoring to be done under the □Operation, Maintenance, and Monitoring□or OMM

element of the remedy. However, EPA will need to supplement the work to be done as currently envisioned in the OMM Scope attached to the Remedial Action Consent Decree to accomplish this. The detailed recommendations previously provided by DEC on the scope of OMM data gathering will inform EPA on DEC's position on the needed monitoring.

A fundamental change in conceptual site model needs to be accounted for in managing the remedial program for this site, now in the Monitored Natural Recovery (previously called by EPA "MNA") phase. The appropriate spatial scale (i.e. pool by pool, rather than averaged over multiple pools) should be used in the design of sediment, water, and fish sampling to be undertaken to understand the performance of the remedy.

EPA should also conduct a scientific, broad based survey of people who fish in the Hudson River or who eat fish from the river, in order to inform the risk assessors as to whether or not the assumptions made during the risk assessment in the 1990s are still valid today. If they are not, then EPA should also review the risk assessment calculations to determine if the understanding of site risks need to be updated.

Section 9.2 Recommendation 2 - Prepare to Optimize Remedy

As the monitoring program and additional studies to be performed under Recommendation 1 above move forward, EPA should be considering the additional response actions that are likely necessary to accomplish the goals in the ROD for achieving the targeted reductions in fish PCB concentrations in the time frames set forth in the ROD. In order to do this, EPA will need to update the conceptual site model, including updating the understanding of the relationships between sediment, water, and fish PCB concentrations, to inform evaluations of potential future response actions. This updating of the conceptual site model, and gathering of data to understand the relationships between media, also should be completed on a time frame commensurate with the time frames in the ROD for reaching the targeted reductions in average fish PCB concentrations; that is, in five to sixteen years. The data gathering needs be done on a spatial scale commensurate with the understanding of the degree to which local sediments control fish PCB concentrations, and be designed to answer the questions in time to allow for further response actions in time frames commensurate with meeting goals of the ROD.

Section 9.3 Recommendation 3 – Expand Site Investigation to the Lower River

As described above, the fish and water data gathered during the dredging work indicate that the degree to which local sediments influence fish PCB concentrations is greater than thought at the time of remedy selection. As a result, there will be little additional improvement in fish PCB concentrations in the lower Hudson, particularly south of Albany, as a result of the dredging. In order to complete the site conceptual model and evaluate the need for remedial action for the Lower Hudson, it will be necessary for EPA to perform a Remedial Investigation / Feasibility Study for the portion of the site between the Federal Dam at Troy and the Battery in New York City.

Section 10 Protectiveness Statements

Section 10.1 Basis for protectiveness statements by Operable Unit

In evaluating the appropriate protectiveness statements for this site, DEC has considered the guidance set forth by EPA for determining protectiveness. Three guidance documents are particularly informative; the EPA "Comprehensive Five-Year Review Guidance" (OSWER 9355.7-03B-P), and "Clarifying the Use of Protectiveness Determinations for Comprehensive Environmental Response, Compensation, and Liability Act Five-Year Reviews" (OSWER 9200.2-111). DEC also consulted OSWER Directive 9355.7-18, "Recommended Evaluation of Institutional Controls: Supplement to the "Comprehensive Five-Year Review Guidance"".

The Hudson River PCBs Site has been divided up into several Operable Units by EPA. DEC has focused upon Operable Unit 2 (the sediment remedy for the upper Hudson selected in the 2002 ROD), and the lower Hudson, in this document, as EPA has not performed any additional response actions for Operable Unit 1 since the last Five-Year Review in 2012.

Section 10.2 Operable Unit 1 (Remnant Site Capping IRM)

For Operable Unit 1, DEC finds that the appropriate statement continues to be "Short Term Protective".

As the site conditions for Operable Unit 1 have not changed since the last review, the protectiveness determination should remain the same.

Section 10.3 Operable Unit 2 (Dredging Remedy for the upper Hudson River)

For Operable Unit 2, DEC finds that the appropriate statement is "Not Protective".

DEC evaluated the protectiveness of the remedial action for Operable Unit 2 as a remedy for which construction has been completed. The dredging element of the remedy was completed in 2015; habitat reconstruction efforts were completed in mid-2016. No further construction is to be done in the river as part of this remedy.

This finding of "Not Protective" is based primarily upon the current conditions. There are known exposures to human and ecological receptors which result in risks beyond EPA's acceptable risk range. DEC considered the finding of "Protectiveness Deferred"; this determination would have been appropriate if conditions were such that the available information did not provide sufficient data and documentation that all human and ecological risks are currently under control, and no unacceptable exposures were occurring. While this may be true for Operable Unit 2 for future risks, there is considerable uncertainty and skepticism, due to lack of data and to the current understanding of site conditions as described above, that the fish PCB targets in the ROD will be met in the intended time frame. As a result at the present time, DEC

considers the determination of "Not Protective" to be the sole appropriate finding under EPA guidance for Operable Unit 2.

Section 10.3 Lower Hudson River (From the Federal Dam at Troy south to New York City)

For the lower Hudson, DEC finds that the appropriate statement is "Not Protective".

Although not required by EPA guidance, DEC has evaluated the site conditions in the portion of the site which has not undergone investigation and remedy selection. As such, one may not answer Question A (Is the remedy functioning as intended by the decision documents?). However, following the same logic as for Operable Unit 2, DEC finds that the appropriate statement for this portion of the site, where there are known exposures to human and ecological receptors which result in risks beyond EPA's acceptable risk range, to be the same as for Operable Unit 2 – Not Protective.

Section 11 Next Review

The next formal "Five Year Review" should be completed once five years have passed since the completion of the current review. However, as this remedy is now entering the MNR phase, EPA should engage in a process which regularly updates the conceptual site model to take into account new data as it comes in, and continually updates EPA's understanding of remedy performance. This understanding should include an update and recalibration of the site mathematical models to take into account the updated site conceptual model as well as the data available since remedy selection. EPA should be prepared to optimize the remedy, including evaluations of the need for further active remediation in the form of additional dredging, as needed to achieve the target fish PCB concentrations in the ROD in a time frame commensurate with the selected remedy.

Section 12 Summary and Conclusions

In evaluating the protectiveness of the dredging remedy for the Hudson River PCBs site, DEC has evaluated the current conditions following EPA's guidance for conducting Five Year Reviews. It is clear that the appropriate determination for the dredging remedy, and for the entire site, is "Not Protective".

This determination is based upon the finding that, despite the substantial remedial work done in constructing the dredging remedy between 2009 and 2015, the risks to human health and the environment are well above the EPA acceptable risk range, and due to the understood incomplete effectiveness of the institutional controls, unacceptable exposures are still occurring. The following actions need to be taken to ensure protectiveness:

- Monitoring at the appropriate spatial and temporal scale, in accordance with the recommendations provided by the State to EPA, to evaluate the performance of the remedy, to determine if the remedy will result in meeting the targeted fish PCB concentrations in the ROD in the time frames specified.
- Prepare to optimize the remedy, as needed determined by the monitoring data. The site conceptual model needs to be updated to take into account the data gathered during since the ROD was issued that showed that higher surface sediment PCB concentrations would be left behind than anticipated, and to take into account the finding based upon the data gathered during the remedial action, that the local sediments appear to be much more important in governing fish PCB concentrations than was thought during remedy selection. This should also include the redevelopment and recalibration of the site mathematical models, to help in understanding remedy performance.
- Expand the investigation of the site to include performing a Remedial Investigation / Feasibility Study for the portion of the site between the Federal Dam at Troy and the Battery in New York City. This work is necessary to determine the nature and extent of PCB contamination in the sediments, water, and biota of the Lower Hudson, and evaluate remedial alternatives to address the currently uncontrolled unacceptable risks to human health and the environment.

Attachment N

An Independent Evaluation of the PCB Dredging Program On the Upper Hudson and Lower Hudson River

An Independent Evaluation of the PCB Dredging Program On the Upper Hudson and Lower Hudson River

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Executive Summary

In February 2002, the General Electric Company (GE) was ordered by the U.S. Environmental Protection Agency (EPA) to conduct targeted dredging of PCB-contaminated sediment in a 40-mile stretch of the Upper Hudson[1] River between Fort Edward and Waterford, NY. GE performed dredging of the Upper Hudson in two phases, beginning in May 2009 and ending in October 2015. Following completion of dredging operations, the Hudson River Foundation convened an expert panel to evaluate the effectiveness of the dredging program on the Upper and Lower Hudson. Based on water column and fish monitoring data and other information that were available through December 2016, the panel concluded: (i) the dredging program met mass removal targets for PCB-contaminated sediments, (ii) the dredging program was effective in reducing PCB concentrations in fish from Thompson Island Pool, (iii) post-dredging PCB concentrations in fish downstream of Thompson Island Pool showed mixed results, (iv) the reduction in Tri+ PCB[2] loads to the Lower Hudson during the 2016 post-dredging period were in part due to below-average flows in the river, (v) water column, sediment and fish in the Lower Hudson below Albany are showing slow responses to the Upper Hudson dredging program due to the complexities of sediment transport in the Lower Hudson, and (vi) additional years of natural attenuation will be required to reduce PCB concentrations in fish throughout the Upper and Lower Hudson to acceptable levels. Modifications to the post-dredging monitoring program and continued evaluation of the next few years of monitoring data are therefore recommended to assess if natural attenuation will be sufficient in reducing PCB concentrations in fish in a reasonable time frame or if additional remedial actions will be required.

Key findings and recommendations of the panel are provided below:

<u>Dredging Operations</u>

- During the dredging program, GE met mass removal targets of 2.65 million cubic yards of contaminated sediment and 70,000 kg Total PCBs that were specified in EPA's 2002 Record of Decision. In addition, the overall release of PCBs passing the Waterford monitoring station was

[1] The Federal Dam at Troy, NY serves as the designated boundary between the Upper Hudson and Lower Hudson River.

[2] Tri+ PCB represents the sum of the PCB compounds with three or more chlorines on the biphenyl structure.

less than one percent of PCBs that were removed from the Upper Hudson in accordance with EPA's revised 2010 resuspension engineering performance standard.

PCB Responses in the Upper Hudson

- Post-dredging water column and fish monitoring data provide a preliminary indication of decreases in PCB concentrations in Upper Hudson fish following the completion of the dredging program. Decreases are most notable in Thompson Island Pool, where post-dredging PCB concentrations in pumpkinseed (a small pelagic feeder) and small forage fish were three to six times lower than observed pre-dredging levels. Note that this section of the Upper Hudson was the focus of much of the dredging operations.

- Post-dredging fish monitoring data show mixed results for sections of the Upper Hudson downstream of Thompson Island Pool. PCB concentrations in pumpkinseed were reduced by approximately a factor of two, and were closely linked to reductions in Tri+ PCB water column concentrations for low flow conditions (< 13,000 cfs). Little or no reductions however were observed for PCBs in small forage fish. This result is consistent with the linkage between small forage fish and contaminant levels in localized sediments, and the limited amount of dredging that was performed in river sections downstream of Thompson Island Pool.

- Post-dredging water column monitoring data show that Tri+ PCB water column concentrations at Waterford were reduced by approximately a factor of two for low flow conditions (< 13,000 cfs). However, based on limited post-dredging monitoring data, little or no reduction in Tri+ PCB water column concentrations was observed at Waterford for high flow conditions (> 13,000 cfs). This latter finding may indicate that Tri+ PCB water column concentrations during high flows are more likely derived from resuspension of localized sediments and not from sediments further upstream where dredging operations were more extensive.

PCB Responses in the Lower Hudson

- Based on pre-dredging and 2016 post-dredging monitoring data, we estimated a 66 percent reduction in Tri+ PCB loads passing Waterford and entering the Lower Hudson. This large reduction however was in part due to the below-average flows in the Upper Hudson during 2016.

If 2016 flows were more comparable to the 2004-2008 pre-dredging period, the Tri+ PCB loads would have been reduced by 15-35 percent.

- Tri+ PCB water column concentrations at Albany follow trends that are similar to observed Tri+ PCB concentrations at Waterford. This is in contrast to observed Tri+ PCB concentrations 70 miles downstream at Poughkeepsie, which were very variable and not correlated to observed PCB concentrations at Albany. This discrepancy is believed to be related to the complexity of sediment transport in the Lower Hudson.

- Sediment transport processes in the Lower Hudson will dampen PCB responses and greatly extend PCB response times to changes in Upper Hudson PCB loads. Based on previous sediment transport and contaminant transport modeling studies, we expect that it would take a decade or more to see appreciable changes in PCB water column, sediment and fish concentrations at many locations in the Lower Hudson.

<u>Moving Forward</u>

- Post-dredging monitoring plans should be modified to enhance the overall utility of the water column, sediment and fish data and to help determine the effectiveness of the Upper Hudson PCB dredging program within the next few years. Recommendations include: (i) EPA Method 1668 (a high resolution, congener-based method) should be used to improve the accuracy and reproducibility of PCB water column, sediment and fish measurements, (ii) the USGS suspended sediment monitoring at Waterford should be re-instated to support evaluations of PCB loads to the Lower Hudson, (iii) additional high flow samples should be collected at Waterford to support evaluations of PCB loads to the Lower Hudson for high flow conditions, and (iv) PCB concentrations should be monitored in surface sediments and sediment cores from selected locations in the Lower Hudson to improve our understanding of time responses in the tidal freshwater and estuarine portion of the river.

- Continued evaluation of post-dredging monitoring data, re-assessment of PCB mass inventory in sediment and re-evaluation of PCB model projections should be performed. This work should include congener-based analyses and modeling of specific PCB congeners and/or PCB homolog groups to enhance the interpretation of post-dredging data and increase the reliability of model

projections. This work will be critical in determining if natural attenuation will be sufficient in reducing PCB concentrations to acceptable levels in a reasonable time frame or if additional remedial action will be required.

Introduction

From the late 1940s to the late 1970s, the General Electric Company (GE) is believed to have discharged one or more million pounds of polychlorinated biphenyls (PCBs) into the Hudson River from its capacitor manufacturing plants in Hudson Falls and Fort Edward, New York. A 200-mile stretch of the Hudson River was subsequently designated by the US Environmental Protection Agency (EPA) as a Superfund Site in September 1984 due to elevated levels of PCBs in water, sediment and fish. This stretch of the river extends for approximately 40 miles from Hudson Falls to Federal Dam at Troy (the designated boundary between the Upper Hudson and Lower Hudson River) and then down to the Battery in New York City (see Figure 1).

An initial Record of Decision (ROD) for the Hudson River was issued by EPA in September 1984 (EPA 1984). In the 1984 ROD, EPA recognized that PCB contamination in the Upper Hudson River sediments was a problem, but selected an interim 'No Action' remedy for the contaminated sediments. This decision was based on monitoring data which showed downward trends in PCB concentrations in fish, sediment and water during the late 1970s/early 1980s. In EPA's view, there was also uncertainty in the reliability and effectiveness of remedial technologies that were available at the time of the 1984 decision.

In 1989, a detailed Reassessment Remedial Investigation/Feasibility Study (RI/FS) of the interim 'No Action' decision for the Upper Hudson River sediments was initiated by EPA. The decade-long reassessment study that followed culminated in the issuance of a new ROD by EPA in February 2002 (EPA 2002). Based on the 2002 ROD, GE was ordered to conduct targeted environmental dredging of PCB-contaminated sediment in a 40-mile stretch of the Upper Hudson River. GE began dredging contaminated sediment from the Upper Hudson in May 2009 and completed the dredging program in October 2015 (GE 2016a). In all, 2.76 million cubic yards (MCYs) of PCB-contaminated sediment were removed from the Upper Hudson.

The Hudson River Foundation (a non-profit organization supporting scientific research integral to the development of sound public policy for the Hudson River and its watershed) convened an expert panel to evaluate the effectiveness of the dredging program. The evaluation was performed using water column and fish monitoring data and other information that was available through December 2016 (i.e., 14 months after the dredging program was complete). Summaries of the EPA 2002 ROD and the Upper

Hudson dredging program are reported below and are followed by the panel's evaluations of the effects of the dredging program on the Upper Hudson and Lower Hudson River.

EPA 2002 Record of Decision

Based on the Reassessment RI/FS, EPA determined that there was "an unacceptable risk to human health and the environment from the consumption of fish from the Hudson River" and that "the unacceptable risk will continue for many decades without active remediation of the PCB-contaminated sediments and control of upstream sources" (EPA 2002). EPA cited the key findings from the geochemical and modeling studies that were conducted as part of the reassessment to support its conclusion for an active remedy of the Upper Hudson sediments. These included:

i. Water column and fish monitoring data showed little decline through the latter part of the 1990s.
ii. Long-term sequestration of PCBs in sediment was not supported by analyses of sediment monitoring and water column data.
iii. Sediment deposition was occurring, on average, in most of the Upper Hudson River, but not at rates or with a consistency sufficient for sequestration of PCB-contaminated sediments.
iv. PCB concentrations in water were currently being driven by PCBs stored in sediments.
v. Mass balance model evaluations and projections indicated that elevated PCB concentrations (and associated human/ecological health risks) were expected to continue for decades without remedial action.

As part of the Reassessment RI/FS, detailed model evaluations were performed to evaluate the effectiveness of five remedial alternatives (EPA 2002). These included the No Action alternative, Monitored Natural Attenuation (MNA) with Upstream Source Control at the GE plant sites, and three active remedies that included Upstream Source Control and targeted dredging and/or capping of contaminated sediment followed by MNA. Details are given in Table 1 for the No Action alternative, MNA, and the three active remedial alternatives: capping (CAP-3/3/Select), dredging removal (REM-3/3/Select) and the more aggressive dredging removal (REM-0/0/3).

For the active remedies, the Upper Hudson was divided into three River Sections (RS): Fort Edward to Thompson Island Dam (RS#1), Thompson Island Dam to Northumberland Dam near Schuylerville (RS#2), and Northumberland Dam to Waterford (RS#3). See Figure 1 insert for the locations. In each river section, target criteria were specified based on PCB mass per unit area (MPA); i.e., the total mass inventory of

PCBs underlying a square meter of surface sediment. MPA targets were expressed in terms of Tri+ PCB (which represents the sum of PCB compounds with three or more chlorines on the biphenyl structure). The decision to use Tri+ PCBs in model evaluations was based on: (i) difficulties encountered in consistently quantifying mono- and di-chlorobiphenyls (CBs) concentrations in the historic datasets (EPA 2000a; Connolly et al. 2000), and (ii) studies showing that Tri+ PCBs provided a very good representation of the total PCB concentrations in fish (EPA 2000a, 2002).

Model projections for the five remedial alternatives were given in the 2002 ROD (EPA 2002) for Tri+ PCB concentrations in fish fillets, for whole-body Tri+ PCB concentrations in largemouth bass, and for Tri+ PCB loads (in kg/yr) passing over the Troy lock and dam. These results were subsequently used in assessing human health risks, ecological risks and potential PCB impacts to the Lower Hudson. Typical model results are presented in Figure 2 for average Tri+ PCB concentrations in fish fillets for a 70-year period with active remedies commencing in 2004. Model results for Tri+ PCB concentrations in fish fillets were compared to interim targets of 0.4 mg/kg-wet weight and 0.2 mg/kg-wet weight, and a Tri+ PCB Remediation Goal of 0.05 mg/kg-wet weight. These values are considered to be protective of individuals consuming one half-pound meal of Upper Hudson fish every two months, one month and every one week, respectively.

As shown in Figure 2, MNA with Upstream Source Control was effective in reducing Tri+ PCB concentrations in fish fillets over the longer time period. Both CAP-3/3/Select and REM-3/10/Select reduced the time to reach the interim targets of 0. 4 mg/kg and 0.2 mg/kg in fish by approximately 11 years. The more aggressive dredging remedy, REM-0/0/3, was not considered to provide an appreciable benefit over REM-3/10/Select. REM-3/10/Select was ultimately selected as the required remedy. Further information on EPA's selection of REM-3/10/Select as the required remedy is given in the 2002 ROD (EPA 2002).

Upper Hudson Dredging Program

The selected remedy for the Upper Hudson, REM-3/10/Select, is summarized in Table 2. As shown in the table, the total area of bottom sediment in RS#1 and RS#2 is approximately 500 acres, with over 3,300 acres of bottom sediment in the longer stretch of RS#3. Target sediment remediation criteria that were specified in the 2002 ROD were subsequently modified to include a MPA of 10 g/m^2 Tri+ PCBs for RS#3

(EPA 2004a). Tri+ PCB concentration criteria for the top 12 inches of sediment were also added (EPA 2004a).

Detailed sediment sampling that was conducted during remedial design phase was used to determine areas targeted for dredging. As shown in Table 2, a large portion of the dredging plan focused on contaminated sediments in Thompson Island Pool (RS#1). Select areas downstream of Thompson Island Dam (RS#2, RS#3) with high levels of PCB contamination were also identified for dredging. In all, 2.65 MCY of contaminated sediment and approximately 70,000 kg of Total PCBs (TPCBs) were slated to be removed from the river bottom.

After dredging and covering with clean backfill, average Tri+ PCB concentration in surficial (0-2 inch) sediments were expected to be reduced substantially for RS#1; smaller reductions in surficial sediment concentrations were expected further downstream (Table 2). In addition, Tri+ PCB concentrations in fish fillets from the Upper Hudson were expected to reach an interim target of 0.4 mg/kg-wet in five years, and 0.2 mg/kg-wet in sixteen years after the completion of dredging program. The PCB Remediation Goal of 0.05 mg/kg-wet was not expected to be met in RS#1 and RS#2 within the 70-year model projection, but was expected to be met in RS#3 43 years after the completion of dredging. Due to the lower initial concentrations of PCBs in the Lower Hudson (compared to RS#3), the Remediation Goal of 0.05 mg/kg-wet was also expected to be attained in 43 years after completion of the dredging program for fish from the majority of the Lower Hudson.

Implementation of the Upper Hudson dredging program was carried out in two phases beginning in 2009. Mechanical dredges with enclosed environmental clamshell buckets were used in both phases to remove PCB-contaminated sediment from the river bottom. Phase 1 was conducted by GE from May – November 2009 following strict Engineering Performance Standards (EPS) (EPA 2004b). Among other conditions, the 2004 EPS included the following:

i. Resuspension: dredging-related releases of Tri+ PCBs should not exceed 200 g/day (based on a 7-day running average Tri+ PCB load at far-field monitoring stations located one mile or more downstream of the dredging area),
ii. Residuals: residual PCB sediment contamination in the dredged areas should be less than 1 mg/kg of Tri+ PCB (prior to placement of one foot of backfill material), and

iii. Productivity: sediment dredging should be scheduled to ensure an overall removal of 2.65 MCY of contaminated sediment over the dredging program.

See EPA (2004b) for further details. During Phase 1, approximately 0.3 MCYs of contaminated sediment and 18,000 kg of TPCBs were removed from approximately 48 acres from a six-mile stretch of the Upper Hudson River near Fort Edward (RS#1).

In 2010, a peer-review panel of scientists was convened by EPA to evaluate the effectiveness of Phase 1 dredging. The peer-review panel concluded that "the 2004 EPS for Resuspension, Residuals and Productivity were not met individually or simultaneously during Phase 1 and cannot be met under Phase 2 without substantive changes" (Bridges et al. 2010). The panel recommended that additional evaluations be performed to better delineate the depth of PCB contamination and that dredged areas be covered with backfill (or capped) in a more-timely fashion to limit downriver releases of PCBs. The 2004 EPS were modified accordingly based on recommendations in Bridges et al. (2010). Changes in the revised EPS for Phase 2 included:

i. Resuspension: dredging-related loss of Tri+ PCBs passing the Waterford monitoring station should not exceed 1.0 percent of the total amount of Tri+ PCBs actually removed from the river bottom during the dredging season, and
ii. Residuals: a maximum of two dredge passes should be employed to limit exposure times of contaminated sediment. In areas with high residual contamination after two dredge passes, sediments should be capped prior to placement of backfill.

See EPA (2010a) for further details. Phase 2 was conducted by GE over five dredging seasons (from 2011 to 2015) following the 2010 revised EPS. During this phase, approximately 2.46 MCYs of contaminated sediment and 128,000 kg of TPCBs were removed from approximately 445 acres of river bottom.

A summary of performance results for the Upper Hudson dredging program is given in Table 3. As shown, 2.76 MCY of contaminated sediments were removed from the Upper Hudson during Phase 1 and Phase 2 dredging. In all, 45,680 kg Tri+ PCBs were removed from the river, with 316 kg (or 0.69 percent) of Tri+ PCBs released past the Waterford monitoring station. Comparable measurements for TPCBs show that 146,000 kg TPCBs were removed from the river, indicating that more than two-thirds of the PCBs were present as mono- and di-CBs. The reported monitoring results confirm that GE met mass removal targets of 2.65 MCY of contaminated sediment and 70,000 kg TPCBs that were specified in the 2002 ROD (EPA 2002) and the Productivity EPS (EPA 2004b). In addition, the overall release of PCBs passing Waterford

was below the 1 percent of PCBs that were removed from the Upper Hudson in accordance with the revised Resuspension EPS (EPA 2010a). More detailed evaluation of the effects of dredging on PCB responses in the water and fish of the Upper Hudson and Lower Hudson are described below.

PCB Responses in the Upper Hudson

Water Column Monitoring

Water column samples were collected throughout the pre-dredging, dredging and post-dredging periods at five far-field monitoring stations along the Upper Hudson. These included Rogers Island (RM 194.5), Thompson Island (RM 187.5), Schuylerville at Lock 5 (RM 182.3), Stillwater (RM 168.4), and Waterford (RM 156.0). See Figure 1. Far-field water samples were primarily analyzed for whole-water PCB concentrations using the modified Green Bay Peak (mGBP) Method. During periods of dredging, additional water samples were collected and analyzed using an Aroclor PCB analytical method with an accelerated turnaround time. These samples were used to rapidly assess compliance with the resuspension EPS. Further details on water column monitoring are given in EPA (2010b).

Tri+ PCB Water Column Responses at Waterford

Observed Tri+ PCB water column concentrations at Waterford are presented in Figure 3 for 2004 through 2016. To provide a more complete picture of the time record, Tri+ PCB concentrations that were determined using mGBP measurements as well as those estimated from Aroclor measurements are included on the figure. Tri+ PCB concentrations are plotted on a log scale to highlight variations over a wide range of observed concentrations.

As shown on Figure 3, Tri+ PCB water column concentrations were highly variable throughout the entire record, with observed concentrations ranging from approximately 1 to 1,000 ng/L. During the pre-dredging period (2004-2008), variations in the observed concentrations appear to be related to seasonal effects. In particular, the highest observed Tri+ PCB concentrations generally coincide with spring high flow events. During the dredging period (2009-2015), elevated Tri+ PCB concentrations were generally associated with dredging operations, which typically occurred from June through October. Very high Tri+ PCB concentrations were also observed in several of the spring high flow events that followed dredging operations (e.g., spring 2010 and 2011). Finally, Tri+ PCB concentrations were observed to be lower for the 2016 post-dredging year (as compared to the observed concentrations for the pre-dredging period).

To help illustrate differences in the pre-dredging, dredging and post-dredging periods, Tri+ PCB water column concentrations are presented in Figure 4 as monthly geometric mean (with geometric standard deviations) for the pre-dredging, dredging and post-dredging periods. Pre-dredging values show a seasonal pattern with highest Tri+ PCB concentrations occurring in April (coinciding with spring high flows) as well as in June and July. The high Tri+ PCBs in June and July are likely due to enhanced bioturbation during the early part of the summer (Erickson et al. 2005) and to high flows that occurred in late June/early July of 2006. During the dredging period (May through October), Tri+ PCB water column concentrations were generally two to three times higher than the pre-dredging period. Tri+ PCB concentrations for the post-dredging period were generally lower than the pre-dredging results by approximately a factor of two.

At this point, it is important to note that the Upper Hudson experienced below-average flows for most of the 2016 post-dredging year. Because of the potential effects of flow on Tri+ PCB water column concentrations, the Tri+ PCB water column concentrations at Waterford were plotted as a function of river flow (Figure 5A). On the figure, observed concentrations are presented as geometric means (with geometric standard deviations) for selected flow bins. For both the pre-dredging and post-dredging periods, Tri+ PCB concentrations decreased with increasing flows for river flows less than approximately 13,000 cfs (or 1.6 times the long-term mean river flow at Waterford). For river flows greater than 13,000 cfs, Tri+ PCB concentrations increased with increasing flows. This latter result is expected and is associated with increased flow-induced erosion of the streambed and the accompanying increase in suspended sediment loads (and particulate phase PCB transport) during the higher flows. Observed PCB homolog patterns are consistent with this finding. For six water samples collected during 2016 July-August low flows (< 13,000 cfs) (Figure 5B), PCB homolog distributions are dominated by lower chlorinated PCBs (e.g., tri-CBs) suggesting that dissolved phase PCBs are more important. For four water samples collected during 2016 February high flows (> 13,000 cfs) (Figure 5C), PCB homolog distributions are shifted to more chlorinated PCBs indicating of a greater contribution of particulate phase PCBs in the water column.

Based on the data presented in Figure 5A, regression equations were developed for Tri+ PCB concentrations as a function of flow. The regression equations were based on an approach previously used to evaluate suspended sediment loads in the Upper Hudson at Waterford (da Luz et al. in prep). In this approach, separate regression equations were developed for lower flow (non-flood) and higher flow (flood) conditions. See the Supplemental Information for additional details. The resulting regression

equations for the non-flood and flood conditions are shown on Figure 5A for both the pre-dredging (solid blue lines) and post-dredging (dashed green lines) periods. As shown, the regression line for post-dredging non-flood period is approximately a factor of two lower than the corresponding regression line for the pre-dredging period. Differences in the regression lines for the pre-dredging and post-dredging periods however showed little or no reduction in Tri+ PCB water column concentrations for flood conditions. This result suggests that Tri+ PCBs in the water column during flood conditions are more likely to be derived from the resuspension of local sediments and not from sediments further upstream where dredging operations were more extensive. However, it is important to note that this finding is currently based on a limited number of high flow observations that were available for the 2016 post-dredging period.

Linking Sediment Concentrations to Water Column Responses

A simplified model was developed as part of a preliminary investigation of the linkage between PCB surface sediment concentrations and water column responses during summer-time low flow conditions. In the model, Tri+ PCBs in the water column are assumed to be primarily comprised of dissolved phase contaminant during low flow periods. A schematic of the simplified model is presented in Figure 6. Briefly, the model represents the Upper Hudson as four consecutive "plug-flow" river reaches. The river reaches correspond to the three River Sections, with the longest River Section (RS#3) divided into RS#3a and RS#3b at Stillwater where the Hoosic River enters the Upper Hudson. Processes considered in the model calculation include flow through the river reaches, PCB diffusion out of (and potentially into) the contaminated sediments, PCB volatilization, and addition of flow from tributaries and surface runoff. For pre-dredging model calculations, average Tri+ PCB concentrations in surface sediments (r_a) for each river reach were based on the Sediment Sampling and Analysis Program (SSAP) that was conducted in 2002-2005 during the remedial design phase (see Table 2). Since post-dredging monitoring data are not yet available for the sediment, post-dredging concentrations of Tri+ PCBs in surface sediments were assigned based on estimates given in EPA (2012) and previously presented in Table 2. An analytical solution of the plug-flow equation was used to calculate Tri+ PCB water column concentrations continuously as a function of distance along each of the four river reaches. Further details of the model and model parameterization are given in the Supplemental Information.

The simplified model was calibrated to the pre-dredging period for a summer-time low flow of 3,500 cfs by adjusting only the PCB diffusive exchange coefficient between the water column and the underlying

pore-water (Figure 7A). Calibration results for the pre-dredging period (solid blue line) show a large increase in Tri+ PCB water column concentrations as the water flows through RS#1 (Thompson Island Pool, TIP) and a continued increase in concentration through RS#2 (to Schuylerville). Tri+ PCB concentrations further downstream (RS#3a,b) appear to level off and remain relatively constant with distance. This result could be interpreted as an indication of little or no additional Tri+ PCB inputs from the underlying sediments in RS#3a,b (Schuylerville to Waterford). According to results from the simplified model, a more appropriate explanation would be that additional Tri+ PCB inputs from the downstream sediments are largely being off-set by losses of Tri+ PCBs by volatilization and possibly back-diffusion into sediments. Dilution of Tri+ PCBs by less contaminated tributary and surface runoff flows are also playing a role.

The simplified model was then applied to the post-dredging period by setting the Tri+ PCB concentrations in the surface sediments to estimated values given in EPA (2012) and previously reported in Table 2. Values for all other model coefficients (including the PCB diffusive exchange coefficient) remained the same as in the pre-dredging model calculation. As shown on Figure 7A, the post-dredging model results (green dashed line) show a large decrease in Tri+ PCB water column concentrations at TID (RM 187.5) and at Schuylerville (RM 182.3). Further downstream, model results for Tri+ PCB water column concentrations show an increase between Schuylerville (RM 182.3) and Waterford (RM 156.0). However, the 2016 post-dredging Tri+ PCB concentrations in the water column (green diamonds of Figure 7A) show a different response than the model results. In particular, the observed Tri+ PCB concentrations at TID (RM 187.2) and Schuylerville (RM 182.3) show a smaller decrease in Tri+ PCB water column concentrations than the model calculations for the post-dredging period. Model-calculated Tri+ PCB concentrations however are comparable to the summer-time low flow concentrations at Waterford (RM 156.0).

The discrepancies between the 2016 post-dredging data and the model results at TID (RM 187.5) and Schuylerville (RM 182.3) suggest that the 2016 post-dredging Tri+ PCB concentrations in surface sediments are higher than the EPA (2012) expected concentrations used in the model calculations. Another contributing factor to the higher Tri+ PCB water column concentrations at TID and Schuylerville is that sediments in the dredging zones may need more time to “stabilize” after six years of dredging. For example, the higher Tri+ PCB water column concentrations at TID and Schuylerville may be due to residual effects of dredging disturbances that are continuing to supply localized resuspension of sediments even during summer-time low flow conditions. This would result in higher Tri+ PCB water column concentrations due to the presence of particulate-phase PCBs that were not considered in the simplified

model calculations. It could therefore be argued that one year of post-dredging monitoring data is not sufficient to evaluate the full benefits of the dredging program.

Based on uncertainties associated with post-dredging modeling results for TID and Schuylerville, we focused our attention on model results for Waterford to evaluate the effects of flow on Tri+ PCB water column concentrations. Model calculations were limited to non-flood conditions where flows were less than 13,000 cfs. As shown in Figure 7B, model results for both the pre-dredging (solid blue line) and post-dredging (dashed green line) periods show decreases in Tri+ PCB water column concentrations at Waterford with increasing flows. In addition, the post-dredging modeling results are approximately a factor of two lower than the pre-dredging results. These finding are consistent with the non-flood regressions previously presented in Figure 5A.

Although the simplified model evaluations are limited to non-flood conditions, non-flood conditions occurred 76% of the time during the 2004-2008 pre-dredging period and 96% of the time for the 2016 post-dredging period. Tri+ PCB water column concentrations during non-flood conditions are therefore likely to play an important role in determining PCB exposure and responses in pelagic fish populations.

Fish Monitoring

Fish samples were collected annually throughout the pre-dredging, dredging and post-dredging periods at Feeder Dam (representative of reference conditions), and five sampling locations in Thompson Island Pool (TP1-5) in RS#1, Northumberland and Fort Miller Pools (ND1-5) in RS#2, and Stillwater Pool (SW1-5) in RS#3. Fish species included in sampling were: black bass (including largemouth and smallmouth bass); ictalurids (including bullhead and catfish); yellow perch; yearling pumpkinseed; and small forage fish (including spottail shiner, banded killifish, mimic shiner and fall fish). TPCBs in fish samples were measured using a modification of the USEPA Method 8082 Aroclor Sum Method. In addition, a small subset of the fish samples was analyzed using the mGBP Method to verify TPCB quantification from Aroclor measurements. This check was important to ensure that TPCB quantification from Aroclor measurements was not being affected by changes in PCB congener patterns that may have occurred as a result of dredging. Data from the mGBP method was also used to confirm that TPCBs in fish were primarily composed of Tri+ PCBs. Further details on fish monitoring are given in EPA (2010b).

PCB Responses in Upper Hudson Fish

Our evaluation of PCB responses in fish focused on yearling pumpkinseed and small forage fish because they are expected to show the most rapid response to changing PCB exposure conditions. TPCB concentrations in yearling pumpkinseed and small forage fish are presented in Figure 8 as monthly geometric mean (with geometric standard deviations) for the pre-dredging and post-dredging periods. As shown on the figure, post-dredging TPCB concentrations in pumpkinseed and small forage fish were 3-6 times lower than pre-dredging concentrations for the five locations in TIP. These reductions are greater than the observed reduction in Tri+ PCB water column concentrations in TIP. The observed reductions in TIP fish however are in line with reductions in dissolved Tri+ PCB water column concentrations that were calculated based on EPA (2010) projections for post-dredging Tri+ PCB concentrations in surficial sediments (see Figure 7A).

Although pumpkinseed and forage fish showed similar reductions in TIP, their responses were quite different at further downstream locations. For the Northumberland / Fort Miller (RS#2) and Stillwater (RS#3) pools, post-dredging TPCB concentrations for pumpkinseed (Figure 8A) were approximately two times lower than pre-dredging concentrations. In contrast, post-dredging TPCB concentrations for small forage fish (Figure 8B) were reported to be higher than pre-dredging concentrations in two of the four Northumberland / Fort Miller pools (RS#2). In three of the five locations in the Stillwater Pool (RS#3), post-dredging TPCB concentrations in the small forage fish were approximately equal to the pre-dredging values.

Although differences in pumpkinseed and forage fish responses may in part be attributed to the relatively small number of forage fish (i.e., 2-3 fish per sampling location) that were collected at many locations during the 2016 post-dredging monitoring, differences in feeding behavior also play a role. For example, PCB concentrations in pumpkinseed (a pelagic feeder) would be expected to be linked to water column concentrations. A factor of two decrease in TPCB pumpkinseed concentrations at the downstream locations is therefore consistent with the calculated reduction in dissolved Tri+ PCB water column concentrations that was previously presented in Figure 7A. In contrast, TPCB exposure for forage fish is expected to be more closely linked to PCB sediment concentrations within a very localized area. Based on EPA (2012) projected reductions in surface sediments concentrations (see Table 2), smaller reductions in TPCB concentrations for forage fish would therefore seem to be quite reasonable for forage fish in the Northumberland / Fort Miller (RS#2) and Stillwater (RS#3) pools.

Summary of Upper Hudson Finding

Our evaluations of PCB responses in the Upper Hudson River provide a preliminary indication of decreases in PCB exposures in the Upper Hudson following the completion of the dredging program. Decreases are most notable in TIP, as evidenced by observed TPCB reductions in pumpkinseed and small forage fish. Simplified model results that were performed as part of our evaluations are consistent with observed reductions in TIP fish. Observed reductions in Tri+ PCB water column concentrations however show a smaller decline. This discrepancy needs to be investigated further, and as discussed previously, may indicate that the 2016 post-dredging Tri+ PCB concentrations in surface sediments are higher than expected or that localized resuspension of contaminated sediments is occurring in areas where the sediment bed was highly disturbed by dredging activities.

Decreases in PCB concentrations are also noted, but are less pronounced at the downstream locations (e.g., Stillwater, Waterford). This is illustrated by observed reductions of approximately a factor of two in TPCB concentrations for pumpkinseed in the Northumberland / Fort Miller and Stillwater pools, and in Tri+ PCB water column concentrations at Waterford during low flow conditions. Simplified model results for summer-time low-flow conditions are consistent with the observed reductions at the downstream locations. The model results also suggest the less contaminated sediments in RS#3 will continue serve as a net source of PCB to the water column during the post-dredging period.

In contrast to pumpkinseed results, small forage fish show little or no reductions in observed TPCB concentrations at the downstream locations. This is not an unexpected result based on the direct linkage of forage fish to localized sediments and to the limited dredging of contaminated sediments in RS#3. Further evaluations on the effect of the Upper Hudson dredging program on responses in the Lower Hudson are described below.

PCB Responses in the Lower Hudson

Water Column Monitoring

In addition to monitoring in the Upper Hudson, water column samples were collected at two far-field stations in the Lower Hudson: Albany (RM 140) and Poughkeepsie (RM 77). Fish monitoring was also performed at Albany / Troy, Catskill and Tappan Zee areas. As previously described, water samples were

analyzed for whole-water PCB concentrations using the mGBP or an Aroclor PCB analytical method, and fish samples were primarily analyzed using a modification of the USEPA 8082 Aroclor Sum Method. Further details are given in EPA (2010b – SOW Attachment B).

Tri+ PCB Loads to the Lower Hudson

Tri+ PCB loads to the Lower Hudson were calculated for pre-dredging and post-dredging periods using USGS daily flow measurements at Waterford and daily Tri+ PCB concentrations that were calculated as a function of river flow (see regressions in Figure 5A). Results for 2004-2008 pre-dredging and 2016 post-dredging periods are represented by the first and second stacked bars on Figure 9. For these calculations, 13,000 cfs was used in differentiating between low flow and high flow conditions. As shown in Figure 9, the total Tri+ PCB loads passing Waterford during the 2004-2008 pre-dredging period averaged 107.7 kg/yr, with 47 percent of the total load (50.8 kg/yr) occurring during low flow and the remaining 53 percent (57.0 kg/yr) occurring during high flow conditions. For comparison, the total Tri+ PCB loads passing Waterford during the 2016 post-dredging period was estimated to be 37.0 kg/yr, with 78 percent of the total load (28.7 kg/yr) occurring during low flow and only 22 percent (8.3 kg/yr) occurring during high flow conditions.

Based on results in Figure 9, there is a 66 percent reduction in the total Tri+ PCB load for the 2016 post-dredging period (compared to the 2004-2008 pre-dredging period). This is comprised of a 43 percent reduction in the Tri+ PCB load for low flow conditions and an 86 percent reduction in the Tri+ PCB load for high flow conditions. This large reduction in Tri+ PCB loads during high flow conditions is in large part attributed to differences in river flow for the pre-dredging and post-dredging periods. During the 2004-2008 pre-dredging period, river flow at Waterford averaged 10,100 cfs and included an average of 86 days per year with flows in excess of 13,000 cfs. In comparison, the 2016 post-dredging period was characterized by lower flows with an average river flow at Waterford of 6,100 cfs and only 15 days with flows exceeding 13,000 cfs.

To demonstrate the effect of flow on Tri+ PCB load reduction, a hypothetical post-dredging scenario was considered (last stacked bar, Figure 9). In this scenario, the Tri+ PCB load was calculated using the 2004-2008 pre-dredging flow record with the post-dredging regression equations. Comparison of the pre-dredging results with the hypothetical post-dredging scenario shows that total Tri+ PCB loads would have been reduced by only 13 percent if river flows for the post-dredging period were comparable to flows

during the pre-dredging period. Tri+ PCB loads for low flow conditions were approximately 27 kg/yr for both the 2016 post-dredging period and hypothetical post-dredging scenario. This indicates that year-to-year variations in river flow will have a small effect on Tri+ PCB loads during low flows. However, Tri+ PCB loads during high flows showed large differences. This result indicates that Tri+ PCB loads during high flow conditions will likely show large year-to-year variations; e.g., from 8.3 kg/yr based on the 2016 flow record to potentially more than 100 kg/yr if the river experiences another year like 2011 with three major high flow events.

Tri+ PCB Water Column Responses in the Lower Hudson

Tri+ PCB water column concentrations at Albany (RM 145) and Poughkeepsie (RM 75) are presented in Figure 10 as monthly geometric means (and geometric standard deviations) for the pre-dredging, dredging and post-dredging periods. Tri+ PCB concentrations at Albany (Figure 10A) follow trends that were previously reported for observed concentrations at Waterford (Figure 4). During the dredging period, Tri+ PCB concentrations at Albany were two to three times higher than the pre-dredging results. Post-dredging Tri+ PCB concentrations at Albany were approximately a factor of two or three times lower than pre-dredging concentrations. Finally, for the pre-dredging, dredging and post-dredging periods, Tri+ PCB concentrations at Albany were a factor of two lower than observed concentrations at Waterford (Figure 4) due to effects of dilution by the Mohawk River.

Observed trends in Tri+ PCB concentrations 70 miles downstream at Poughkeepsie (Figure 10B) were less discernible. Monthly geometric means during the dredging period were typically less than the pre-dredging concentrations. Monthly geometric means for the post-dredging period were also found to be very variable and not correlated to observed Tri+ PCB concentrations at Albany. The reasons for these discrepancies in observed Tri+ PCB water column concentrations at Albany and Poughkeepsie are believed to be related to the complexity of sediment transport in the Lower Hudson as described below.

Dynamic Responses in the Lower Hudson

The Lower Hudson is characterized as an estuary with tidal flows affecting the entire 154-mile stretch from Albany to New York City. In addition, saltwater intrusion (and density-driven flows) typically affect transport patterns in the lower 30-50 miles. About 30-35 percent of the freshwater flow into the Lower Hudson is from the Upper Hudson. Another 25-30 percent is attributable to the Mohawk River which enters the Lower Hudson at the head of tide above Albany. The remainder of the freshwater flow is

associated with a number of smaller tributaries (e.g., Catskill Creek, Esopus Creek, the Wallkill River) that enter the Lower Hudson downstream of Albany.

Over a 10-year period (from 2004-2014), approximately 15 million tonnes of suspended sediment was discharged into the tidal freshwater section of the Lower Hudson above Poughkeepsie (Figure 11). A large portion of the suspended sediment load was associated with high flow events (e.g., Tropical Storms Irene and Lee in 2011). Overall, PCB-contaminated sediments from the Upper Hudson account for approximately 35 percent of the incoming suspended sediment load to the Lower Hudson.

In the tidal freshwater section of the Lower Hudson, incoming sediments are continually subject to settling and tidally-induced resuspension. This has two effects on PCB transport: First, suspended sediments (and particulate-phase PCBs) may settle and spend extended periods of time on the river bottom before they are resuspended and transported further downriver. This in effect causes particulate-phase PCBs to be transported through the Lower Hudson more slowly than the river water. Second, only about half of the incoming suspended sediment to the tidal freshwater section of the river is ultimately transported past Poughkeepsie (green dashed line versus blue solid line on Figure 11). This indicates that there is a substantial amount of PCB-contaminated sediments that will be retained in the bottom sediments above Poughkeepsie. Although sediment deposition rates in the tidal freshwater Hudson will show large spatial variations, the average net-accumulation of sediment in the tidal freshwater section of the river is expected to be on the order of 5-10 mm/yr. If we assume that the top 5-10 cm of surficial sediment is relatively well mixed by physical processes and bioturbation, a decade or more would be required to bury recently-deposited sediments below the surficial sediment layer. In addition to tidal effects, trapping of contaminated sediments along the river bottom is further complicated in the downriver section by density-induced (or estuarine) circulation which causes a net movement of bottom waters in the landward direction and enhanced trapping of contaminated sediment.

A more detailed picture of sediment transport in the Lower Hudson is provided by sediment transport modeling results for three moderate high flow events during an 80-day period corresponding to the 2014 spring freshet (Figure 12). Figure 12(a) shows the flow record at Green Island (which includes the flow contributions from both the Upper Hudson and Mohawk Rivers). The tidal signal at the Battery in New York City is also included in the figure. Model results for cross-sectional averaged suspended sediment concentrations from river inputs are presented as a color contour plot in Figure 12(b). In this panel,

distance along the Lower Hudson from Albany (Km 250) to the Battery in New York City (Km 0) is plotted on the y-axis and time is plotted along the x-axis. The three gray dashed lines on the figure indicate the travel time of water along the river for the three high flow events. On average, the river water during the high-flow events takes several days to reach Poughkeepsie (Km 125) and 10-20 days to reach the Battery in New York City, and much longer during low-flow periods. In comparison, the incoming sediment mass takes three to ten or more times longer to reach the Battery, depending on the sediment settling velocity. As the incoming suspended sediments are transported downriver, the sediment signal becomes more dispersed and a large portion of the sediment mass is deposited along the river bed (Figure 12(c)). The net deposition of new sediment along the river bed is presented in Figure 12(c) as the total sediment mass (suspended + bed) at the end of the model period (blue line) and as the normalized cumulative mass distribution of new sediment from the Battery to the Troy lock and dam (gray line). During the simulated period, approximately 65 percent of the incoming suspended sediment was deposited above Poughkeepsie and the remaining 35 percent was deposited further downriver.

The effect of sediment dynamics on PCB transport in the Lower Hudson has been examined in previous modeling studies (Farley et al. 1999, 2006). The continuous interaction of the overlying water with sediments (through setting, resuspension, and pore water exchange) and the large capacity of the sediments to sorb PCBs work together to dampen the PCB responses downstream and to greatly extend PCB response times to changes in Upper Hudson PCB loads. This finding was supported by model simulations that were performed as part of the Contamination Assessment Reduction Project (CARP) (HydroQual 2008). Based on these studies, we expect that it would take a decade or more to see appreciable changes in PCB concentrations at many locations in the Lower Hudson.

Summary of Lower Hudson Finding

Our evaluations of PCB responses in the Lower Hudson River show that the 66 percent reduction in Tri+ PCB loads for the post-dredging period was in large part the result of low river flows during 2016. Higher Tri+ PCB loads to the Lower Hudson are therefore likely to occur if the river experiences moderate or high flows over the next few years. Under these conditions, reductions in Tri+ PCB loads to the Lower Hudson are more likely to be in the range of 15-35 percent (compared to the pre-dredging period).

With the exception of very low flow years, Tri+ PCB loads to the Lower Hudson are expected to be dominated by particulate-phase transport of PCBs during high flow. PCB responses will therefore strongly

depend on suspended sediment loads and sediment transport processes in the Lower Hudson. Our work in this area indicates that a large portion of the suspended sediment (and particulate-phase PCBs) that is discharged into the Lower Hudson will be deposited along the river bottom. Because of physical and biological processes, the newly-deposited sediment will mix with the top 5-10 cm of bottom sediment. Tri+ PCB concentrations in surficial sediments of the Lower Hudson are therefore expected to reflect an average of the past 5-10 years of incoming PCB loads. Bottom sediments at Poughkeepsie and locations further downriver are therefore expected to show delayed responses to annual changes in Tri+ PCB loads. Since Tri+ PCB concentrations in the overlying water are expected to be largely controlled by tidally-induced resuspension of localized sediments, similar delayed responses are expected for Tri+ PCB water column concentrations.

Moving Forward

Results from our evaluations indicate that the Upper Hudson dredging program has been most effective in reducing PCB exposure levels in TIP, where 53 percent of the river bottom was targeted for dredging. PCB reductions in TIP were most noticeable for pumpkinseed and small forage fish, which showed a factor of three to six decrease from the 2004-2008 pre-dredging period to the 2016 post-dredging period. Smaller reductions (by approximately a factor of two) in the 2016 post-dredging monitoring data were observed further downstream toward Waterford for Tri+ PCB water column concentrations and TPCB concentrations in pumpkinseed. However, little or no reductions were observed for TPCBs in small forage fish at the downstream locations. This latter finding reflects the linkage of TPCB concentrations in forage fish to localized sediment contamination levels and the limited areas that were targeted for dredging between Schuylerville and Waterford. Finally, PCB responses in the Lower Hudson (e.g., at Poughkeepsie) did not exhibit any clear trends, and as described previously, appear to be responding very slowly to changes in PCB inputs from the Upper Hudson.

Based on 2016 post-dredging monitoring, TPCB concentrations in fish throughout the Upper and Lower Hudson remain above interim target levels and remediation goal specified in the ROD (EPA 2002). As described in the ROD (EPA 2002), additional years of MNA will be required to meet TPCB target levels and remediation goals for fish. Post-dredging monitoring is therefore expected to continue into the foreseeable future to determine if MNA will be effective in reducing PCB concentrations to acceptable levels or if additional remedial action will be required. Current monitoring plans for the water column

and fish in the Upper Hudson are described in EPA (2010b). Additional information on sediment sampling is provided in GE (2016b). The basic elements of the current monitoring plan include:

- Water Monitoring: Weekly sampling of the water column at four far-field monitoring stations in the Upper Hudson, and monthly collection of water column samples at Albany and Poughkeepsie in the Lower Hudson. Whole water samples are to be analyzed using the mGBP method (with a subset of samples analyzed using EPA Method 1668).
- Sediment Monitoring: Collection of surface sediment samples in the dredged and non-dredged areas of the Upper Hudson every three years. For 2016-2017 sediment survey, 226 samples are being collected and will be analyzed for PCBs (using Aroclor PCB Method GEHR9082) and TOC (using the Lloyd Kahn Method). At this time, there are currently no plans for sampling sediments in the Lower Hudson.
- Fish Monitoring: Annual collections of a variety of fish species at a number of sampling locations in TIP, Northumberland / Fort Miller Pools and Stillwater Pool. Fish samples will also be collected in the Lower Hudson once every two years at Albany, Catskill and Tappan Zee. TPCB concentrations for whole body and fish fillets will be analyzed using a modification of USEPA 8082 Aroclor Sum Method (with a subset of samples analyzed using the mGBP Method).

Although current monitoring plan provide a reasonable framework for assessing the effectiveness of the Upper Hudson PCB dredging program, we recommend the following modifications be incorporated into the plan to enhance the overall utility of the water column, sediment and fish monitoring data. Recommendations for complementary modeling studies are also provided.

Water Monitoring

- EPA Method 1668 (a high resolution, congener-based method) should be used in analyzing PCB water column concentrations in the Upper and Lower Hudson. EPA Method 1668 will provide a more reliable and reproducible measure of TPCB and Tri+ PCB water column concentrations, particularly as PCB concentrations decrease in time. In addition, EPA Method 1668 will provide a more accurate measure of PCB congener concentrations that can be used in supporting congener fingerprinting analyses and more detailed model evaluations. Finally, PCB analyses using EPA Method 1668 will be back compatible with previous PCB analyses for the Lower Hudson that were conducted as part of the Contamination Assessment Reduction Project (CARP).

- Since a large portion of the Tri+ PCB load to the Lower Hudson is likely to be associated with particulate-phase PCB transport, the USGS suspended sediment monitoring should be re-instated at Waterford. This information is critical in efforts to quantify sediment loads and particulate-phase PCB loads to the Lower Hudson.
- To date only a limited number of post-dredging water column samples have been collected at Waterford during high flows. Additional high flow sampling at Waterford is needed to support evaluations of PCB loads to the Lower Hudson during high flows.
- In the Lower Hudson, PCB water column concentrations are likely to be controlled by tidally-induced resuspension of localized sediments. PCB concentrations in whole water samples are therefore expected to vary over the tidal cycle and from one tidal cycle to the next. Water samples for the Lower Hudson stations should be analyzed for dissolved and particulate concentrations to distinguish effects of tidal resuspension of bottom sediment.

Sediment Monitoring

- Analysis of 2016-2017 surface sediment samples should provide a useful "post-dredging" baseline of Tri+ PCB concentrations in surface sediments. However, analysis of the surface sediment samples by EPA Method 1668 would provide more accurate and reliable measure of Tri+ PCB concentrations, especially since PCB congener patterns are likely to change during the post-dredging period. In addition, more accurate PCB congener-based sediment data could be used in congener finger-printing and other model evaluations.
- Sediments will play a major role in determining PCB exposure concentrations and response times for the Lower Hudson. PCB concentrations in sediment should also be monitored in the Lower Hudson. This should include the collection of surface sediments at selected locations in the tidal freshwater and estuarine reaches of the Lower Hudson. Sediment cores should also be collected and used in developing sediment core chronologies at select locations. This information will be critical in documenting time responses in PCB contamination levels in the Lower Hudson.

Fish Monitoring

- A special study that was conducted to evaluate the potential effects of sample preparation on TPCB measurements showed a surprisingly large analytical variation for paired fillet samples. The observed variations in paired samples is attributed to differences in sample preparation and potential inaccuracies in quantifying TPCB concentrations based on Aroclor identification. Efforts

should therefore be continued to ensure consistency in fish sample preparations (including fillet preparation). In addition, EPA Method 1668 should be used in place of the less accurate Aroclor Method in analyzing PCB concentrations in Upper and Lower Hudson fish.

- Intra-species variability in fish populations can result in large variations in TPCB concentrations in fish. Increasing the number of fish samples should therefore be considered to ensure that the fish results provide the required statistical power for evaluating not only the attainment of interim targets and the remediation goal, but also changes or trends in TPCB concentrations in fish over time.

Modeling

Even the most elaborate monitoring program can only provide snapshots of PCB contamination levels in a system as complex as the Hudson. We therefore strongly recommend the following congener finger-printing analyses and model evaluations.

- Congener fingerprinting analyses of the water, sediment and fish data should be performed to help identify temporal changes in PCB sources (e.g., background, GE plant site, TIP sediments, downriver sediments) during the post-dredging period.
- Simple and complex models should be used to assist in interpreting Tri+ PCB, PCB homolog and/or PCB congener monitoring results for the Upper Hudson. For example, model results should be used in evaluating the relative importance of diffusive exchange in controlling losses of PCBs from surface sediments and to investigate the potential of PCB migration from deeper sediments.
- Model evaluations should be used to help track year-to-year changes in PCB inventory in Upper Hudson sediments and to update projections for PCB concentrations in fish during the post-dredging recovery period.
- Finally, model evaluations should be performed for the Lower Hudson to further our understanding and update projections of PCB time responses in the tidal freshwater and estuarine sections of the river.

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Table 1. Summary of Remedial Alternatives Considered in Model Projections for the 2002 Record of Decision (EPA 2002)

Remedial Alternative	Description	Sediment Remediation Criteria[1]	Implementation Period[2]
No Action	Relies solely on natural attenuation processes (e.g., burial by cleaner sediments, biodegradation, bioturbation and dilution) to reduce PCB concentrations in sediments and surface water.		
Monitored Natural Attenuation (MNA), with Upstream Source Control[3]	Natural attenuation processes with a separate source control action near the GE Hudson Falls plant.		
CAP-3/10/Select[4]	Capping (after removal of 1.73 MCY of sediment to allow for cap placement). A layer of fill material would be placed on top of the cap to limit scour.	3 g m^{-2} in RS #1 10 g m^{-2} in RS #2 Select areas in RS #3	6 years followed by MNA
REM-3/10/Select[4,5]	Removal of 2.65 MCY of sediment. Dredged areas would be covered by a layer of fill material.	3 g m^{-2} in RS #1 10 g m^{-2} in RS #2 Select areas in RS #3	6 years followed by MNA
REM-0/0/3[4]	Removal of 3.82 MCY of sediment. Dredged areas would be covered by a layer of fill material.	0 g m^{-2} in RS #1 0 g m^{-2} in RS #2 3 g m^{-2} in RS #2	8 years followed by MNA

[1] Sediment remediation criteria are expressed on a mass per unit area basis; i.e., the total mass inventory of PCBs underlying a square meter of surface sediment.

[2] For model projections, the implementation period for the active remediation alternatives was assumed to commence in 2004.

[3] For model projections with Upstream Source Control, Tri+ PCB load upstream of Thompson Island Pool is considered to be reduced from 0.16 kg/day to 0.0256 kg/day by January 1, 2005.

[4] The three active remedies (CAP-3/10/Select, REM-3/10/Select, REM-0/0/3) all include Upstream Source Control.

[5] For REM-3/10/Select model projections, Tri+ PCB releases during dredging were assumed to be equal to 0.13% of the total Tri+ PCBs removed from the river bottom. Tri+ PCB releases during dredging were not considered in model projections for CAP-3/10/Select and REM-0/0/3 based on sensitivity model runs for REM-3/3/Select which showed little or no difference in projected Tri+ PCB concentrations in fish for assumed Tri+ PCB releases of 0, 0.13% and 2.5% of the total Tri+ PCBs removed from the river bottom.

Table 2. Summary of Proposed Remedy for the Upper Hudson (REM-3/10/Select) [1]

	River Sect. #1	River Sect. #2	River Sect. #3	Total
Section length (miles)	6.3	5.1	29.5	40.9
Total Area (acres)	528	463	3,360	4,351
Tri+ PCB sediment remediation criteria (mass per unit area, concentration in top 12 inches) [2]	3 g m^{-2} 10 mg kg^{-1}	10 g m^{-2} 30 mg kg^{-1}	10 g m^{-2} 30 mg kg^{-1}	
Area remediated (acres, percent of total)	282 (53%)	76 (16%)	135 (4%)	493
Volume sediment removed (MCY)	1.56	0.58	0.51	2.65
Total PCB mass removed (kg) [3]	36,000	24,300	9,500	69,800
Average Tri+ PCB surface (0-2 inch) sediment concentration: before / after dredging (mg kg^{-1}) [4]	14.2 / 1.9	11 / 7.1	3.3 / 3.1	
Expected time to reach 0.4 / 0.2 / 0.05 mg kg^{-1} Tri+ PCBs in fish fillets (years after dredging is complete)	5 / 16 / > 70	5 / 16 / > 70	5 / 16 / 43	

[1] Values as reported in the 2002 ROD (EPA 2002) except where noted.

[2] Target sediment remediation criteria, as initially specified in the 2002 ROD, were subsequently modified to include a MPA of 10 g/m^2 Tri+ PCBs for RS#3 and a Tri+ PCB concentration criteria for the top 12 inches of sediment (EPA 2004a). Other factors such as sediment texture, depth and bathymetry were also considered in delineating dredging areas.

[3] PCB mass removals were reported in the 2002 ROD (EPA 2002) in terms of Total PCBs (and not Tri+ PCBs).

[4] Average Tri+ PCB concentrations (before dredging) were based on the Sediment Sampling and Analysis Program (SSAP) that was conducted in 2002-2005 during the remedial design phase. Average Tri+ PCB concentrations (after dredging) were estimated based on expected post-dredging concentrations of 0.25 mg kg^{-1} Tri+ PCBs in surface sediment. See EPA (2012) for details.

Table 3. Performance Summary for the Upper Hudson Dredging Program[1]

	Year	Dredged Season	Dredged Sediment (MCY)	Total PCBs Removed (kg)	Tri+ PCBs Removed (kg)	Tri+ PCBs Released Past Waterford	Tri+ PCBs Released Past Waterford (%)
Phase 1	2009	May 15 – Oct 27	0.296	18,230	5,350	71.3	1.3
	2010						
Phase 2-1	2011	Jun 6 – Nov 8	0.363	27,200	9,070	29.8	0.33
Phase 2-2	2012	May 9 – Nov 16	0.663	33,370	10,080	30.6	0.30
Phase 2-3	2013	Apr 29 – Nov 5	0.628	32,460	9,275	99.3	1.07
Phase 2-4	2014	May 7 – Nov 4	0.583	26,570	8,915	39.8	0.45
Phase 2-5	2015	May 7 – Oct 3	0.230	8.185	2,991	44.7	1.49
Total			2.764	146,000	45,680	316	0.69

[1] Values as reported in GE (2016a).

Figure 1. The Hudson River PCB Superfund Site extending from Hudson Falls, NY to the Battery in New York City. The Federal Dam at Troy is the designated boundary between the Upper Hudson and Lower Hudson. From EPA (2011).

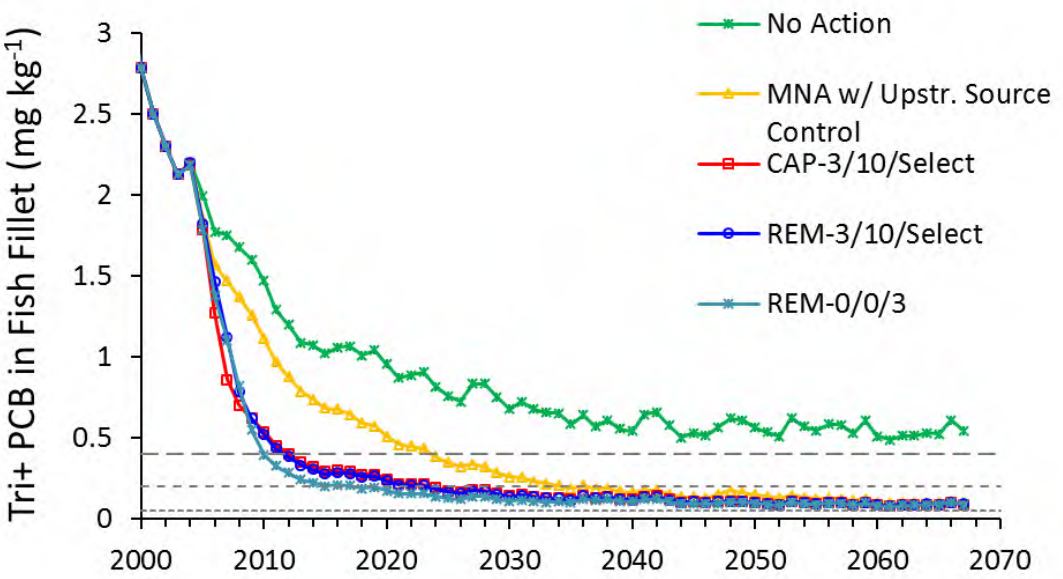

Figure 2. Model projections for Tri+ PCB concentrations in fish fillets from the Upper Hudson. Model results are expressed in terms of a species-weighted diet (36% brown bullhead, 6% carp, 2% eel, 38% bass, 9 % walleye, 9% perch) with river section averages weighted by river section length (15.4% for RS #1, 12.5% for RS #2, 72.1% for RS #3). The three dashed gray lines correspond to the interim targets of 0.4 mg/kg-wet weight and 0.2 mg/kg-wet weight, and Tri+ PCB remediation goal of 0.05 mg/kg-wet weight for protection of human health. Model projection from Table 11-2 (EPA, 2002).

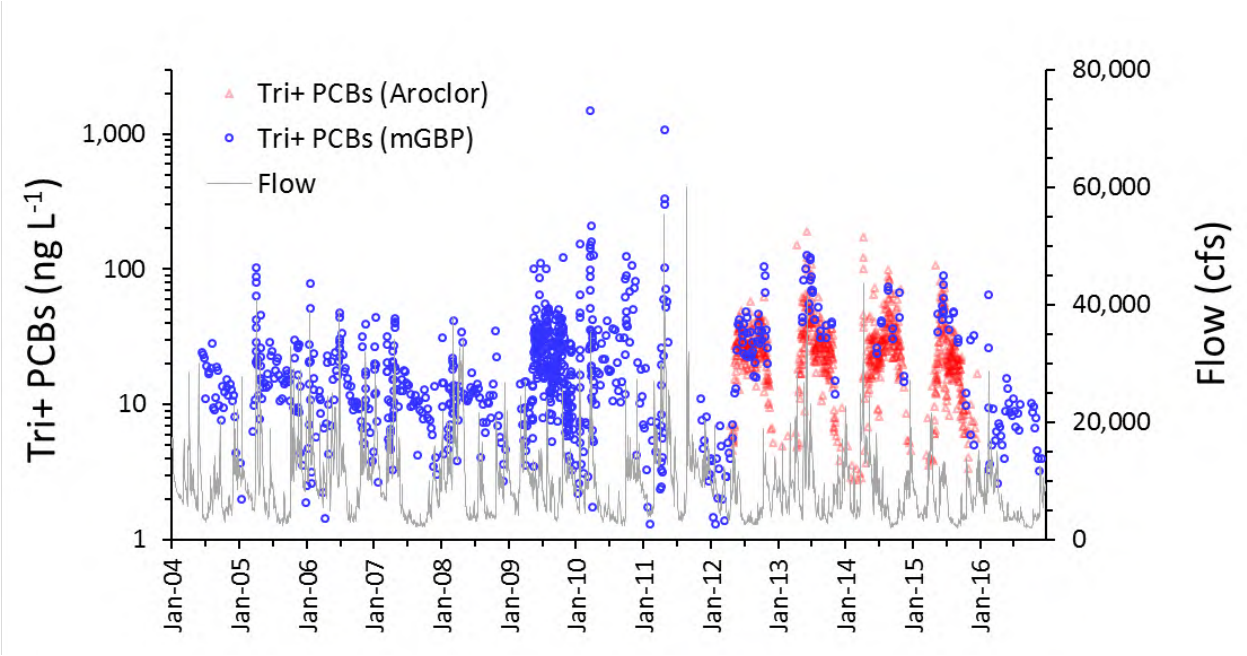

Figure 3. Tri+ PCB whole water concentrations at Waterford for pre-dredging (2004-08), Phase 1 dredging (2009), Phase 1 evaluation (2010), Phase 2 dredging (2011-15) and post-dredging (2016) periods. Tri+ PCBs analyses were determined using a modified Green Bay Peak Congener method (mGBP) with non-detectable peaks set equal to zero or were estimated from PCB Aroclor measurements (Aroclor). Daily flow measurements at Waterford are included for comparative purposes.

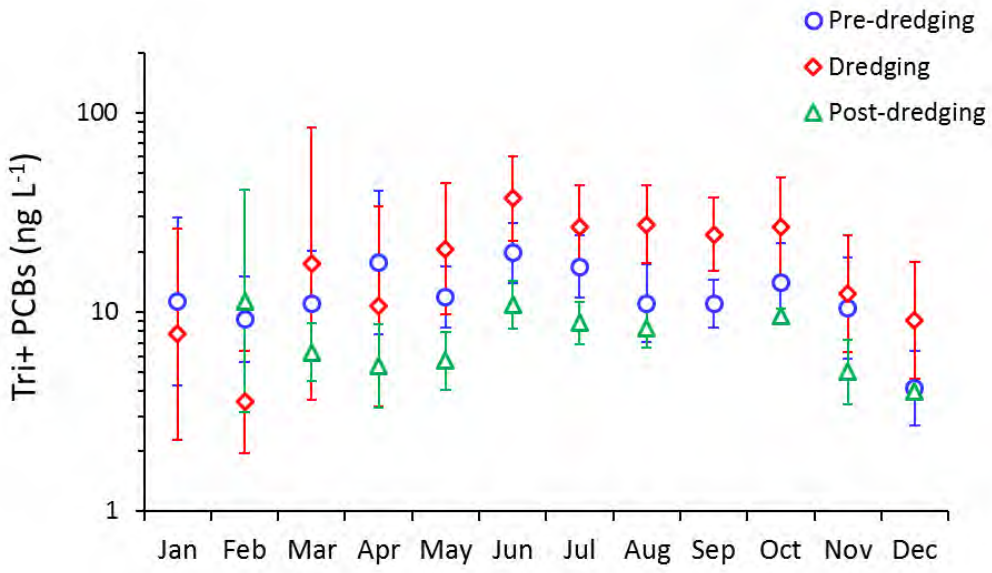

Figure 4. Monthly geometric means (and geometric standard deviations) for Tri+ PCB water column concentrations at Waterford. Whole water concentrations were determined using either the modified Green Bay Peak (mGBP) or an Aroclor PCB analytical methods. Pre-dredging period is based on 2004-2008 measurements; Dredging period is based on 2009-2015 measurements and includes the Phase 1, Phase 1 evaluation and Phase 2 dredging years; Post-dredging period is based on 2016 measurements.

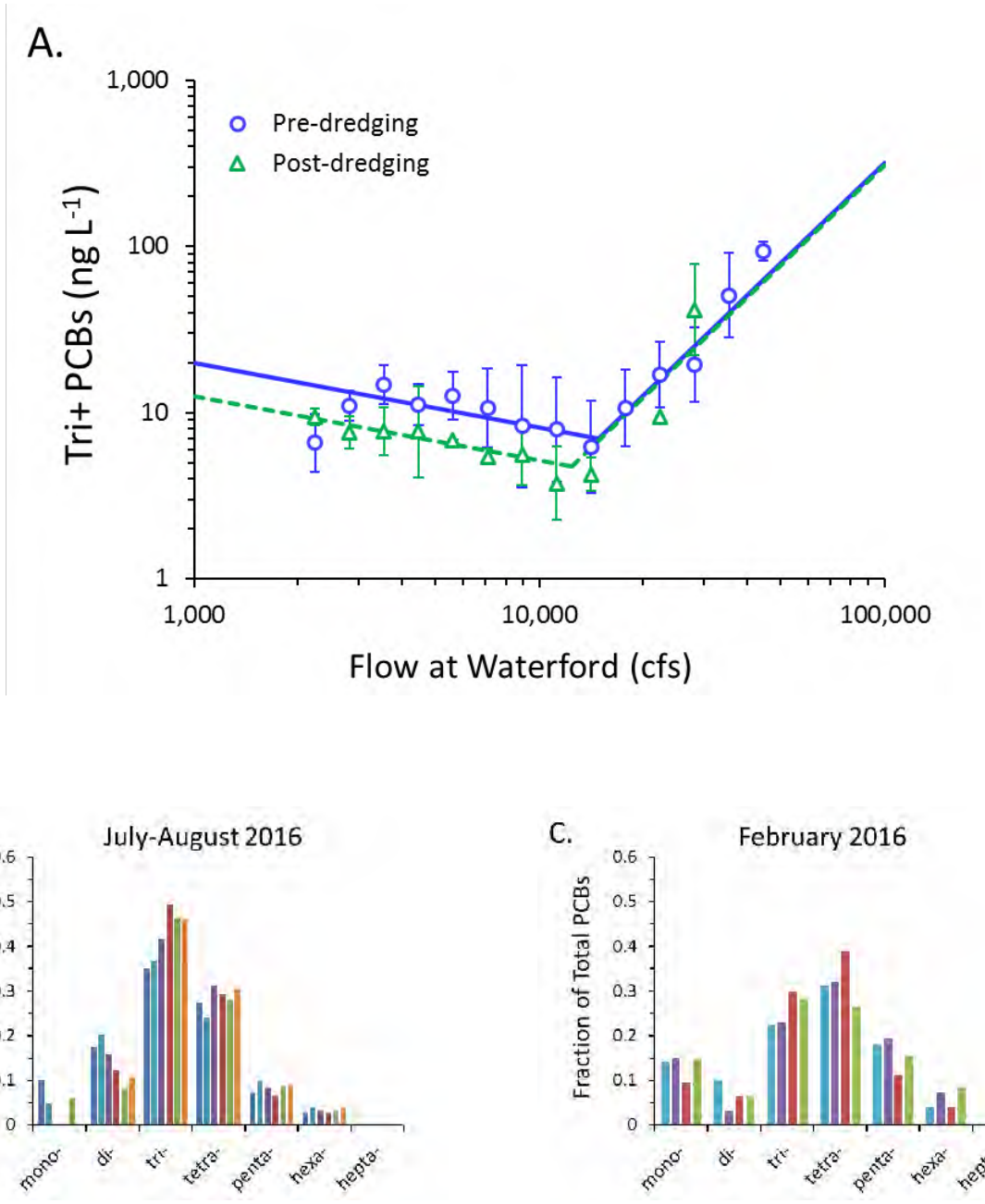

Figure 5. Tri+ PCB water column responses at Waterford. (A) Tri+ PCB concentrations versus flow. Pre-dredging (open blue circles) and post-dredging (open green triangles) are represented as geometric means (with geometric standard deviations) for selected flow bins. The corresponding regression equations are given by the solid blue lines and the dashed green lines for non-flood and flood flow conditions. (B) PCB homolog distributions for six water column samples (color bars) collected during 2016 July-August low flows (< 13,000 cfs). (C) PCB homolog distributions for four water column samples (color bars) collected during 2016 February high flows (> 13,000).

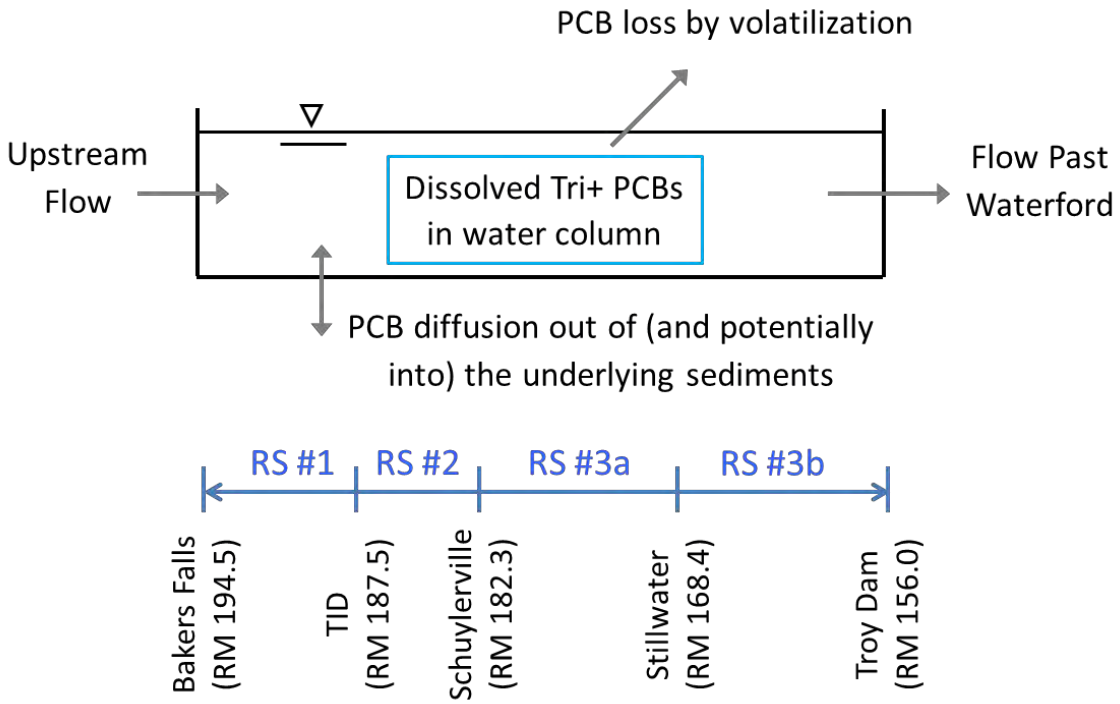

Figure 6. Simplified model for Tri+ PCB transport through the Upper Hudson during summer-time conditions. The model represents the Upper Hudson as four consecutive "plug flow" river reaches and includes the effects of flow, water inflows, PCB diffusion out of (and potentially into) the contaminated sediments, and PCB volatilization.

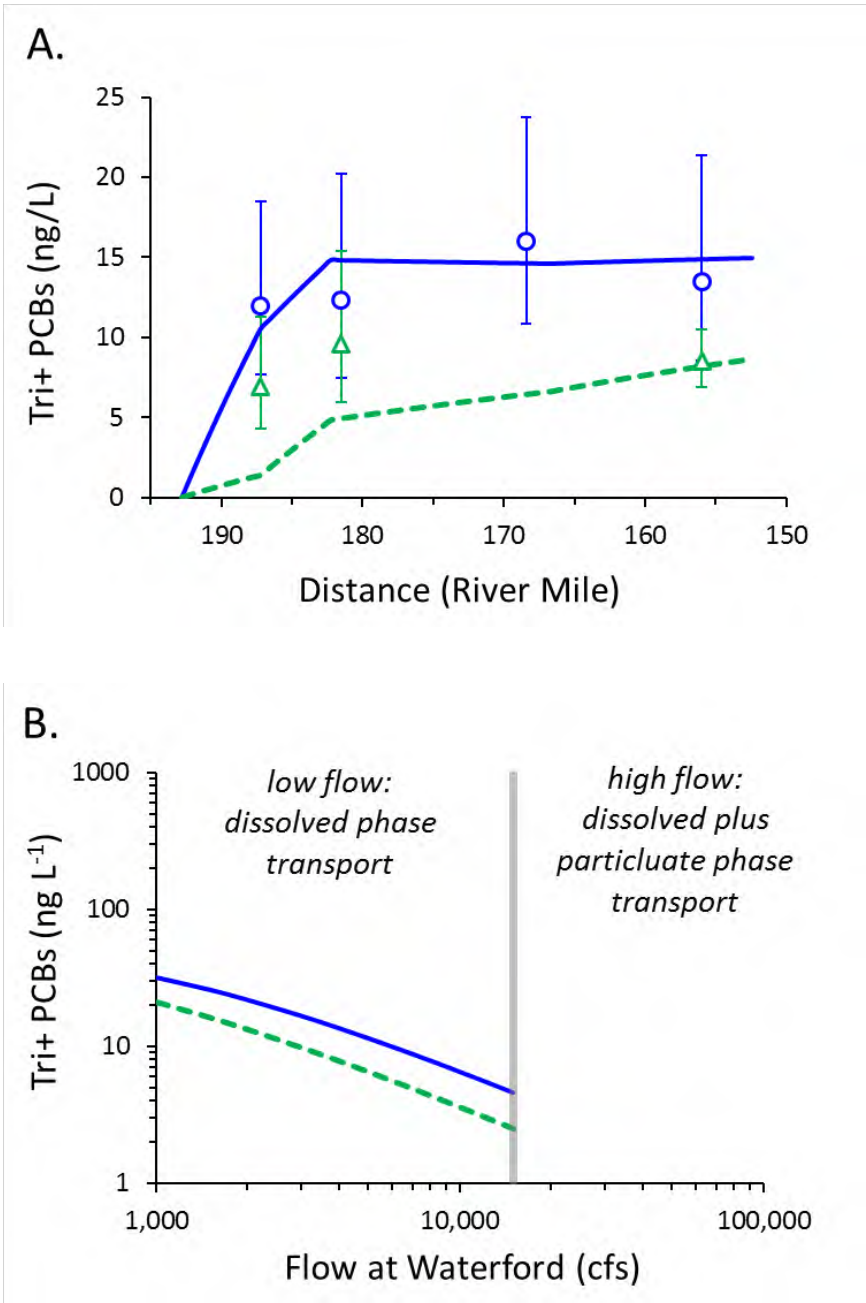

Figure 7. Simplified model results for (A) Tri+ PCB water column concentrations versus River Mile for a summer-time low flow of 3,500 cfs, and (B) Tri+ PCB water column concentrations at Waterford versus flow. Pre-dredging and post-dredging model results are given by the blue solid line and green dashed lines, respectively. For comparison, geometric means (and geometric standard deviations) for Tri+ PCBs for summer-time low flows between 2,500 – 4,500 cfs are included on panel A as blue circles and green triangles for Thompson Island Dam (RM 187.5), Schuylerville (RM 182.3), Stillwater (RM 168.4) and Waterford (RM 156.0).

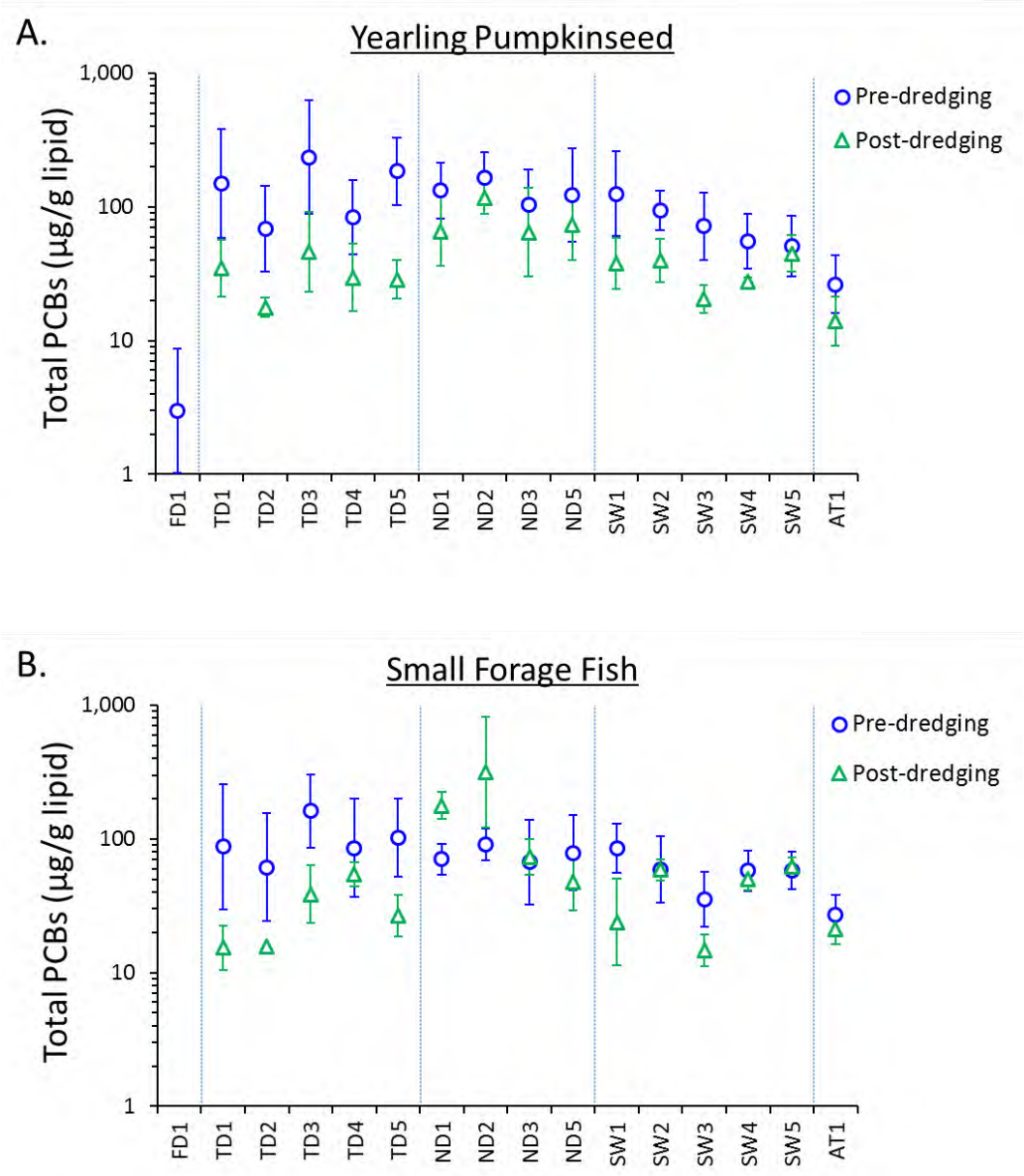

Figure 8. Lipid-normalized PCB concentrations in (A) yearling pumpkinseed and (B) small forage fish for samples pools upstream of the GE plants (FD1), in Thompson Island (TD1-5) in RS#1, Northumberland (ND1-5) in RS#2, Stillwater (SW1-5) in RS#3, and in the Albany Turning Basin (AT1). Observed concentrations are presented as geometric means (and geometric standard deviations) for pre-dredging (2004-2008) and post-dredging (2016) periods. Data provided by Kevin Farrar (NYS DEC).

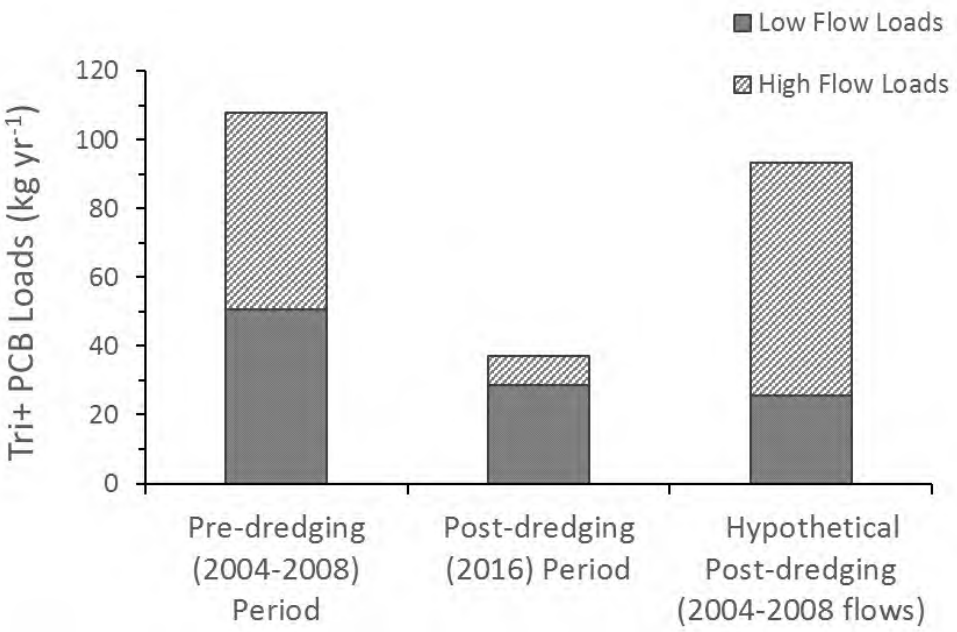

Figure 9. Estimated Tri+ PCB loads to the Lower Hudson for the 2004-2008 pre-dredging period, the 2016 post-dredging period and a hypothetical post-dredging period based on 2004-2008 flow record. Results are presented as stacked bars for low-flow (< 13,000 cfs) and high-flow (> 13,000 cfs) loads.

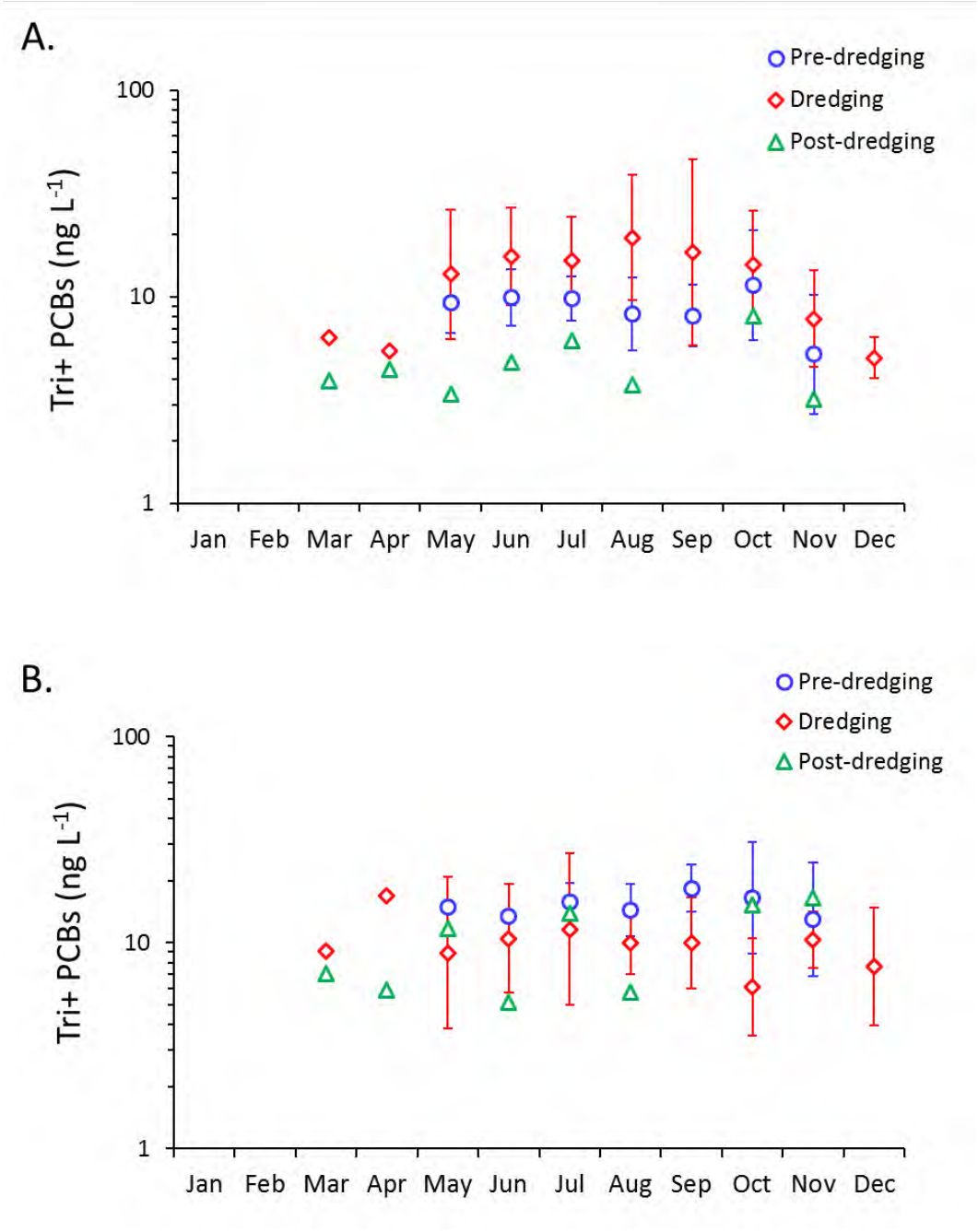

Figure 10. Monthly geometric means (and geometric standard deviations) for Tri+ PCB water column concentrations at (A) Albany (RM 145) and (B) Poughkeepsie (RM 75). Whole water concentrations were determined using either the modified Green Bay Peak (mGBP) or an Aroclor PCB analytical methods. Pre-dredging period is based on 2004-2008 measurements; Dredging period is based on 2009-2015 measurements and includes the Phase 1, Phase 1 evaluation and Phase 2 dredging years; Post-dredging period is based on 2016 measurements.

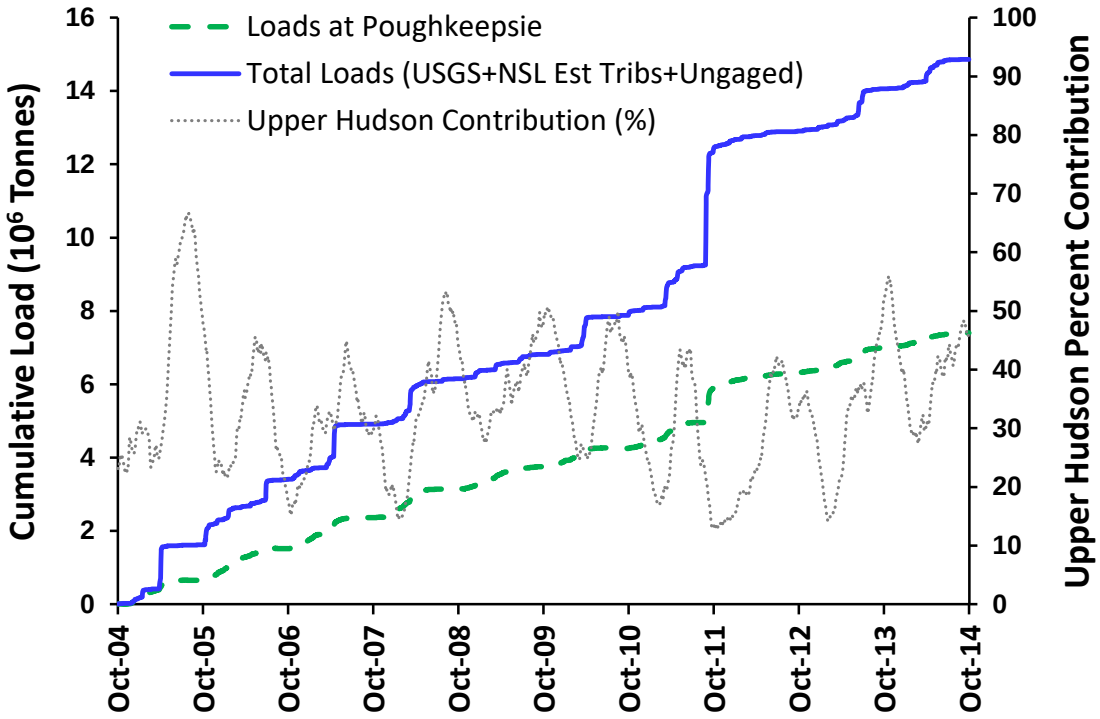

Figure 11. Record of Sediment Loads to the Tidal Freshwater Section of the Hudson River. The solid line represents the cumulative suspended sediment load entering the tidal freshwater Hudson (above Poughkeepsie). The cumulative suspended sediment load was obtained using observed USGS monitoring data, with the modified Normalized Sediment Load (mNSL) function used to fill in missing information for gaged and ungaged portions of the watershed (see da Luz et al. in prep.). The dotted line represents cumulative sediment loads passing Poughkeepsie. Dashed gray line represents the 90-day rolling average of percentage of daily total suspended sediment load attributed to the Upper Hudson. From da Luz et al. (in prep).

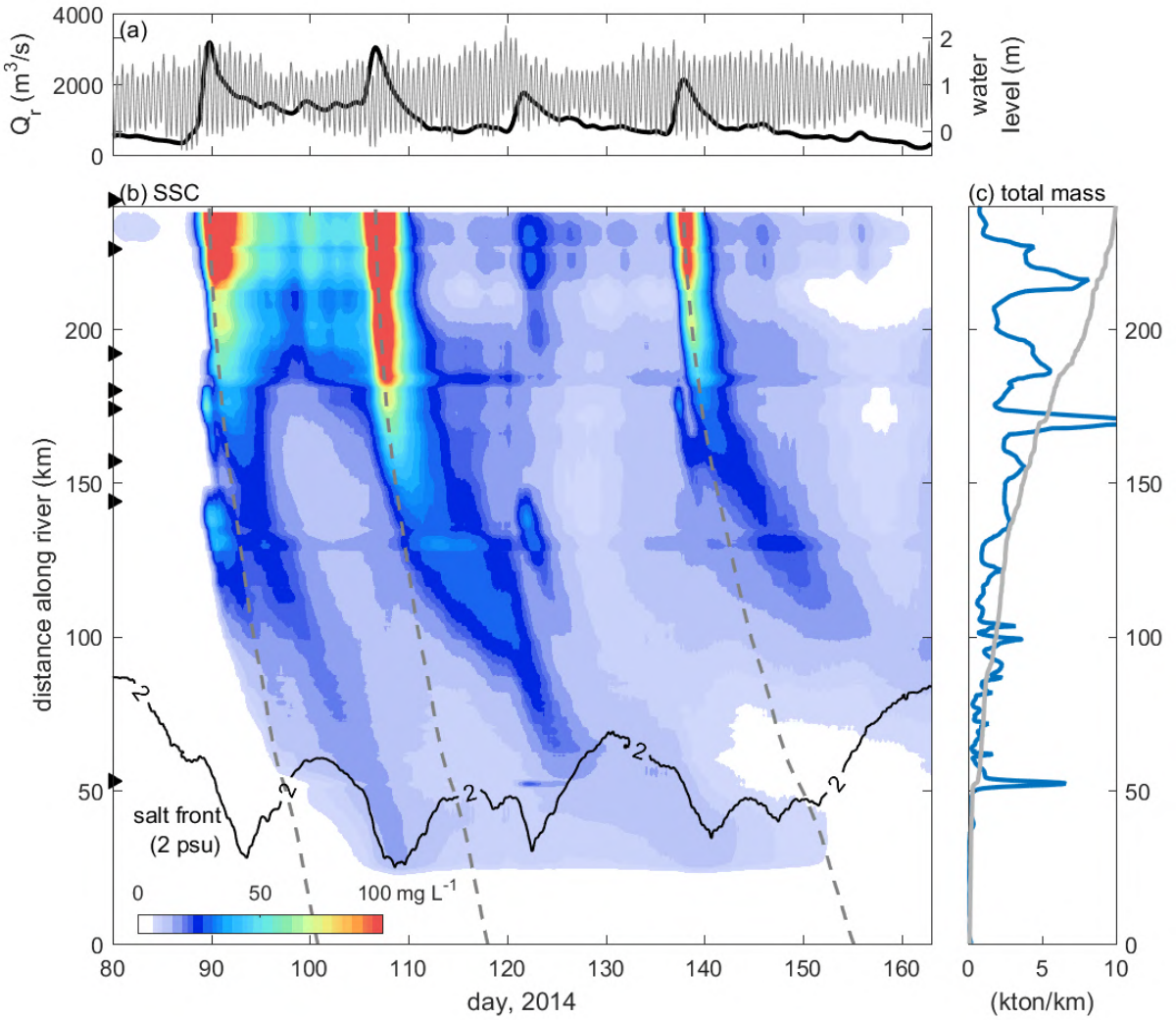

Figure 12. Sediment transport model results for the Lower Hudson from Troy lock and dam (Km 250) to the Battery in New York City (Km 0). (a) River discharge at Green Island with three high flow events during the 80-day simulation period corresponding to day 80 through day 160 in 2014. (b) Cross-sectional averaged suspended sediment concentrations from river inputs as a function of distance along the river and time. Location of tributary inputs are marked with triangles on the y-axis. The rate of water advection associated with the three high flow events is marked by the gray dashed lines. The 2-psu isohaline of bottom salinity is marked in black. (c) Total sediment mass (suspended + bed) at the end of the model period. Gray line is the cumulative mass distribution from the Battery to the Troy lock and dam normalized by the total mass. From Ralston and Geyer (submitted).

An Independent Evaluation of the PCB Dredging Program on the Upper Hudson and Lower Hudson River

Supplemental Information

1. Development of Regression Equations for Tri+ PCB Water Column Concentrations at Waterford as a Function of River Flow
2. Estimation of Tri+ PCB Loads Passing Waterford and Entering the Lower Hudson
3. A Simplified Mass Balance Model to Investigate the Linkage between Sediment Concentrations and Water Column Responses during Summer-time, Low-flow Conditions

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1. Development of Regression Equations for Tri+ PCB Water Column Concentrations at Waterford as a Function of River Flow

Methods

Tri+ PCB concentrations at Waterford were evaluated as a function of river flow using approaches that have previously been applied in examining the effects of flow of suspended sediment load (Ralston and Geyer 2009; da Luz et al. in prep.). In these approaches, suspended sediment loads (in kg/day) are related to river flow using separate log-log regression equations for low flow (non-flood) and high flow (flood) conditions. A similar approach is used here in relating suspended sediment concentration (in mg/L), and subsequently, Tri+ PCB water column concentrations (in ng/L) to river flow.

For suspended sediment, regression equations were developed using paired observations of daily-averaged suspended sediment concentrations and river flow that were collected at Waterford for the 2004-2008 pre-dredging period by the New York U.S. Geologic Survey (NY-USGS) [see Wall et al. (2008) for details]. The paired observations were fit using separate regressions lines for non-flood and flood conditions:

Non-flood conditions: $$\log C = \log a_1 + b_1 \log Q \quad \text{(Eq. S-1)}$$

Flood conditions: $$\log C = \log a_2 + b_2 \log Q \quad \text{(Eq. S-2)}$$

where log a and b represent the intercept and slope of the regression lines. Determination of the delineation for the non-flood and flood conditions (i.e., the break point, BP), log a_1, b_1 and b_2 values was accomplished by minimizing the sum of the squares about the regression lines for the non-flood and flood conditions using Solver in Microsoft Excel. In fitting the regression equations, the intercept of the regression equation for the flood condition (log a_2) was fixed and was set at:

$$\log a_2 = \log a_1 + (b_1 - b_2) \cdot \log BP \quad \text{(Eq. S-3)}$$

to ensure that the regression equation for flood conditions matched the regression equation for non-flood conditions at the break point. Variations of the suspended sediment concentrations about the regression lines were assumed to be normally distributed (in log space) and were quantified by the standard deviation of the residuals of the log suspended sediment concentrations ($S_{\log C}$) across the entire range of flows.

For Tri+ PCB water column concentrations, regression equations were developed using paired observations for Tri+ PCB water column concentrations at Waterford and USGS daily flows. The paired observations were again fit to the regression equations (Eqs. S-1 and S-2). The method followed the approach described above for suspended sediment with one exception. Since Tri+ PCB water column concentrations are expected to be dominated by particulate phase PCBs for the high flow (flood) conditions, the slope of the regression for flood conditions (b_2) was fixed and set equal the b_2 value that was determined for the suspended sediment – flow regression. Because of the large number of observations (particularly for suspended sediment concentrations), suspended sediment concentrations and Tri+ PCB concentrations were binned according to ranges of river flow for graphical presentations.

Results

Regressions for suspended sediment – flow regressions during non-flood and flood conditions are presented graphically in Figure S-1A. The corresponding regression coefficients are given in Table S-1. As shown in Figure S-1A, the suspended sediment concentrations increase slightly as a function of flow up to 10,400 cfs. At higher flows, there is a more substantial increase in suspended sediment concentrations with flow. This finding is consistent with previously reported for the Upper Hudson and for many other rivers, and is attributed to increased erosion of the bottom sediment during periods of high flow (Ralston and Geyer 2009; da Luz et al. in prep.)

Similar results for the Tri+ PCB – flow regressions are presented graphically in Figure S-1B for both 2004-2008 pre-dredging period and the 2016 post-dredging period. See Table S-1 for the corresponding regression coefficients. As shown in Figure S-1B, Tri+ PCB concentrations decrease as a function of flow up to approximately 13,000-15,000 cfs. These results do not align with the suspended sediment – flow regression and suggest that Tri+ PCB water column concentrations are associated with dissolved (and not particulate) phase PCBs. Tri+ PCB water column concentrations also appear to be primarily associated with dissolved phase PCB up to 13,000-15,000 cfs. At higher flows, Tri+ PCB concentrations increase with increasing flows with a slope that is consistent with the suspended sediment – flow regression for flood conditions (b_2 = 2.2). This result indicates that Tri+ PCB water column concentrations are primarily associated with particulate phase PCBs for the higher flows.

A comparison of Tri+ PCB – flow regressions for the 2004-2008 pre-dredging and 2016 post-dredging periods (Figure S-1B) shows that the regression line for post-dredging non-flood period is approximately

a factor of two lower than the corresponding regression line for the pre-dredging period. Differences in the regression lines for the flood conditions however show little or no change for the pre-dredging and post-dredging periods. This latter finding is currently considered tentative because of the limited number of high flow observations that were available for the 2016 post-dredging period.

2. Estimation of Tri+ PCB Loads Passing Waterford and Entering the Lower Hudson

Method

Tri+ PCB loads to the Lower Hudson were calculated for pre-dredging and post-dredging periods using USGS daily flow measurements at Waterford and daily Tri+ PCB concentrations that were calculated as a function of river flow (see previous section). In this approach, daily estimates of log C were determined for non-flood and flood conditions using regression equations (Eqs. S-1 and S-2), previously-determined regression coefficients (Table S-1) and daily flow at Waterford (USGS Gaging Station 01335770). Since the regression equations were developed in log space, the computed log C value corresponds to the median or 50th percentile value of the probability distribution of the daily Tri+ PCB water column concentration. Median concentrations were therefore converted into arithmetic means as:

$$C = 10^{\left(\log C + \frac{2.303}{2} S^2_{\log C}\right)} \qquad \text{(Eq. S4)}$$

where the value of 2.303 corresponds to the natural log of 10. The daily Tri+ PCB load passing Waterford was then calculated by multiply the daily flow at Waterford (Q) times the estimated Tri+ PCB water column concentration (C). Finally, the annual Tri+ PCB load passing Waterford was determined by summing the daily Tri+ PCB loads.

Results

Annual Tri+ PCB loads passing Waterford and entering the Lower Hudson are presented in Table S-2 for 2004-2008 pre-dredging period and the 2016 post-dredging periods. For these calculations, 13,000 cfs was used in differentiating between low flow and high flow conditions. The total Tri+ PCB loads passing Waterford during the 2004-2008 pre-dredging period averaged 107.7 kg/yr, with 47 percent of the total load (50.8 kg/yr) occurring during low flow and the remaining 53 percent (57.0 kg/yr) occurring during high flow conditions. For comparison, the total Tri+ PCB loads passing Waterford during the 2016 post-

dredging period was estimated to be 37.0 kg/yr, with 78 percent of the total load (28.7 kg/yr) occurring during low flow and only 22 percent (8.3 kg/yr) occurring during high flow conditions.

During the 2004-2008 pre-dredging period, river flow at Waterford averaged 10,100 cfs and included an average of 86 days per year with flows in excess of 13,000 cfs. In comparison, the 2016 post-dredging period was characterized by lower flows with an average river flow at Waterford of 6,100 cfs and only 15 days with flows exceeding 13,000 cfs. Because of the importance of flow on the Tri+ PCB load, a hypothetical post-dredging scenario was considered using the 2004-2008 pre-dredging flow record with the post-dredging regression equations (Table S-2). Comparison of the pre-dredging results with the hypothetical post-dredging scenario shows that total Tri+ PCB loads would have been reduced by only 13 percent if river flows for the post-dredging period were comparable to flows during the pre-dredging period. Tri+ PCB loads for low flow conditions were approximately 27 kg/yr for both the 2016 post-dredging period and hypothetical post-dredging scenario. This indicates that year-to-year variations in river flow will have a small effect on Tri+ PCB loads during low flows. However, Tri+ PCB loads during high flows showed large differences. This result indicates that Tri+ PCB loads during high flow conditions will likely show large year-to-year variations; e.g., from 8.3 kg/yr based on the 2016 flow record to potentially more than 100 kg/yr if the river experiences another year like 2011 with three major high flow events.

3. A Simplified Mass Balance Model to Investigate the Linkage between Sediment Concentrations and Water Column Responses during Summer-time, Low-flow Conditions

Model Description

A simplified model was developed as part of a preliminary investigation of the linkage between PCB surface sediment concentrations and water column responses during summer-time low flow conditions. In the model, Tri+ PCBs in the water column are assumed to be primarily comprised of dissolved phase contaminant during low flow periods. A schematic of the simplified model is presented in Figure S-2. As shown, the model represents the Upper Hudson as four consecutive “plug-flow” river reaches. The river reaches correspond to the three River Sections, with the longest River Section (RS#3) divided into RS#3a and RS#3b at Stillwater where the Hoosic River enters the Upper Hudson. Processes considered in the model calculation include flow through the river reaches, PCB diffusion out of (and potentially into) the contaminated sediments, PCB volatilization, and addition of flow from tributaries and surface runoff.

The plug-flow, mass balance equation that was applied to each river reach is given as:

$$\frac{dC}{dt^*} = \frac{k_f^{'}}{h}\left[C_{pw} - C\right] - \frac{k_v^{'}}{h} C \qquad \text{(Eq. S-5)}$$

where the term on the left-hand side represents the change in the Tri+ PCB water column concentration as the river water flows downstream, the first term on the right-hand side represents the net gain of Tri+ PCB from diffusion out of (and potentially into) the contaminated sediments, and the last term on the right-hand side represents the loss of Tri+ PCB by volatilization as the water flows downstream. Specific notation is given as: C = Tri+ PCB water column concentration (in ng/L); t* = travel time (in days) which is equal to the distance downriver (x) divided by the river velocity (U); k_f' = diffusive exchange coefficient between the pore-water and the overlying water (in m/day); h = average depth of the river (m); C_{pw} = Tri+ PCB concentration in the underlying pore-water (in ng/L); and k_v' = the volatilization rate coefficient in m/day). For our calculations, the Tri+ PCB pore-water concentrations for each river reach (C_{pw}) were calculated based on equilibrium partitioning:

$$C_{pw} = \frac{r_a}{K_D} \qquad \text{(Eq. S-6)}$$

where r_a is the Tri+ PCB concentration in the surface sediments (in mg/kg); and K_D is the equilibrium partition coefficient (in L/kg).

The analytical solution for the plug-flow, mass balance equation (Eq. S-5) is given as:

$$C = \frac{k_f^{'} C_{pw}}{\left(k_f^{'} + k_v^{'}\right)} \cdot \left[1 - e^{-\left(\frac{k_f^{'} + k_v^{'}}{h}\right) \cdot t^*}\right] + C_o \cdot e^{-\left(\frac{k_f^{'} + k_v^{'}}{h}\right) \cdot t^*} \qquad \text{(Eq. S-7)}$$

where C_o represents the Tri+ PCB concentration at the beginning of the river reach. For the first river reach, C_o is set equal to the Tri+ PCB concentration Bakers Falls (RM 194.5). For subsequent river reaches, Co is determined from a mass balance calculation at the beginning of the reach:

$$Q_u \cdot C_u + Q_t \cdot C_t = Q \cdot C_o \qquad \text{(Eq. S-8)}$$

where Q_u and C_u represent the river flow and Tri+ PCB water column concentration at the end of the previous reach; Q_t and C_t represent the river flow and Tri+ PCB water column concentration associated with tributary inflows; and Q represents the river flow for the river reach.

Methods

Model parameters and coefficients for the four river reaches are given in Table S-3 for a summer-time, low-flow of 3,500 cfs (99.1 m^3/sec) at Waterford. Channel geometry (length, width, depth) and drainage area were obtained from information in EPA (2000a). Flow (Q) was considered to be constant through each river reach and was scaled according to the total drainage area. For simplicity, tributary and surface runoff flows were assumed to enter the river at the beginning of each reach. This assumption is expected to have a minor effect on the model calculations because the major tributaries to the Upper Hudson, Batten Kill and the Hoosic River, enter at the beginning of river reaches RS#3a and RS#3b. Based on the river geometry and flow rate, the average velocity in each river reach was calculated by dividing the flow (Q) by the average depth (h) and average width (b), and the travel time (t*) through each river reach was calculated by dividing the length of the river reach by the average velocity (U).

For pre-dredging model calculations, average Tri+ PCB concentrations in surface (0-2 inch) sediments (r_a) for each river reach were based on the Sediment Sampling and Analysis Program (SSAP) that was conducted in 2002-2005 during the remedial design phase. See EPA (2012) for details. Tri+ PCB pore-water concentrations were calculated from the Tri+ PCB surface sediment concentrations using the equilibrium partitioning relationship in Eq. S-6. For these calculations, the K_D value was estimated from $K_D = f_{oc} \times K_{oc}$ where f_{oc} (the fraction organic carbon in surface sediments) was taken as 0.03 and K_{oc} (the organic carbon – water partition coefficient) was were taken 10^6 L/kg. The volatilization rate coefficient (k_v') was calculated based on two-film theory. For this calculation, the transfer through liquid-side of the interface was assumed to control volatilization, and the transfer rate coefficient for the liquid-side of the interface was estimated using the O'Connor-Dobbins formula. Based on these assumptions, k_v' was computed as:

$$k_v' = \sqrt{D_w \frac{U}{h}} \qquad \text{(Eq. S9)}$$

where D_w is the molecular diffusivity of PCBs in water and was taken as 5×10^{-6} cm^2 sec^{-1}. The final model coefficient, the diffusive exchange coefficient between pore-water and the overlying water (k_f'), was adjusted to match the calculated Tri+ PCB water column concentrations to Tri+ PCB water column concentrations during summer-time low flows (corresponding to 3,000 – 4,000 cfs at Waterford).

Comparable model calculations were performed for post-dredging conditions by modified only the Tri+ PCB concentrations in the surface sediment. All other model parameters and coefficients (including the calcibrated k_f' value) remained unchanged. Since post-dredging monitoring data are not yet available for the sediment, post-dredging concentrations of Tri+ PCBs in surface sediments were assigned based on estimates given in EPA (2012).

Finally, model calculations were performed over a wider range of summer-time low flows at Waterford (i.e., 1,000 to 13,000 cfs). Particulate-phase PCBs were expected to control Tri+ PCB water column concentrations for flows greater than 13,000 cfs at the Waterford monitoring station. For the higher flows, the simplified model (which is based on dissolved-phase transport of Tri+ PCBs) would no longer apply.

Pre-dredging Model Calibration

The simplified model was calibrated to the observed Tri+ PCB water column concentrations for the pre-dredging period by adjusting the PCB diffusive exchange coefficient between the water column and the underlying pore-water (k_f') to 0.05 cm/day. For the model calibration, a flow at Waterford of 3,500 cfs was considered to be representative of summer-time low-flow at Waterford. Comparison of the calibrated model (blue solid line) with observed Tri+ PCB water column concentrations at TID, Schuylerville, Stillwater and Waterford (blue open circles) is shown in Figure S-3A. The observed concentrations are represented as geometric means (with geometric standard deviations) based on 2004-2008 summer-time Tri+ PCB measurements that were taken on days with river flows of 3,000 – 4,000 cfs at the USGS Waterford monitoring station. The calibrated k_f' value of 0.05 cm/day was found to be comparable to values previously reported in modeling studies of the Upper Hudson (Connolly et al. 2000; Erickson et al. 2005).

Post-dredging Model Projection

The simplified model was used as part of a preliminary investigation investigating the potential effects of dredging on Tri+ PCB water column concentrations during a summer-time low-flow of 3,500 cfs at Waterford. As shown by the green dashed line in Figure S-3A, post-dredging model results show a large decrease in Tri+ PCB water column concentrations (compared to the pre-dredging modeling results). Observed Tri+ PCB water column concentrations for the 2016 post-dredging period are shown by the green triangles in Figure S-3A. The observed concentrations are again as geometric means (with

geometric standard deviations) summer-time Tri+ PCB measurements that were taken on days with river flows of 3,000 – 4,000 cfs at the USGS Waterford monitoring station. As shown, the model projection is aligned to the observed Tri+ PCB concentrations at Waterford (RM156.0). Model projections however are lower than the observed Tri+ PCB water column concentration at TID (RM 187.5) and Schuylerville (RM 182.3).

Discrepancies between the simplified model results and the 2016 post-dredging data at TID (RM 187.5) and Schuylerville (RM 182.3) suggest that the 2016 post-dredging Tri+ PCB concentrations in surface sediments are higher than the EPA (2012) estimated concentrations that were used in the model calculations. However, another plausible explanation is that sediments in the dredging zones need time to "stabilize" after six years of dredging. For example, the higher Tri+ PCB water column concentrations at TID and Schuylerville may be due to residual effects of dredging disturbances that are continuing to cause supply localized resuspension of sediments even during summer-time low flow conditions. This would result in higher Tri+ PCB water column concentrations due to the presence of particulate-phase PCBs that were not considered in the simplified model calculations. It could therefore be argued that one year of post-dredging monitoring data may is not sufficient to evaluate the full benefits of the dredging program.

Effect of River Flow on Tri+ PCB Water Column Response

The simplified model was used to examine the effects of river flow on Tri+ PCB water column concentrations for summer-time low-flow conditions (i.e., < 13,000 cfs). As presented in Figure S-3B, model results for both pre-dredging and post-dredging periods show decreases in Tri+ PCB water column concentrations at Waterford with increasing flows. In addition, the post-dredging modeling results are approximately a factor of two lower than the pre-dredging results. These findings are consistent with the non-flood regressions previously presented in Figure S-1 and suggest that Tri+ PCB water column concentrations are controlled by Tri+ PCB diffusion from the underlying sediments during low-flow conditions.

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Table S-1. Regression Coefficients for Suspended Sediment and Tri+ PCB Water Column Concentrations[a]

	Suspended Sediment Concentrations (mg/L)	Tri+ PCB Water Column Concentrations (ng/L)	
	2004-2008 Pre-dredging Period	2004-2008 Pre-dredging Period	2016 Post-dredging Period
log a_1	-1.66	2.44	2.42
b_1	0.59	-0.38	-0.43
BP	10,050	15,450	12,400
log a_2[b]	-8.14	-8.38	-8.35
b_2	2.2	2.2[c]	2.2[c]
$S_{\log C}$	0.30	0.24	0.18

[a] Regression coefficients (log a_1, b1, BP, b_2) were determined for the log-log relationships for non-flood and flood conditions (Eqs. S-1 and S-2).

[b] log a_2 values were calculated using Eq. S-3 to ensure that the regression equation for flood conditions matches the regression equation for non-flood conditions.

[c] For the Tri+ PCB regressions, the slope for flood conditions (b_2) was fixed and set equal to the b_2 value that was determined for the suspended sediment – flow regression.

Table S-2. Estimated Annual Tri+ PCB Loads Passing Waterford and Entering the Lower Hudson.

Calendar Year	Average Annual Flow (cfs)	Number of days with high flows[a]	Annual Tri+ PCB Loads (kg yr^{-1})		
			Non-flood	Flood	Total
Pre-dredging Period[b]					
2004	8,822	50	56.3	18.8	75.2
2005	10,055	83	50.1	63.1	113.1
2006	11,801	112	56.2	61.7	117.9
2007	8,743	71	45.9	49.1	95.1
2008	11,185	114	45.4	92.0	137.4
Average	10,121	86.0	50.8	57.0	107.7
Post-dredging Period[c]					
2016	6,105	15	28.7	8.3	37.0
Hypothetical Post-dredging Period[d]					
2004	8,822	50	30.8	25.0	55.8
2005	10,055	83	26.0	73.3	99.3
2006	11,801	112	24.5	77.7	102.3
2007	8,743	71	24.1	57.9	82.0
2008	11,185	114	23.1	104.1	127.2
Average	10,121	86.0	25.7	67.6	93.3

[a] Number of days with flows exceeding 13,000 cfs at Waterford.

[b] Tri+ PCB loads for the pre-dredging period are based on regression coefficients for the pre-dredging period (see Table S-1) and the 2004-2008 flows at the USGS Waterford monitoring station.

[c] Tri+ PCB loads for the post-dredging period are based on regression coefficients for the post-dredging period (see Table S-1) and the 2016 flows at the USGS Waterford monitoring station.

[d] Tri+ PCB loads for the hypothetical post-dredging period are based on regression coefficients for the post-dredging period (see Table S-1) and the 2004-2008 flows at the USGS Waterford monitoring station.

Table S-3. Model Parameters and Coefficients for Summer-time, Low-flow Model Calculations[a]

		RS#1	RS#2	RS#3a	RS#3b
Length	km	9.03	8.19	24.49	23.01
Drainage Area	mi^2	2,971	2,971	4,455	4,573
Average Width	m	304.9	226.6	207.0	299.6
Average Depth (h)	m	2.36	2.90	3.79	3.80
Flow (Q)[b]	m^3/sec	64.4	64.4	96.5	99.1
Average Velocity (U)[c]	m/sec	0.09	0.10	0.12	0.09
Travel Time (t*)	days	1.17	0.97	2.30	3.06
Diffusive Exch. Coef. (k_f')	m/day	0.05	0.05	0.05	0.05
Volatilization Rate Coef. (k_v')	m/day	0.38	0.36	0.35	0.29
Partition Coef. (K_D)	L/kg	3×10^4	3×10^4	3×10^4	3×10^4
Pre-dredging Tri+ PCB Concentrations in Surface Sediments					
Tri+ PCBs Surf. Sediment (r_a)	mg/kg	14.2	11.0	3.3	3.3
Tri+ PCBs Pore-water (C_{pw})	ng/L	473	367	110	110
Post-dredging Tri+ PCB Concentrations in Surface Sediments					
Tri+ PCBs Surf. Sediment (r_a)	mg/kg	1.9	7.1	3.1	3.1
Tri+ PCBs Pore-water (C_{pw})	ng/L	63	237	103	103

[a] See text for explanations of how model parameters and coefficients were obtained for the four river sections.

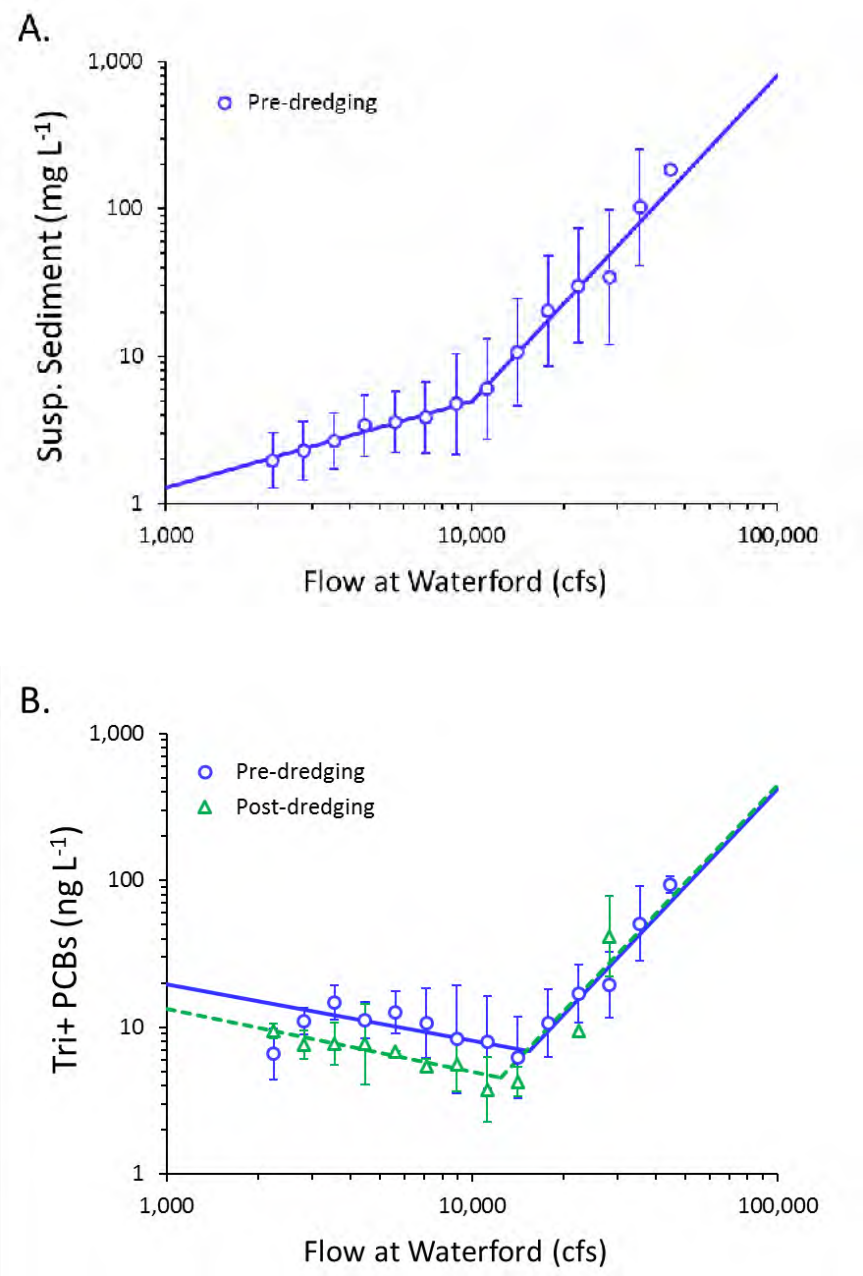

Figure S-1. Suspended sediment and Tri+ PCB water column responses at Waterford. (A) Suspended sediment concentrations versus river flow, and (B) Tri+ PCB concentrations versus river flow. Observed 2004-2008 pre-dredging (open blue circles) and 2016 post-dredging (open green triangles) concentrations are represented as geometric means (with geometric standard deviations) for selected flow bins. The corresponding regression equations are given by the solid blue lines and the dashed green lines for non-flood and flood flow conditions.

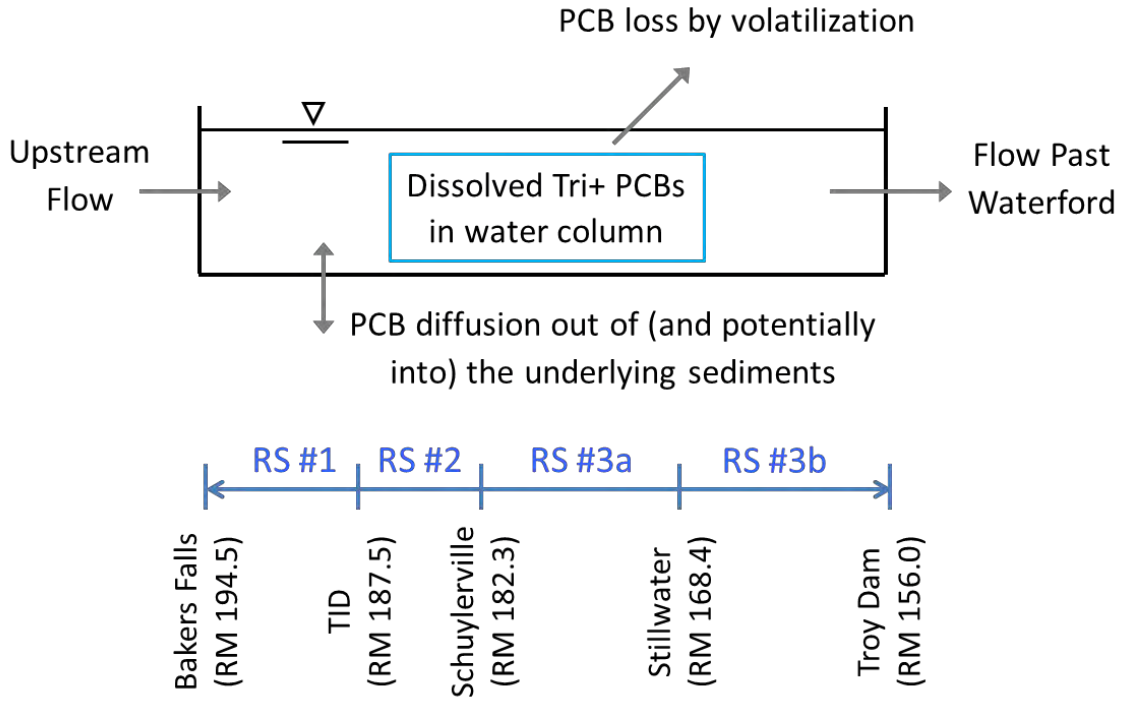

Figure S-2. Simplified model for Tri+ PCB transport through the Upper Hudson during summer-time conditions. The model represents the Upper Hudson as four consecutive "plug flow" river reaches and includes the effects of flow, water inflows, PCB diffusion out of (and potentially into) the contaminated sediments, and PCB volatilization.

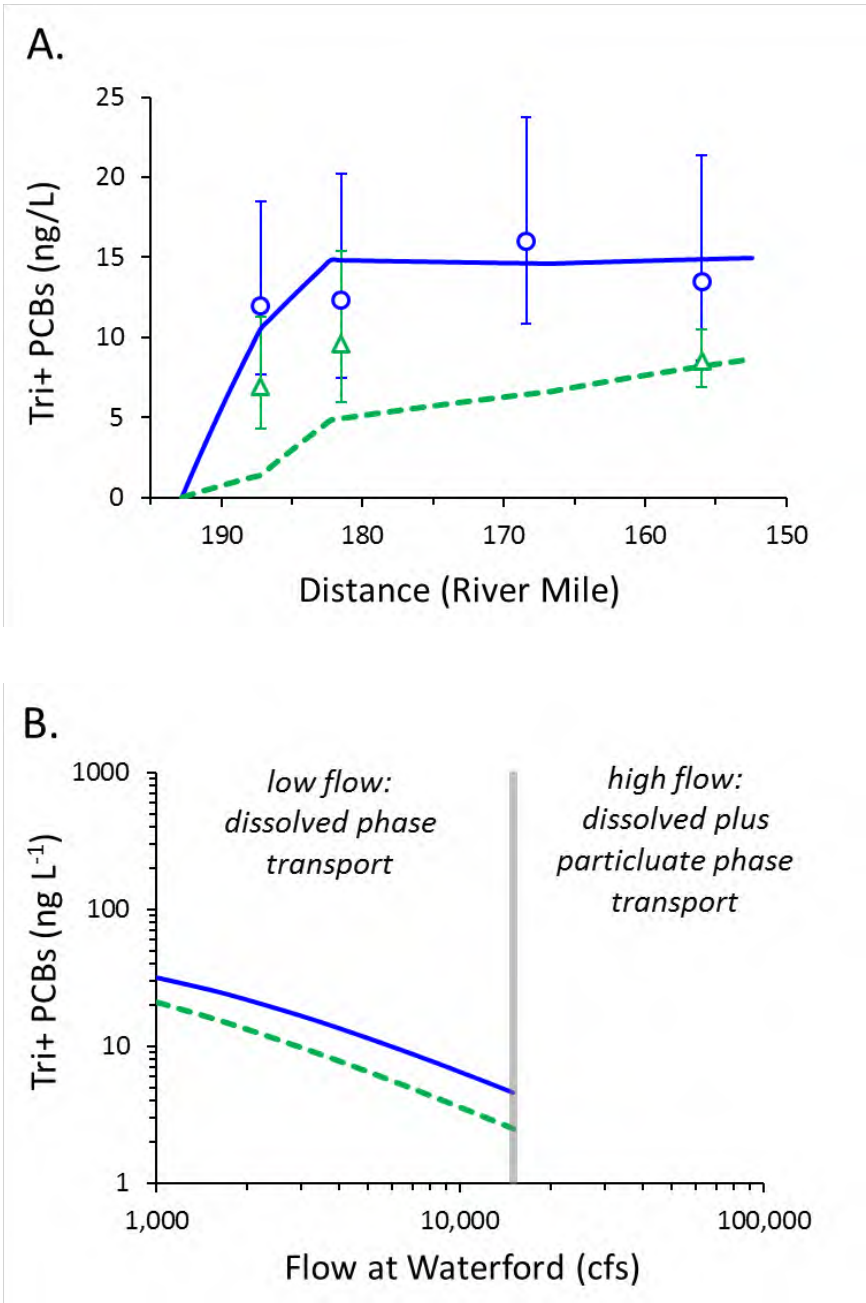

Figure S-3. Simplified model results for (A) Tri+ PCB water column concentrations versus River Mile for a summer-time low flow of 3,500 cfs, and (B) Tri+ PCB water column concentrations at Waterford versus flow. Pre-dredging and post-dredging model results are given by the blue solid line and green dashed lines, respectively. For comparison, geometric means (and geometric standard deviations) for Tri+ PCBs for summer-time low flows between 2,500 – 4,500 cfs are included on panel A as blue circles and green triangles for Thompson Island Dam (RM 187.5), Schuylerville (RM 182.3), Stillwater (RM 168.4) and Waterford (RM 156.0).

Attachment O

S.S. Papadopulos & Associates, Inc., Hudson River PCBs Site Proposed Second Five Year Review – Technical Review August 2017

Hudson River PCBs Site Proposed Second Five Year Review – Technical Review

Prepared for:

Scenic Hudson, Inc.
Riverkeeper, Inc.

Prepared by:

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August 2017

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REPORT

Summary Points:

- Data interpretation for fish tissue in EPA's proposed 2017 FYR ignores the very large degree of uncertainty introduced by the data transformation procedure applied to calculate fish recovery rates. Data interpretation to derive fish recovery rates must consider the very large uncertainty introduced by the data transformation procedure as well as the uncertainty inherent to fish tissue results to establish a degree of confidence in the calculated recovery rates. There is a strong likelihood that any recovery rate calculated based on the available data for fish tissue is so uncertain as to be meaningless for predicting fish recovery in the Hudson river.
- Transforming PCB concentration data from different laboratories, different analytical methods and different field studies into a PCB homologue equivalent database introduces a very large degree of uncertainty on the transformed data. EPA did not test the effect of that uncertainty on the confidence that can be attached to its fish tissue recovery rates.
- EPA's conclusion that the recovery rate for fish is on track to meet the goals of the ROD is not supported by the data with any reasonable degree of confidence or scientific certainty. EPA's procedure to calculate a recovery rate for fish in the Hudson river is too uncertain and is unreliable to support EPA's conclusion that the goals of the ROD will be achieved as previously predicted in the ROD.
- EPA's procedure to calculate recovery rates is at the upper end of the range of rates that the data could potentially support (were the transformed data not impaired by unaccounted for uncertainty).
- EPA's approach for calculating fish tissue trends included rib-out sample sets taken by GE in 2007 and 2008. Compared to the rib-in data (NYSDEC standard fillet samples), the rib-out measurements are consistently lower. Lower concentrations for these samples in 2007-2008 influence the trends calculated for the period 1995-2008 toward faster recovery rate predictions.

- Overall, fish tissue recovery rates are highly variable. The use of an arithmetic or weighted average rate is unrepresentative of this variation and deceptive when making conclusions about the protectiveness of the remedy.
- Using Aroclor-based data without transforming the data to homologue equivalents avoid the uncertainty inherent to the data transformation procedure. Calculating trends using the Aroclor data yields an average recovery rate that is different and substantially lower compared to the rate calculated using the homologue equivalent data. This demonstrates that the uncertainty introduced by the data transformation procedure is significant.
- The slowest fish tissue recovery rates hold more weight when considering the remedy effectiveness, since these species will continue to be a pathway to human exposure past the timeframe asserted by EPA. The use of an average recovery rate applied to all fish species conceals the variability in individual recovery rates by species.
- Using slightly different approaches to data interpretation results in consistently lower average recovery rates than the EPA reported 8% per year decline in fish tissue. These differently calculated average rates correspond to post dredging recovery times of about 20 years to reach 0.4 mg/kg in wet weight Tri+ PCB concentration, and 30-40 years to reach 0.2 mg/kg. This compares with the ROD predictions that the 0.4 mg/kg goal would be reached 5 years post dredging and the 0.2 mg/kg goal 16 years post dredging.

1. Introduction

This report concerns the United States Environmental Protection Agency Hudson River PCBs Superfund Site. In June 2017, EPA issued its Proposed Second Five Year Review of the Site regarding Operable Units (OUs) 1 and 2. In this Review, EPA included analyses of certain selected water column, sediment, and fish tissue PCB measurement datasets. Based on these analyses and comparison with output from models used in the Risk Assessment and Feasibility Study, EPA concluded that the selected remedy for the site, REM-3/10/Select, will be protective, and that no further remedial action is required.

The Site is formally defined as the 200-mile stretch of the Hudson River located in New York State from the Village of Hudson Falls to Battery Park in New York City. The Upper Hudson consists of the upper 40-mile stretch in between Hudson Falls and the Federal Dam at Troy, while the Lower Hudson consists of the remainder of the river. The Upper Hudson is subdivided into River Sections (RSs) 1, 2, and 3.

The original sources of PCB contamination were two General Electric facilities, located at Fort Edward and Hudson Falls. Between 1947 and 1977, these plants discharged an unknown quantity of PCBs into the river. Beginning in 1976, the New York State Department of Environmental Conservation closed fisheries and issued fish consumption advisories due to high levels of PCBs found in Hudson River fish.

River Section 1 includes Remnant Deposits that were capped following the 1984 Record of Decision for OU1. The ROD also included an interim no-action decision regarding the contaminated sediments of the Upper Hudson. Between 1989 and 2000, a multi-phase Reassessment RI/FS was conducted to reevaluate the decision concerning the sediments. This study included the development of several models to predict the transport, fate, and bioaccumulation of PCBs in sediment, water column, and fish. These models were used to forecast the recovery times for several remedial alternative plans, with active alternatives each defined by different RS-specific dredging criteria. These criteria were based upon the metric of Tri+ PCB mass per unit area (MPA) in grams per square meter, which is calculated by multiplying PCB concentration by core length and

solid specific weight (sediment density). MPA was used because it provides a more useful and appropriate measure for spatial delineation purposes than concentration, which is highly variable with sediment depth. Tri+ PCBs are defined by the EPA as the sum of all congeners in a sample containing 3 or more chlorine atoms. Tri+ PCBs are considered to pose more of a risk to human health and the environment, and are considered to more readily bioaccumulate than monochlorobiphenyls or dichlorobiphenyls. For this reason Tri+ PCBs and TPCBs were assumed to be approximately equal or interchangeable in the interpretation of fish tissue data conducted by EPA. Based on congener data obtained by EPA in the 1990s, Tri+ PCBs were found to represent 90% or more of total PCB burden in fish samples.

As part of the RI/FS, a system was developed to calculate a metric for fish tissue concentrations weighted by species and River Section length to represent a typical angler's fish diet. This weighted average metric is based upon three fish species (with weights of 0.47 for largemouth bass, 0.44 for brown bullhead, and 0.09 for yellow perch). River Sections are weighted by their proportional length in miles (0.154 for RS1, 0.125 for RS2, and 0.721 for RS3). This set of weighting factors puts emphasis on largemouth bass and brown bullhead in River Section 3, causing these fish to dominate the calculation of "average" recovery rates in the proposed FYR.

The Reassessment RI/FS was the basis for EPA's 2002 decision that the sediments posed an unacceptable risk to human health and the environment. The 2002 ROD set RS-specific criteria to be used to dredge contaminated sediments in the Upper Hudson, as well as ultimate and interim goals for fish tissue PCB concentrations. The ultimate goal of 0.05 mg/kg Tri+PCBs in wet-weight fish fillet was not attained within the 70 years period modeled in the RI/FS and was not a basis for selecting the remedy. The predicted interim goals included 0.4 mg/kg within 5 years after the completion of dredging and 0.2 mg/kg within 16 years. The Lower Hudson was not targeted for dredging, and no specific goals were set for fish tissue concentrations in that portion of the river. However, the ROD assumed that fish tissue concentrations in the Lower Hudson would decline following remediation of the Upper Hudson, and the active remedy in the Upper Hudson was intended to be protective of both the Upper and Lower

River. The Lower Hudson was predicted to recover much more quickly than the Upper Hudson since PCB concentrations in fish were lower than those in the Upper Hudson.

The 2002 ROD and 2004 Final Decision defined the dredging target areas as any areas having the following contamination levels or greater. EPA defined surface sediment as the top 12 inches of river bottom sediment.

- RS1: An MPA of 3 g/m^2 and a surface concentration of 10 mg/kg Tri+ PCBs
- RS2: An MPA of 10 g/m^2 and a surface concentration of 30 mg/kg Tri+ PCBs
- RS3: An MPA of 10 g/m^2 and a surface concentration of 30 mg/kg Tri+ PCBs (Hot Spots 36, 37, and the southern portion of 39)

The values of 3 and 10 g/m^2 were determined to be "breakpoints where a small change in MPA would mean a large increase in sediment area or mass to be remediated" (USEPA 2002, p. 64). In this way, the 3 and 10 MPA values were intended to maximize the efficiency of the remediation criteria.

Dredging was planned in two phases. These phases occurred later than the original ROD plan, which had called for dredging between 2005 and 2010.

- Phase 1, RS1: Delineated in 2005, dredged in 2009
- Phase 2, RS2 and RS3 (with a return to RS1): Delineated in 2007, dredged in 2011-2015

To delineate the areas to be dredged for both phases, sediment data were collected as part of the Sediment Sampling and Analysis Program (SSAP) between 2002 and 2005. The data were used in a kriging interpolation procedure to delineate dredge areas both horizontally and vertically. The design of this sampling program for RS2 and RS3 was intended to identify contaminated areas, not to characterize the distribution of PCBs across entire areas of the river in an unbiased manner. This has important consequences when comparing the SSAP data to 2016 OM&M post-dredging data, as discussed further below. However, EPA used these data as the primary source of pre-dredging data to assess sediment recovery, while acknowledging the bias present in the RS2 and RS3 sample design (USEPA 2017, Appendix 4). During the SSAP period, it was found that the RI/FS methods had consistently underestimated the depth of contamination in certain

areas. This led a larger volume of sediment over a smaller spatial area to be included in the dredging delineation than had been estimated in the ROD.

In total, an estimated 2.75 million cubic yards of sediment and 155,760 kg total PCBs were removed from the Upper Hudson during dredging implementation, compared to the original estimates of 2.65 million cubic yards containing 70,000 kg of total PCBs.

2. About the Data: Transformation from Aroclor to Homologue Equivalent Estimates

- Prior to the analysis of fish tissue concentration trends, EPA developed regression equations to convert Aroclor based concentration data into "homologue equivalent" data.
- A considerable amount of these data were transformed using an equation from paired samples that is a decade older than the transformed data (extrapolation).
- The process of transforming PCB concentration data introduced considerable uncertainty and systematic bias into the overall analyses, and that uncertainty is unaccounted for in EPA's estimates of fish tissue recovery rates.

Several analytical methods and laboratories were used over a period of more than two decades to generate the PCB concentration data for the Hudson river sediment. The analytical methods have included M8082, for Aroclor measurement; M1668, mGBM and NYSDEC M91-11 for specific congener measurements; and M680, for homologue measurement. M8082 was the method used to analyze the bulk of the data used by EPA in the 2017 Proposed FYR. This method is known to result in inaccuracy, due to its neglect of overlap in congener content among Aroclor mixtures ("double counting"). Different labs have different ways of reducing double-counting, leading to increased uncertainty. In addition, a proportion of the constituents of Aroclor mixtures will change over time in response to environmental exposure. Compositional changes for PCBs in sediment and fish tissue occur by dechlorination, which leaves behind lighter congeners than were originally present, and/or volatilization and dissolution, which leaves behind a mixture enriched with heavier congeners. M8082 assumes that the Aroclor mixtures in environmental samples remain as for the original PCB product, leading to inaccuracy in analytical results and adding uncertainty to data interpretation.

EPA used a regression procedure to convert all fish tissue $TPCB_{Aroclor}$ results into TPCB "Homologue Equivalent," or $TPCB_{HE}$, values (this is illustrated in Table A5-20 in the 2017 FYR). It was these transformed data that were plotted in the 2017 Proposed FYR fish tissue trend analyses to support a weighted average 8% recovery rate for fish tissue Tri+ PCB concentrations. For each subset of data (by time period and laboratory)

between 1990 and 2013, EPA utilized the existence of paired samples that had been measured with both M8082 and a homologue or congener method. A separate regression was performed on each of these matched pair sets, and the geometric mean was used as the estimated proportionality factor to transform the broader subset of data from $TPCB_{Aroclor}$ to $TPCB_{HE}$. Regression equations that had been calculated for use in the ROD for data prior to 1998 were re-used without modification for the 2017 Proposed FYR. Uncertainty in the geometric mean was estimated using a bootstrap analysis, and the use of an adjustment factor was found to be statistically justified via a Wilcoxon signed rank test. However, the uncertainty in the geometric mean was not carried over to the next step in the analyses, namely the calculation of fish tissue trends. Rather, the analyses of fish tissue trends assumed that $TPCB_{HE}$ transformed values were measured data, and uncertainty in the percent rate of decline was calculated by measuring the standard error of the coefficient. The data transformation procedure carries a very large degree of uncertainty, and this uncertainty is unaccounted for in EPA's estimates of recovery rates.

Uncertainty due to extrapolation is also an issue for the 2017 interpretation, particularly regarding data collected by NYSDEC and analyzed by Mississippi State Chemical Laboratories. For this data subset, paired samples were only available from 1999-2000; the regression factor resulting from these paired samples was extrapolated onto data collected a decade into the future, from 2001-2011 (EPA 2017, Table A5-20). The extrapolated data included in the 2017 fish tissue analyses (n=3412) represent about 36% of all data included in the 2017 fish tissue analyses (n=9387). This percentage is higher for those species and River Section combinations with data limited to a timeframe within 2000-2011.

3. 2017 Proposed Five Year Review Fish Tissue Trend Analysis

- EPA used specific criteria for data to be included in the fish tissue trend analyses. This included the use of inconsistent rib-out data collected by GE in 2007 and 2008.
- EPA chose a lipid normalized approach as the most conservative method for examining fish tissue trends.
- Based on an analysis that excluded about 50% of the total data, EPA asserted transforming the data from Aroclor based to homologue equivalent measurements had virtually no effect on fish tissue trends.

The goal of the 2017 analysis was to estimate a recovery rate for Tri+ PCB concentrations in fish tissue over time. EPA considered data from periods of disturbance to Monitored Natural Attenuation unusable for the assessment. For this reason, EPA excluded data prior to 1995 (to avoid the effects of the Allen Mill event and to avoid uncertainty due to noncomparable historical analytical methods), as well as data collected after 2008 (to exclude the effects of dredging, which began in 2009). EPA did not include 2016 data because concentrations had not had enough time to reach equilibrium following disturbance and resuspension due to dredging.

EPA used three techniques to examine fish tissue concentrations: a wet weight basis, a lipid normalized basis, and a lipid restricted basis. Fish tissue concentrations on a wet weight basis are reported in mg PCBs/kg fish tissue. Lipid normalized concentrations are wet weight concentrations that have been divided by the lipid content of the fish and are reported in mg PCBs per kg of lipid. This method controls for the effect of changes in lipid content on PCB concentration, but assumes that PCB concentration and lipid content are perfectly correlated, which is never the case. This method makes datasets to appear comparable across time. Since a general decrease in the lipid content of fish was observed across the time period represented by the data, lipid normalized trends show slower rates of decline than wet weight concentrations. The lipid restricted analysis attempted to control for the non-linear relationship between lipid content and PCB concentration by analyzing fish in groups of similar lipid content. This method, while sound in theory, reduced sample sizes to unprofitably small numbers in practice. EPA

therefore chose the lipid normalized method as the means of estimating trends in fish tissue concentrations.

EPA examined two groups encompassing eight species of fish: sport fish, including brown bullhead, largemouth bass, smallmouth bass, striped bass, white perch, and yellow perch; and forage fish, including pumpkinseed and spottail shiner. Fish tissue data used in the 2017 analysis included NYSDEC data from 1995 to 2006 and GE data from 2004 to 2008. Data through 2016 were plotted, but not included in the trendlines.

Throughout the period, NYSDEC prepared standard fillet samples for sport fish and whole body composite samples for forage fish. GE generally followed the same procedures, but beginning in 2007, GE prepared fillet samples by removing the rib from the fillet, creating a dataset of "rib out" samples. This sampling method is inconsistent with that used by NYSDEC. In 2014 a special study was conducted to test the usability of the rib out data. The test used was: "If the margin of error between rib-on and rib-off measurements is less than 20% of the average of lipid normalized PCB concentrations with a 95% level of confidence, then the measurements are considered interchangeable" (USEPA 2015). The study used paired samples (two samples from the same fish) from largemouth and smallmouth bass ("black bass") collected specifically for the study. Wet weight rib out measurements were found to be different by a factor of two or more from rib in measurements and were deemed not usable; lipid normalized paired samples were found to have an average difference of less than 20%, but the difference for individual paired samples could be up to 75%. Importantly, the NYSDEC standard fillet results were found to be consistently greater than for the rib out fillet measurements. The measurements differed by a factor of two in a quarter of the cases. Despite this, EPA concluded that the 2007-2008 GE lipid normalized data are comparable to prior standard samples. These two years of data were included in the lipid normalized trends presented by EPA that were averaged to yield an 8% recovery rate for Tri+PCBs in fish. Yet again, the procedure to justify the use of the rib out dataset carries a large uncertainty and the effect of this uncertainty on trends was not evaluated.

EPA plotted $TPCB_{HE}$ transformed values vs year for each of the eight species in RSs 1-7 (with RSs 1-3 representing the Upper Hudson and RSs 4-7 representing the

Lower Hudson). For species/RS combinations where records were insufficient, a trendline was not calculated. This applied to spottail shiner, striped bass, and white perch in the Upper Hudson sections, spottail shiner in RSs 5-7, and smallmouth bass, largemouth bass, brown bullhead, and yellow perch in RS 7.

EPA then took the weighted average of the recovery rates calculated for sport fish species in the Upper Hudson; three species are represented in this weighted average 8% rate, which EPA found to be consistent with a first order half-life value of 8 years and consistent with model output for rates of decline in PCB concentration. EPA chose to include three sport fish species since these proportions of species are considered to represent the typical anger's fish diet, and therefore the pathway for human consumption and exposure to the contaminants. The so-called "Frankenfish" approach in weighting the average by species and River Section length was developed in the mid-1990s and it is unclear whether these proportions are still representative of the population's diet, especially in light of demographic changes. Different groups within the population may consume different species or use different preparation techniques than the EPA analyses assume.

In an effort to test the effect of data transformation into homologue equivalent measurements on the estimated decay rate, EPA calculated average decay rates by species and river section and plotted these against River Mile for both $TPCB_{HE}$ and $TPCB_{Aroclor}$ measurements. However, about 50% of the samples used in the $TPCB_{HE}$ trend analyses were eliminated for this step by selecting only those species-River Section combinations with at least 100 samples and 8+ years of data. This elimination procedure censors out a large portion of the data and the effect of this has not been statistically evaluated.

4. Replication and Variation of Fish Tissue Concentration Trends

- EPA's technique (i.e., decisions as to which data were included in the trend analyses) was replicated.
- Variations on EPA's technique were plotted to investigate the effects of data inclusion on the fish tissue recovery rates.
- Overall, fish tissue rates are highly variable, and the use of an average rate is unrepresentative of this variation and deceptive when making conclusions about the protectiveness of the remedy.
- Average rates calculated for each variation on the EPA technique were consistently lower than that calculated for the replication of the EPA technique.

The analyses presented here were conducted as a basic and preliminary means of showing the uncertainty in EPA's predictions, specifically regarding the 8% per year recovery rate for Upper Hudson sport fish species on a lipid normalized basis. The development of a method for reducing that uncertainty to reasonable levels would require more rigorous statistics in handling the data. The methods employed by EPA and followed here are inadequate to make confident estimates about how long fish in the Hudson will truly take to recover and meet the goals of the ROD.

Depending on which data are included or excluded (i.e. rib out data or Aroclor-based measurement data), the average recovery rate differs substantially. These variations in data inclusion, along with a replication run using the same parameters reported by EPA, were plotted using R statistical software, and an exponential curve was fit to the data (Figure 2, Figures 2A – 3O, attached at the end of this report). The results were coefficients taken as the percent per year declines in TPCB concentrations. The average 8% rate is shown to be uncertain when it is not reproducible with the slight variations in data inclusion. In fact, the variations consistently produced average rates of recovery lower than the rate calculated using EPA's approach. EPA's approach therefore results in recovery rates that are systematically biased high; the EPA rate is at the fastest end of the range of recovery rates found by applying slight changes (within the reasonable range) to the EPA procedure.

Furthermore, in EPA's reported rates (Table A3-3) and in all the subset variations used here for comparison, the individual rates of recovery vary drastically by species and river section, with the fastest rate of recovery at -18% per year (a negative rate indicates a decrease in concentration) and the least promising rate an increasing trend of +4% per year (Tables 1 and 2). Importantly, the use of an average rate, while useful in representing the central tendency of recovery rates, is deceptive in determining EPA's protectiveness statement for the Site, because those fish populations with slow recovery rates or slightly increasing trends have half-lives several decades longer than the 8 years suggested by the 8% rate. These populations will continue to be an exposure risk for human health beyond the timeframe suggested by the 2017 Proposed FYR.

Table 1 – Lipid Normalized $TPCB_{HE}$ vs. SDATE Recovery Rates (%/year) and Corresponding Half Lives (years); Negative Half-life Indicates Increasing Trend

		Trend A (Exclude Rib Out)		Trend B (Include rib out; EPA method)	
		Recovery Rate	Half-life	Recovery Rate	Half-life
RS 1	Largemouth Bass	-3.97	17.48	-9.29	7.46
	Brown Bullhead	-6.53	10.61	-8.16	8.50
	Yellow Perch	-10.62	6.52	-15.00	4.62
	Smallmouth Bass	-17.50	3.96	-12.76	5.43
	Pumpkinseed	-5.00	13.87		
RS 2	Largemouth Bass	-3.20	21.64	-7.63	9.09
	Brown Bullhead	1.81	-38.28	-2.27	30.59
	Yellow Perch	-15.55	4.46	-20.18	3.43
	Smallmouth Bass	-13.99	5.00	-15.24	4.55
	Pumpkinseed	-5.28	13.13		
RS 3	Largemouth Bass	-6.37	10.89	-10.75	6.45
	Brown Bullhead	-2.53	27.37	-3.13	22.14
	Yellow Perch	-18.56	3.73	-16.76	4.14
	Smallmouth Bass	-0.36	191.10	-3.87	17.94
	Pumpkinseed	-9.78	7.09		
	Arithmetic Mean Recovery Rate for Sport Fish	-8.12	~ 8	-10.42	~ 6
	Arithmetic Mean Recovery Rate Including Pumpkinseed	-7.83	~ 8		
	Species and Length Weighted Average Recovery Rate ("Frankenfish")	**-5.31**	**~ 13**	**-7.96**	**~ 8**
	Max Rate (Slowest recovery)	1.81	-38.28	-2.27	30.86
	Min Rate (Fastest recovery)	-18.56	3.73	-20.18	3.43
	Standard Deviation of Rates	6.26		5.74	
	Standard Error of the Mean	1.62		1.66	

Table 2 – Lipid Normalized $TPCB_{Aroclor}$ vs. SDATE Recovery Rates (%/year) and Corresponding Half-Lives (Years); Negative Half-life Indicates Increasing Trend

		Trend A (Exclude Rib Out, Include 2016 data for RS1)		Trend B (Include Rib Out)	
		Recovery Rate	Half-life	Recovery Rate	Half-life
RS 1	Largemouth Bass	-6.19	11.20	-10.12	6.85
	Brown Bullhead	-4.37	14.95	-6.05	11.46
	Yellow Perch	-8.36	8.29	-12.82	5.41
	Smallmouth Bass	-9.69	7.15	-7.29	9.51
	Pumpkinseed	-5.05	30.93		
RS 2	Largemouth Bass	-2.44	12.59	-10.78	6.43
	Brown Bullhead	3.34	-20.76	-3.34	20.73
	Yellow Perch	-10.06	6.89	-16.20	4.28
	Smallmouth Bass	-7.42	9.34	-10.81	6.41
	Pumpkinseed	-17.97	3.86		
RS 3	Largemouth Bass	-6.28	11.05	-9.54	7.27
	Brown Bullhead	-1.54	45.04	-1.79	38.72
	Yellow Perch	-13.03	5.32	-12.34	5.62
	Smallmouth Bass	4.16	-16.66	0.38	-184.47
	Pumpkinseed	-9.76	7.10		
	Arithmetic Mean Recovery Rate for Sport Fish	-5.16	~ 13	-8.39	~ 8
	Arithmetic Mean Recovery Rate Including Pumpkinseed	-6.31	~ 11		
	Species and Length Weighted Average Recovery Rate ("Frankenfish")	**-4.39**	**~ 15**	**-6.92**	**~ 9**
	Max Rate (Slowest Recovery)	4.16	-16.66	0.38	-184.47
	Min Rate (Fastest Recovery)	-17.97	3.86	-16.20	4.28
	Standard Deviation of Rates	5.79		4.91	
	Standard Error of the Mean	1.58		1.42	

Several data criteria were chosen to create variations on the data subset used by EPA. EPA's lipid normalized analysis generated a trendline for $TPCB_{HE}$ data between 1995 and 2008 for all three Upper Hudson River Sections, and included the GE rib out samples taken in 2007 and 2008. To address changes in the average decay rate caused by uncertainty in the transformation of $TPCB_{Aroclor}$ to $TPCB_{HE}$, two separate trendlines were calculated for each of these datasets. Trend A excludes rib out data taken by GE and Trend B includes the rib out data. The species-weighted average and arithmetic average decay rates were calculated for sport fish species (largemouth bass, brown bullhead, and yellow perch included in the weighted average, with smallmouth bass included in the arithmetic mean). Both Trend A and Trend B apply to the period between 1995 and 2008.

Trend A was also applied to records for pumpkinseed, for which the majority of samples were whole-body. Rib out fillet samples were therefore not an issue for this species and there is no Trend B trendline for pumpkinseed rates.

Trend B represents the same data subset used by EPA. When applied to the original $TPCB_{Aroclor}$ data, this trend represents a variation on EPA's method; when applied to the transformed $TPCB_{HE}$ data, this trend represents the replication of EPA's method. A diagram of the comparative variations of EPA's method is shown in Figure 1.

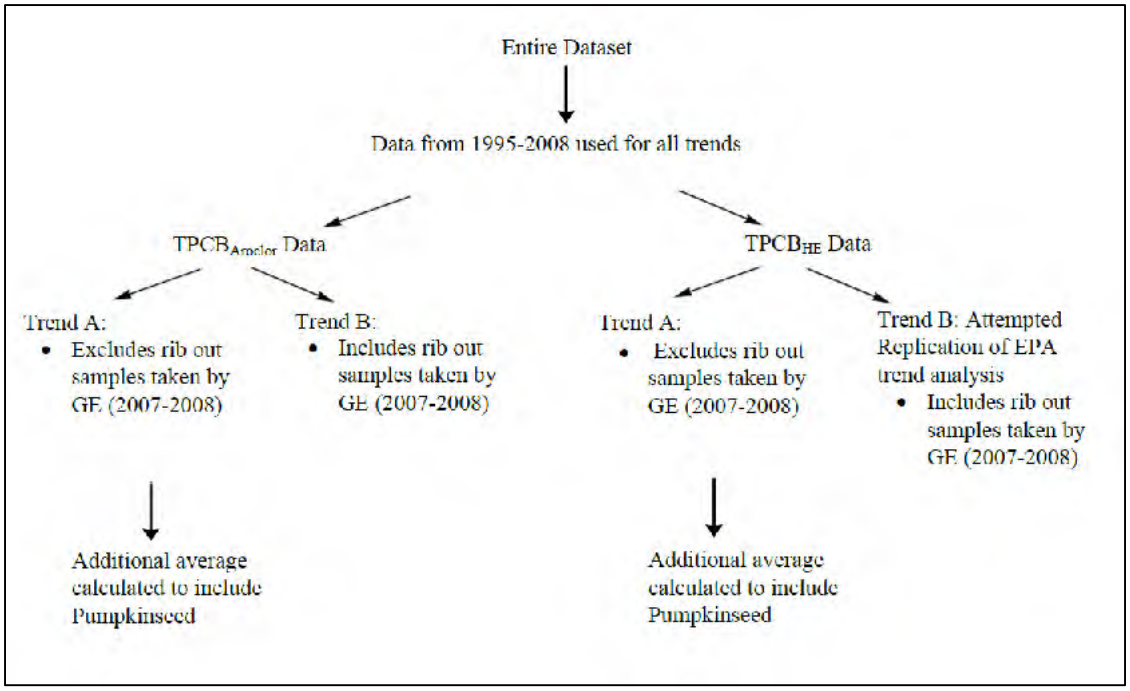


Figure 1 – Variations of EPA Data Criteria for Fish Samples Through Time.

The replication of EPA's analysis reproduced the weighted average 8% rate reported by EPA. Individual rates by species and RS for $TPCB_{HE}$ Trend B are different than those reported in Table A3-3, indicating that $TPCB_{HE}$ Trend B is not a true replication of the EPA process and likely includes differences in data criteria. In this analysis, it was assumed that the sampling design, which was to target fish of legal length (defined for each species in the Baseline Monitoring Program Quality Assurance Project Plan, QEA 2003), resulted in the entire database consisting of adult fish samples. Upon further query of the database, it is now known that no more than 10% of the data used for trends here were samples under the minimum legal length. Despite this possible discrepancy between the EPA method and the replication method, the replication achieved similar variation between rates and an 8% weighted average. Consistency between subsets used in this report allows a general and relative comparison of the EPA results with results from potential changes to that approach.

Table 1 shows the results of $TPCB_{HE}$ trends, while Table 2 shows the results of $TPCB_{Aroclor}$ trends. $TPCB_{HE}$ results show average recovery rates higher by 2-3% per year (arithmetic average) or 1% per year (weighted average) than the $TPCB_{Aroclor}$ average

recovery rates for both Trends A and B. In both Tables, Trend A shows average recovery rates lower than Trend B rates by about 2-3% per year. Inclusion of pumpkinseed recovery rates did not notably change the average recovery rates for Trend A in either Table. Out of all four different approaches, the species weighted average rate for $TPCB_{HE}$ Trend B is the fastest and differs from other rates by 25-40%.
It is apparent that by selecting these criteria for data to be included in the 2017 Proposed FYR fish tissue trend analysis, EPA overestimated the average rate of decline for adult sport fish in the Upper Hudson. For comparison, recovery times to reach interim concentration goals were calculated for each weighted average recovery rate using a simple exponential decay equation (Table 3).

Table 3 – Recovery Times for Species and Length Weighted Average Recovery Rates (Current Average Wet Weight Concentration: 1.3 mg/kg)

Approach	Rate	Years to 0.4 (mg/kg)	Years to 0.2 (mg/kg)
HE Trend A	-0.05	22	35
HE Trend B (EPA)	-0.07956	15	24
Aroclor Trend A	-0.04393	27	43
Aroclor Trend B	-0.0692	17	27

Exponential Equation:

$$y = ae^{-kt}$$

$$t = \frac{\ln\left(\frac{y}{a}\right)}{-k}$$

a = Current average wet weight concentration (1.3 mg/kg)
y = Interim goal wet weight concentration (.4 or .2 mg/kg)
k = Exponential decay rate
t = Time to interim goals in years

More important than the difference between average rates, however, is the difference between individual rates with variation in the method. For example, consider largemouth bass trends for RS2 (Figure 2). Removing the rib-out samples from the

analysis causes the recovery rate to drop from 7.63% per year to 3.2% per year in $TPCB_{HE}$ measurements and from 10.78% per year to 2.44% per year in $TPCB_{Aroclor}$ measurements. The difference in half-life values calculated from these rates is an additional 10-20 years for TPCB concentrations to reach half of their present value. If inclusion of the rib out data produced a trendline truly representative of fish tissue MNA recovery, then the rate of recovery would not be consistently slower across species and River Sections once those data are removed. It is these individual recovery rates and half-life predictions that are relevant to the protectiveness of the remedy, not an oversimplified average.

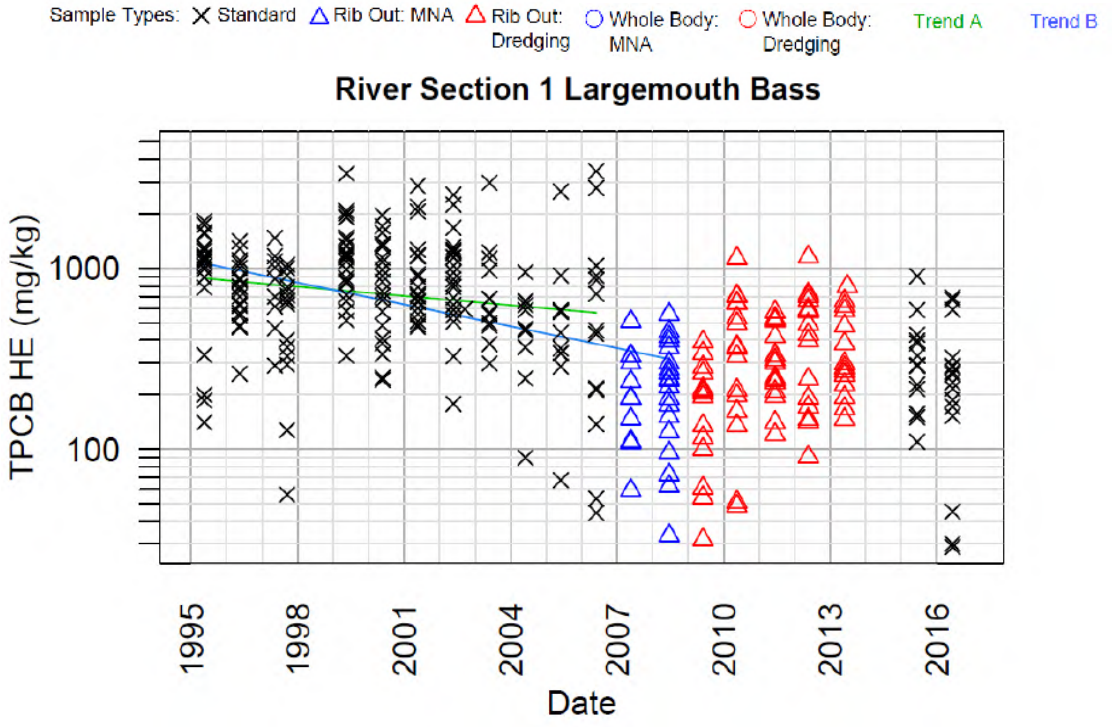


Figure 2 – Example Plot of Differences in Trend (A and B, in Green and Blue, Respectively) due to Exclusion of Rib Out (A) and Inclusion of Rib Out (B). The Trends Produce Very Different Half-life Estimations, with Trend A at 17.5 Years and B at 7.5 Years.

As a further illustration of the large uncertainty and unsuitability of the fish data for determining the protectiveness of the remedy, pre-dredging data were sectioned into three intervals: 1995-1999, 2000-2004, and 2005-2008. For each of these intervals, as a

basic test of the consistency of the rate of decline for each species and RS combination, a different trendline was plotted. These plots (Figure 3, Figures 4 A-R at end of document) show extreme variability of trends between time periods, demonstrating that choosing arbitrary parameters for data to be included in the analysis is not successful in capturing the data to derive a reproducible rate for PCB decline. The annual mean TPCB concentration was also plotted for each species and River Section combination (Figure 4, Figures 5 A-R at end of document).

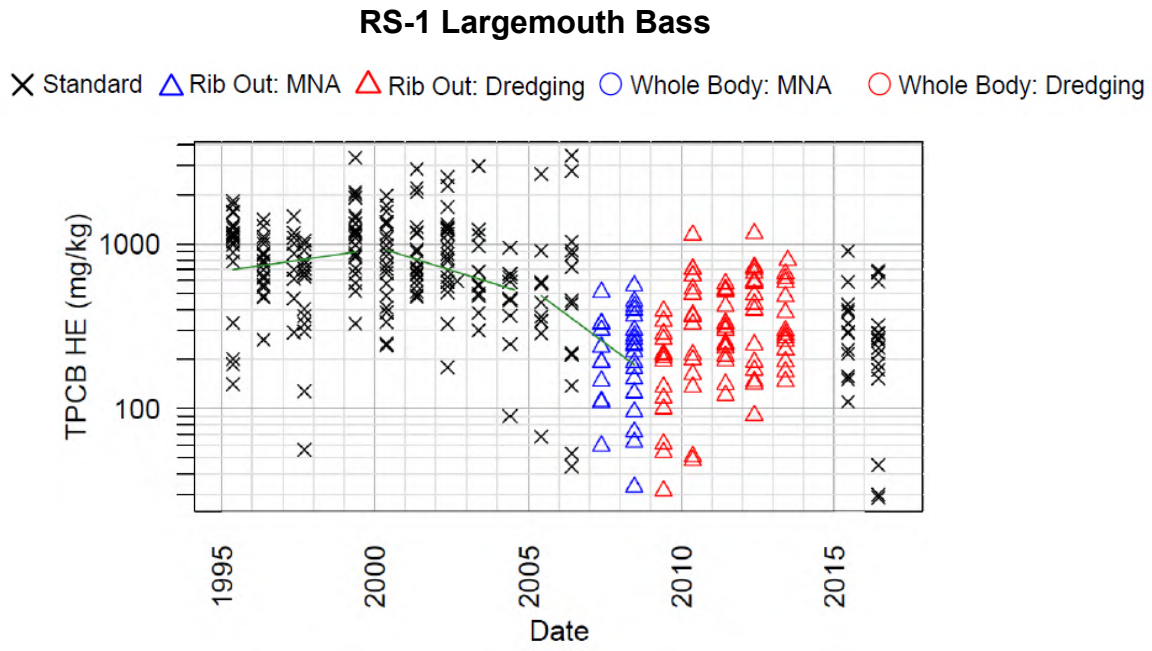


Figure 3 – Example Plot of Differences in Trend (Green Lines) due to Selection of Sediment Data Intervals (1995-1999, 2000-2004, and 2005-2008). The Slopes for These Trends Range from Positive to Negative, Highlighting the Uncertainty Associated with Trend Determination.

Annual Mean TPCB Concentration, RS 1 Largemouth Bass

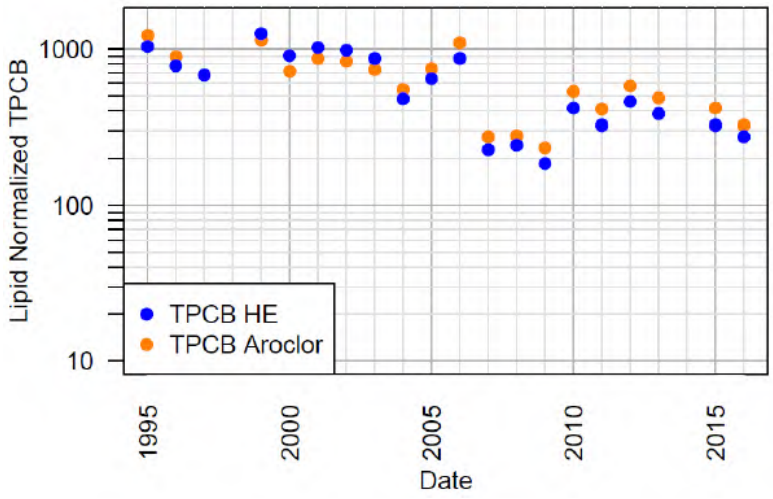


Figure 4 – Example Plot of Annual Mean TPCB Concentration. The Change in the Relationships Between the $TPCB_{Aroclor}$ to $TPCB_{HE}$ Concentrations Shows the Two Methods Are Not Equivalent.

5. Surface Sediment Cumulative Plots

- Preliminary cumulative distribution plots of surface sediment TPCB concentrations show a general improvement between SSAP (2002-2005) and OM&M (2016) datasets.
- When the SSAP dataset is separated into dredging and non-dredging area sample sets, cumulative distribution plots show lesser degrees of improvement in non-dredging areas than the improvement shown by plotting all SSAP samples. Non-dredging areas in River Section 1 show very little or no improvement (Figure 5A).

As a comparative exercise to examine pre-dredging vs post-dredging surface sediment data, TPCB values (Aroclor sum measurements) from the SSAP and 2016 OM&M programs were plotted on the x-axis vs cumulative probability on the y-axis. Each plot (Figures 5-7) show an improvement in surface sediment concentrations between the two programs. However, the plots are precursory due to differences between sampling programs. Specifically, the SSAP was designed with the goal of delineating dredging areas and therefore focused in River Sections 2 and 3 on consolidated sediments of suspected elevated contamination, while the OM&M program sampled only non-dredging areas in all three River Sections. EPA acknowledges the difficulties and biases created by these differences in the Proposed FYR.

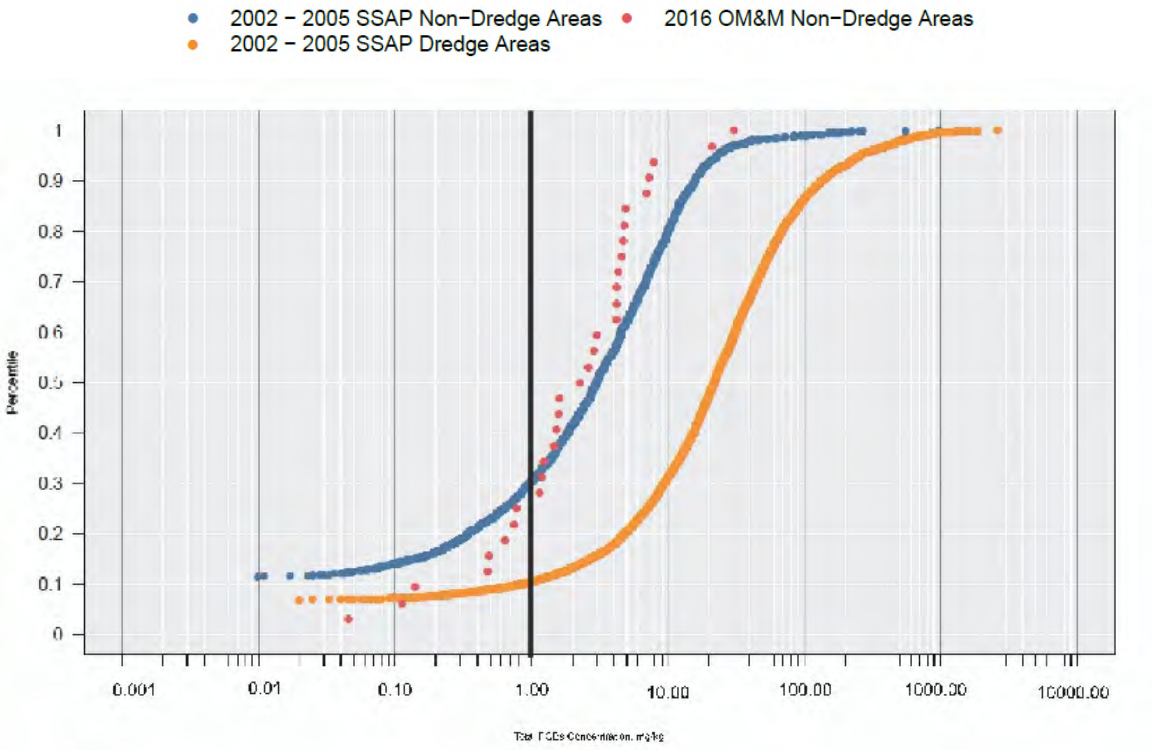


Figure 5A – Cumulative Distribution of TPCB Concentration in Surface Sediment Samples (Top 12 inches or less) in River Section 1

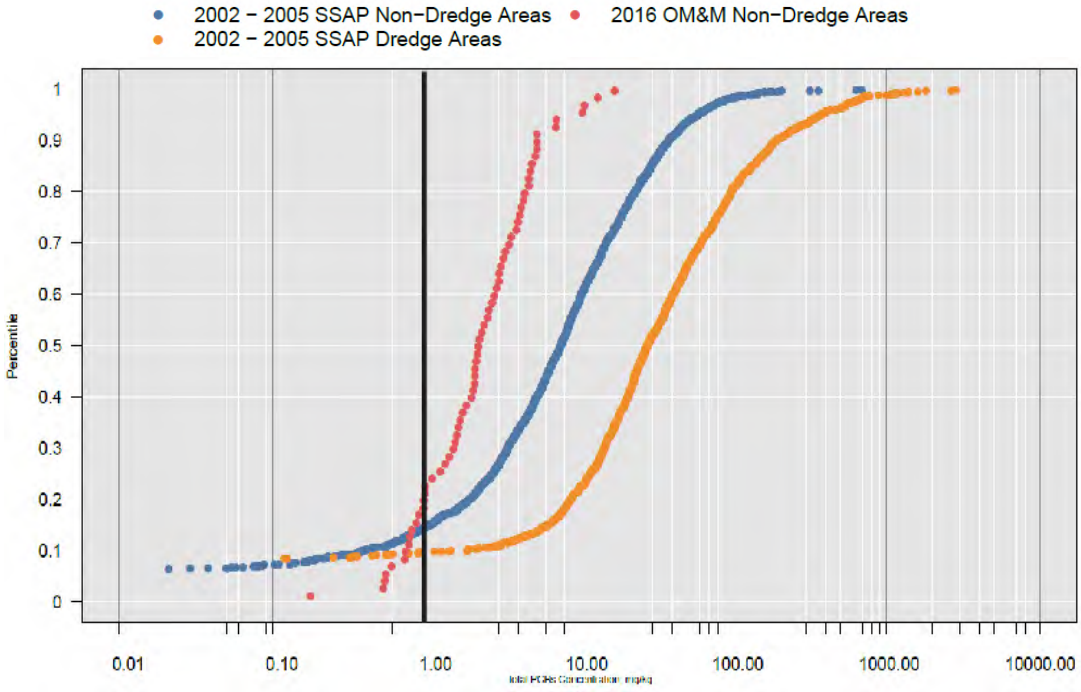


Figure 5B – Cumulative Distribution of TPCB Concentration in Surface Sediment Samples (Top 12 inches or less), in River Section 2

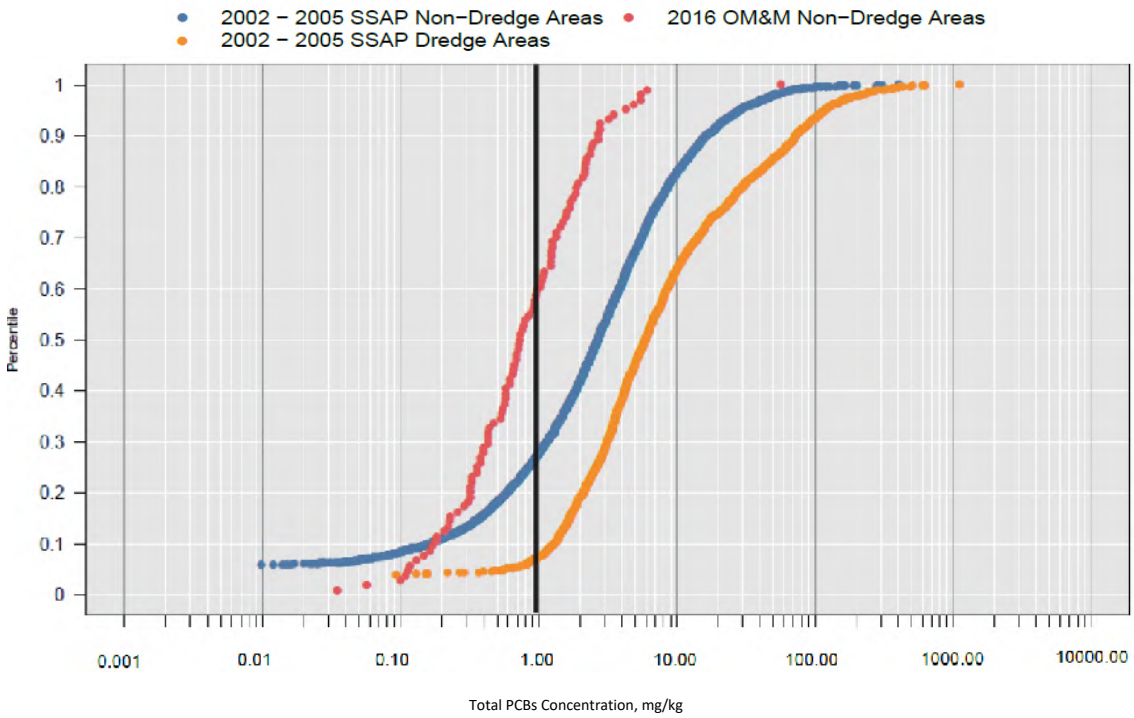


Figure 5C – Cumulative Distribution of TPCB Concentration in Surface Sediment Samples (Top 12 inches or less), in River Section 3

6. Conclusions and Future Analyses

- Both the analyses presented here and those presented by EPA cannot determine the protectiveness of the remedy with any degree of confidence.
- The slowest fish tissue recovery rates hold more weight when considering the remedy effectiveness, since these species will continue to be a pathway to human exposure past the timeframe asserted by EPA. The use of average recovery rates does not consider the variability in individual recovery rates by species.
- Future analyses should focus on quantifying and minimizing uncertainty both in the data transformation process and the comparability between datasets, including the rib in/out fish data and SSAP/OM&M sediment datasets.

Several concerns regarding the remediation-period data merit continued attention and more thorough investigation. The most pressing of these is the need for a procedure to carry the uncertainty of transforming $TPCB_{Aroclor}$ measurements into $TPCB_{HE}$ measurements into the fish tissue trend analysis. Additionally, more rigorous testing than used here or in the Proposed FYR may show comparability (or a lack of comparability) between $TPCB_{Aroclor}$ and $TPCB_{HE}$ measurements.

An additional question to be pursued is the potential effects of sample depth on rates of PCB decay in surface sediment over time. Surface sediment data is composed largely of samples with start and end depths of 0-2 inches or 2-12 inches, but it is possible that samples from 0 to 6 inches may show a different distribution of concentrations characteristic of the biotic zone. Cumulative distribution plots are expected to be useful for expanding on this question.

Overall, the preliminary analyses here need much refinement and intensification in order to make confident statements about recovery from contamination in the Upper Hudson and to reliably predict achievement of the goals of the ROD. They do show, however, that fish tissue concentration decay rates are extremely variable and that an 8% average decay rate is an highly uncertain, biased high, and oversimplified representation of this variation. The EPA 8% rate of recovery exaggerates the estimate of the rate of

natural recovery in the Hudson River. At present, it cannot be concluded from any of the analyses performed that rates of recovery are on track with the ROD model output. The data does not support EPA's conclusion that the goals of the ROD will be achieved.

7. References

Quantitative Environmental Analysis, LLC (QEA). 2003. Quality Assurance Project Plan for the Hudson River PCBs Site, Baseline Monitoring Program. September, 2003.

U.S. Environmental Protection Agency (USEPA). 2002. Record of Decision and Responsiveness Summary for Hudson River PCBs Site. December, 2002.

U.S. Environmental Protection Agency (USEPA). 2004. Final Decision Regarding General Electric Company's Disputes on Draft Phase 1 Dredge Area Delineation Report and Draft Phase 1 Target Area Identification Report. July 22, 2004.

U.S. Environmental Protection Agency (USEPA) OSWER-OSRTI Environmental Response Team. 2015. the Hudson River PCBs Superfund Site Community Advisory Group (CAG) Meeting, Saratoga Springs, NY, 1 October 2015. http://www.hudsoncag.ene.com/files/MSG_CAG_Oct2015_Hudson%20Fish_v01oct15_FINAL.pdf

United States Environmental Protection Agency (USEPA). 2017. Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site. Prepared by EPA Region 2.

FIGURES

Figure 2A: River Section 1 Largemouth Bass
Coefficients: A −3.965 %, B −9.288 %
Half Life: A 17.481 years, B 7.463 years

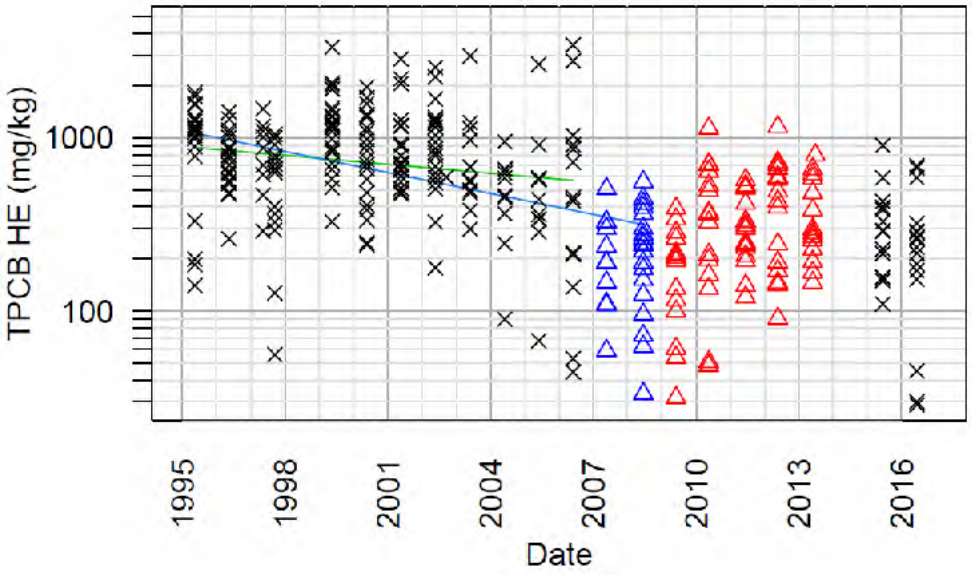


Figure 2B: River Section 1 Brown Bullhead
Coefficients: A −6.534 %, B −8.155 %
Half Life: A 10.608 years, B 8.499 years

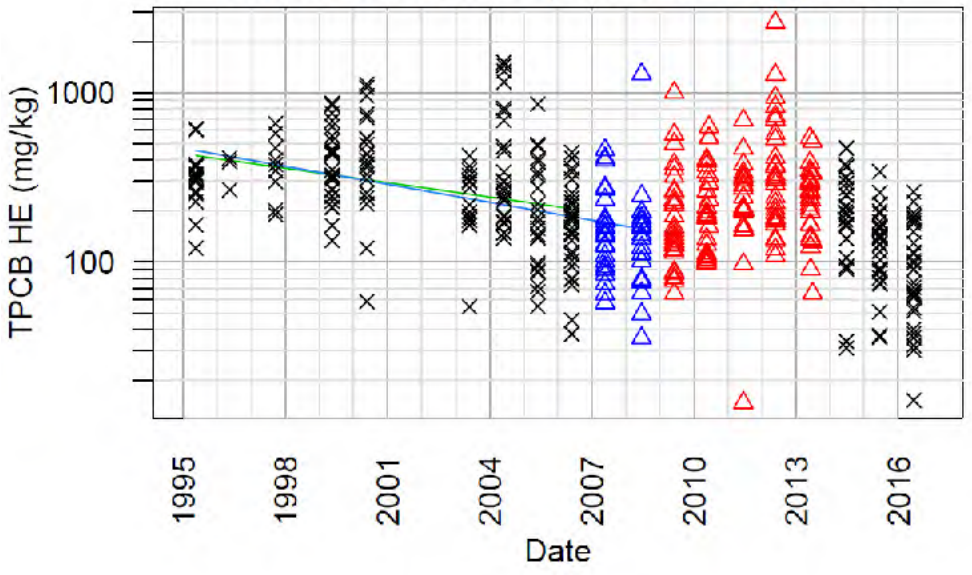


Figure 2C: River Section 1 Yellow Perch
Coefficients: A −10.624 %, B −15 %
Half Life: A 6.524 years, B 4.621 years

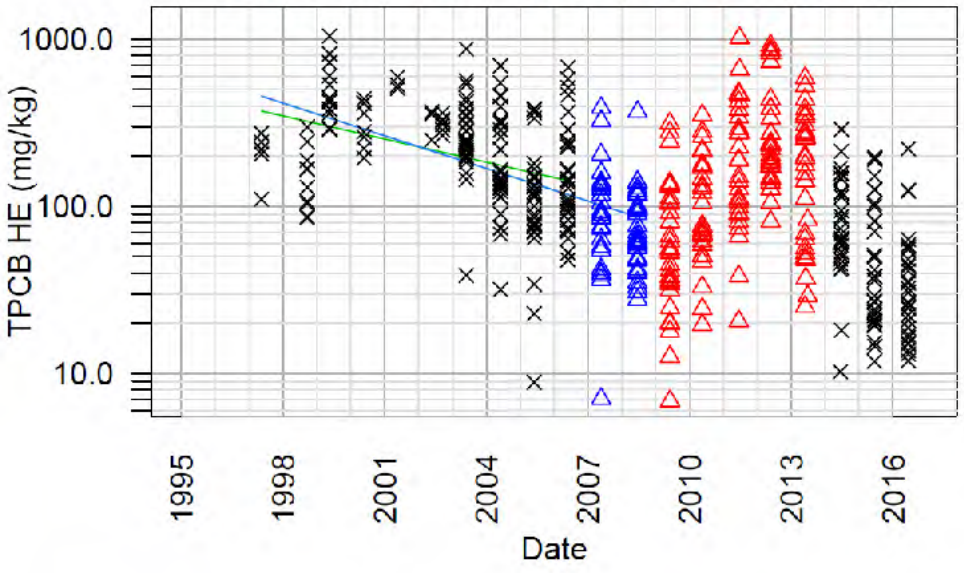
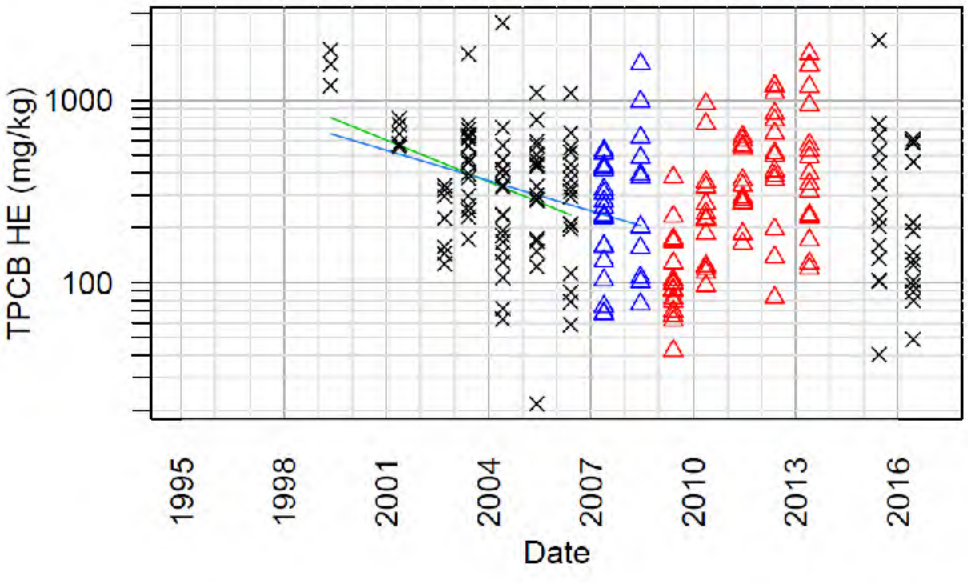


Figure 2D: River Section 1 Smallmouth Bass
Coefficients: A −17.5 %, B −12.763 %
Half Life: A 3.961 years, B 5.431 years



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 2E: River Section 1 Pumpkinseed
Coefficient: −4.998 %
Half life: 13.869

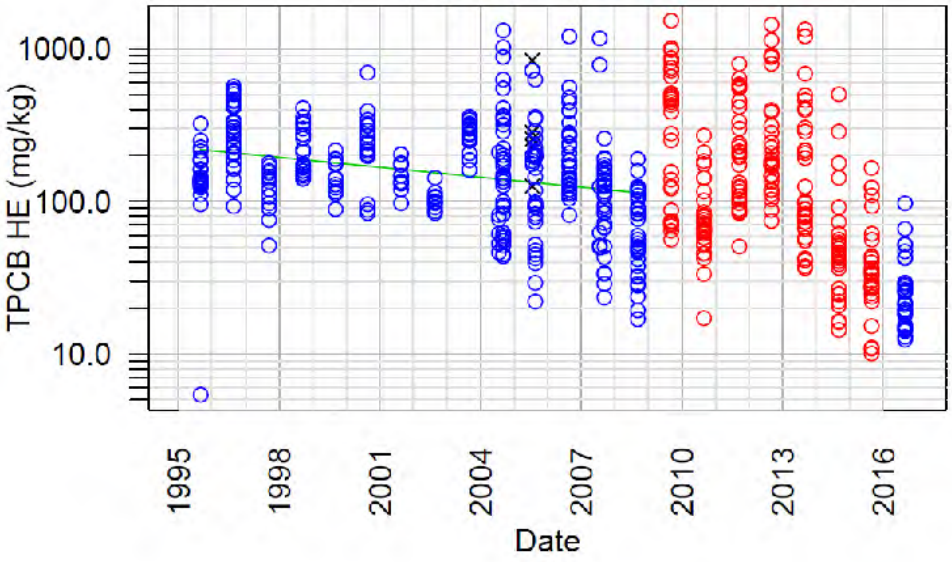


Figure 2F: River Section 2 Largemouth Bass
Coefficients: A −3.202 %, B −7.627 %
Half Life: A 21.644 years, B 9.088 years

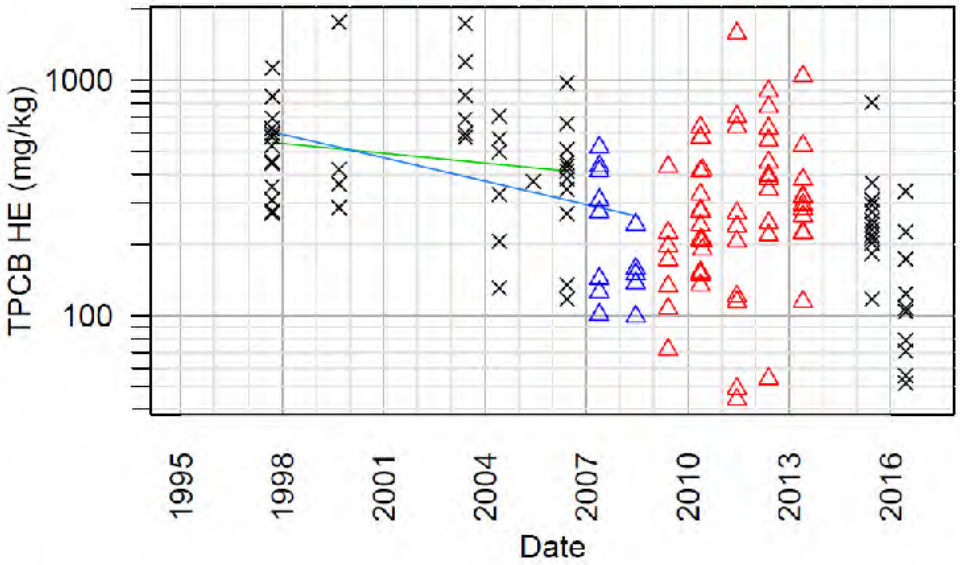


Figure 2G: River Section 2 Brown Bullhead
Coefficients: A 1.811 %, B −2.266 %
Half Life: A −38.28 years, B 30.586 years

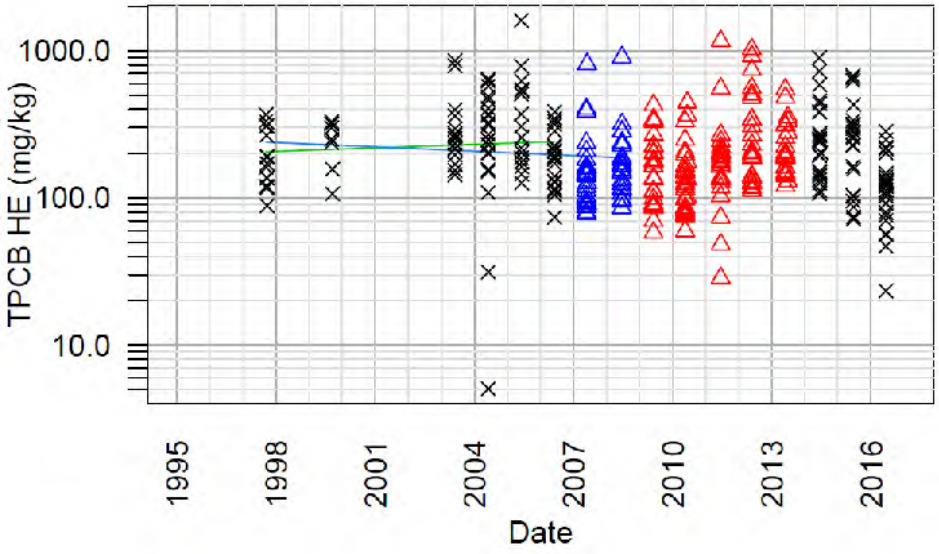
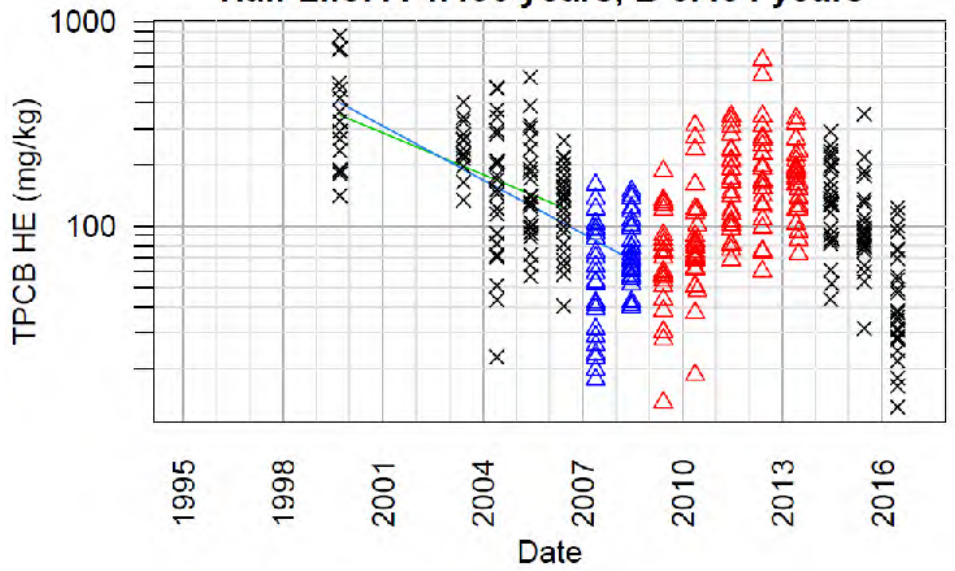


Figure 2H: River Section 2 Yellow Perch
Coefficients: A −15.554 %, B −20.182 %
Half Life: A 4.456 years, B 3.434 years



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 2I: River Section 2 Smallmouth Bass
Coefficients: A −13.865 %, B −15.236 %
Half Life: A 4.999 years, B 4.549 years

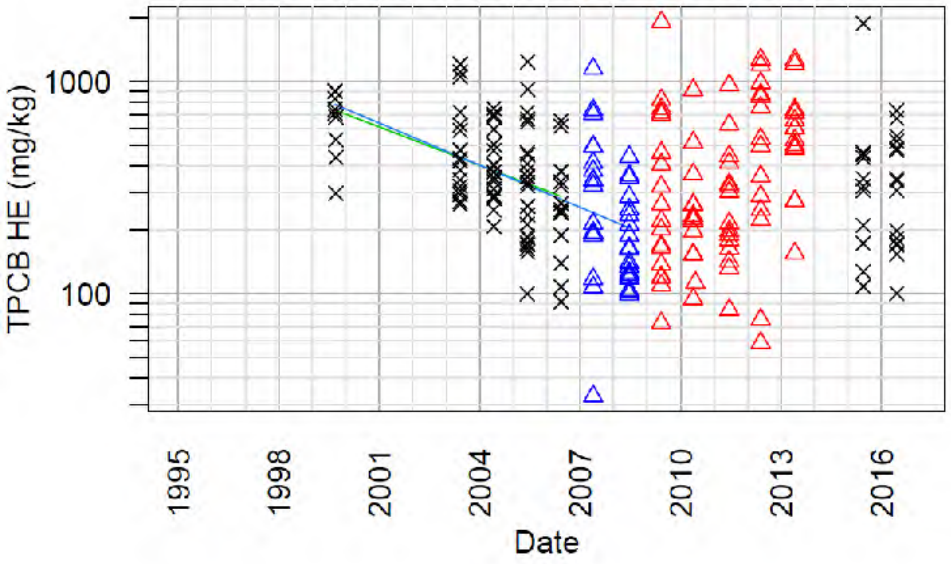


Figure 2J: River Section 2 Pumpkinseed
Coefficient: −5.279 %
Half life: 13.13

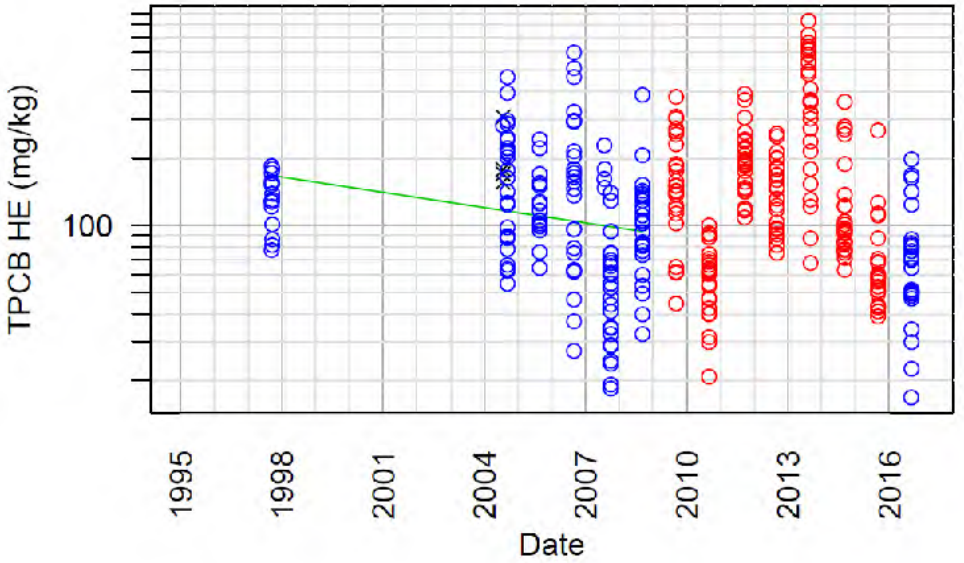


FIgure 2K: River Section 3 Largemouth Bass
Coefficients: A −6.368 %, B −10.75 %
Half Life: A 10.886 years, B 6.448 years

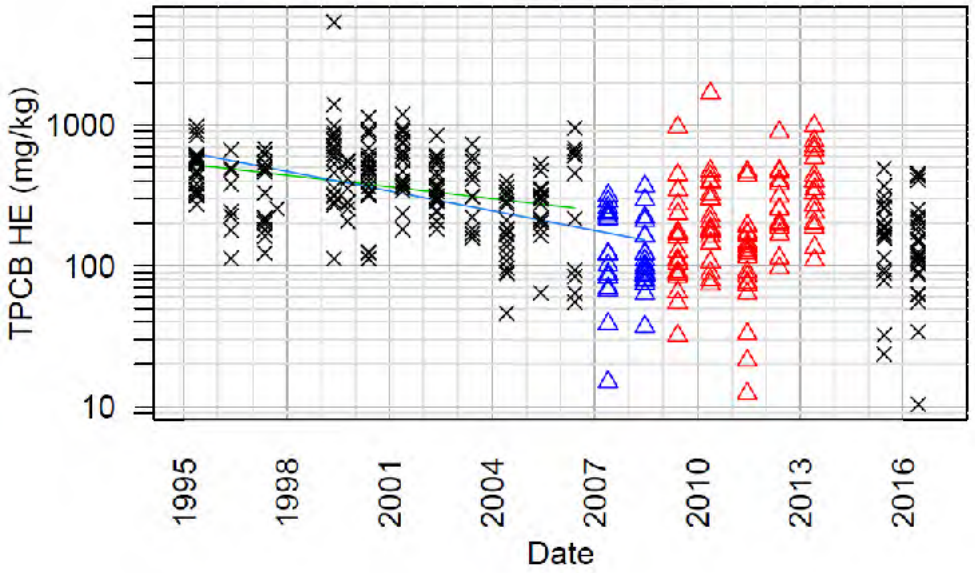
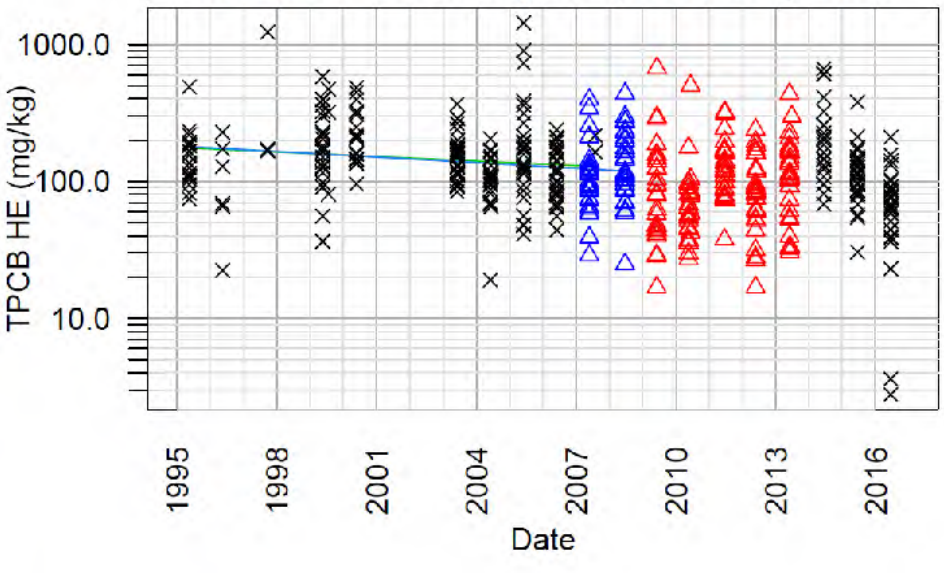


Figure 2L: River Section 3 Brown Bullhead
Coefficients: A −2.533 %, B −3.131 %
Half Life: A 27.369 years, B 22.136 years



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 2M: River Section 3 Yellow Perch
Coefficients: A −18.562 %, B −16.76 %
Half Life: A 3.734 years, B 4.136 years

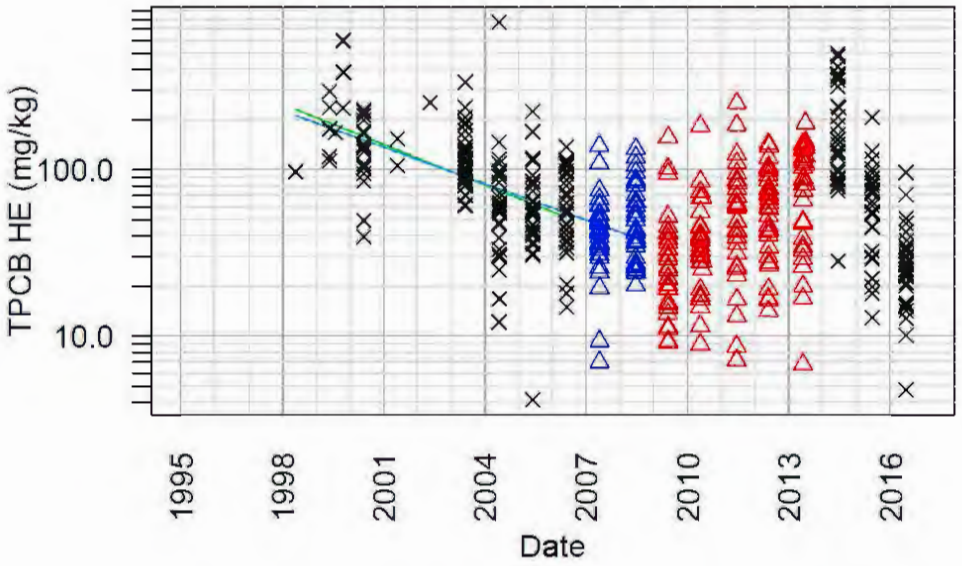


Figure 2N: River Section 3 Smallmouth Bass
Coefficients: A −0.363 %, B −3.865 %
Half Life: A 191.101 years, B 17.936 years

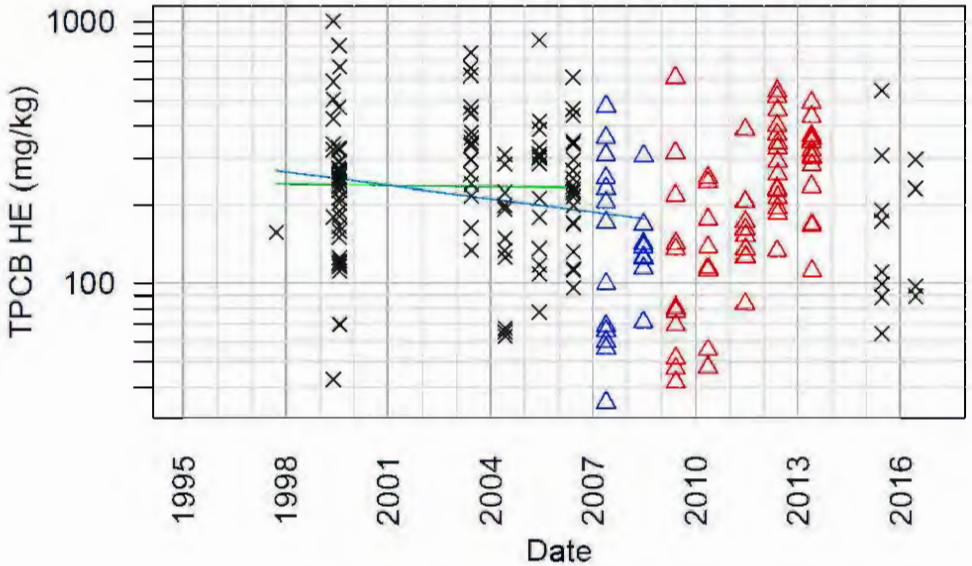
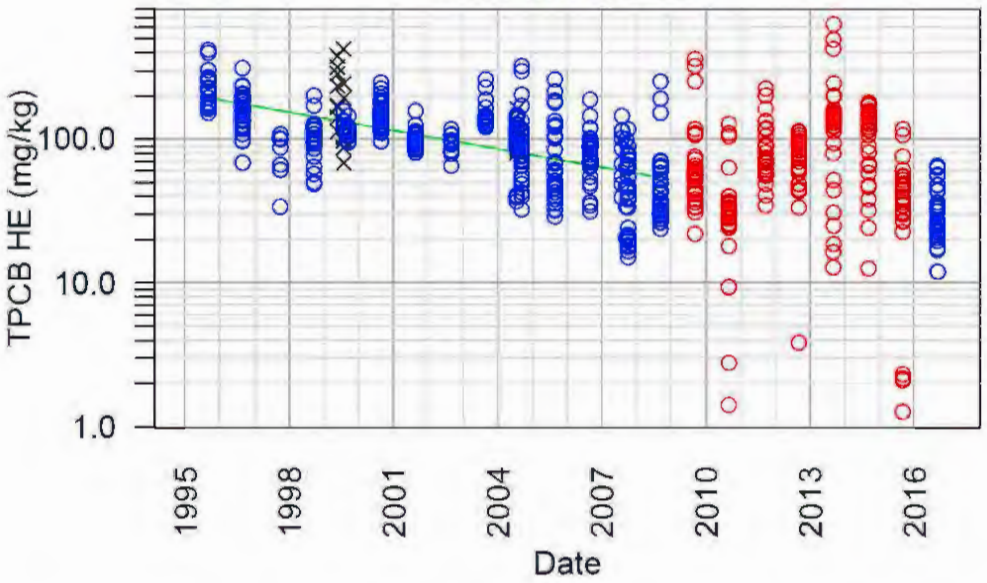


Figure 2O: River Section 3 Pumpkinseed
Coefficient: −9.783 %
Half life: 7.085



Sample Types: X Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 3A: River Section 1 Largemouth Bass
Coefficients: A −6.187 %, B −10.118 %
Half Life: A 11.202 years, B 6.851 years

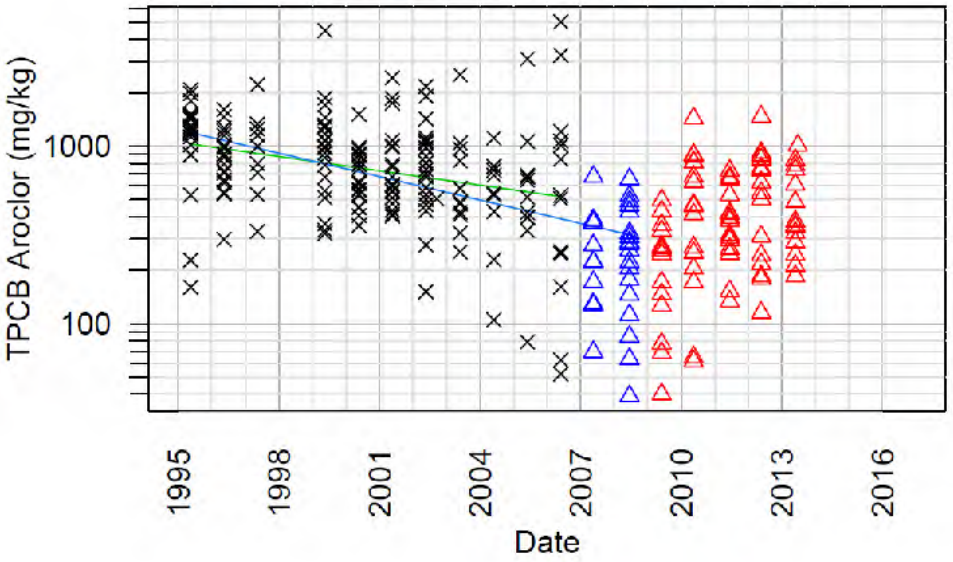


Figure 3B: River Section 1 Brown Bullhead
Coefficients: A −4.635 %, B −6.051 %
Half Life: A 14.954 years, B 11.456 years

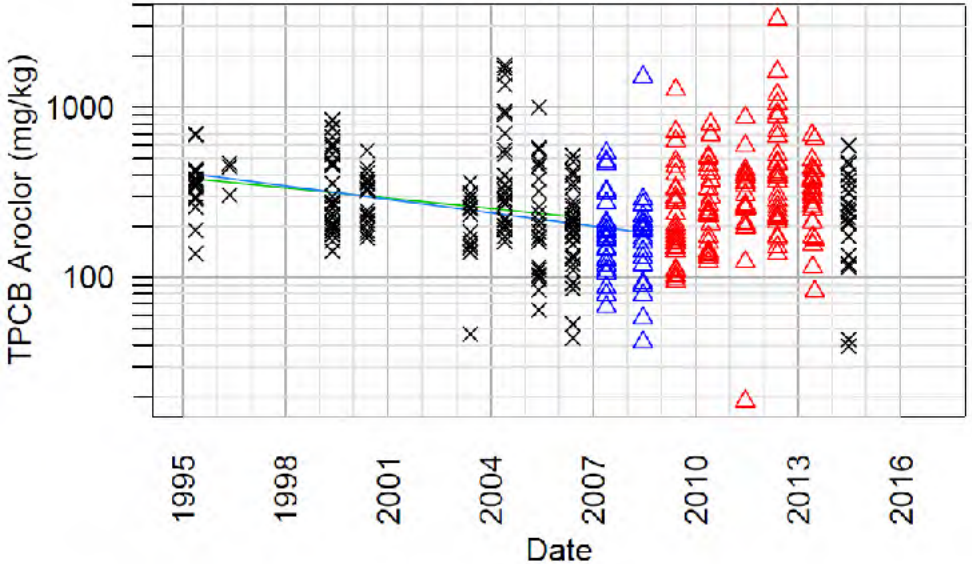


Figure 3C: River Section 1 Yellow Perch
Coefficients: A −8.357 %, B −12.817 %
Half Life: A 8.294 years, B 5.408 years

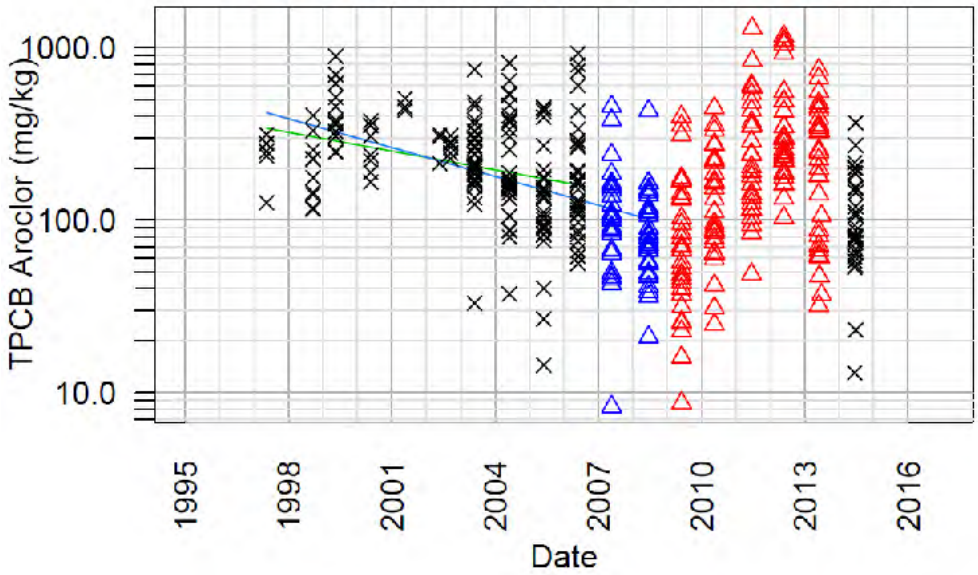
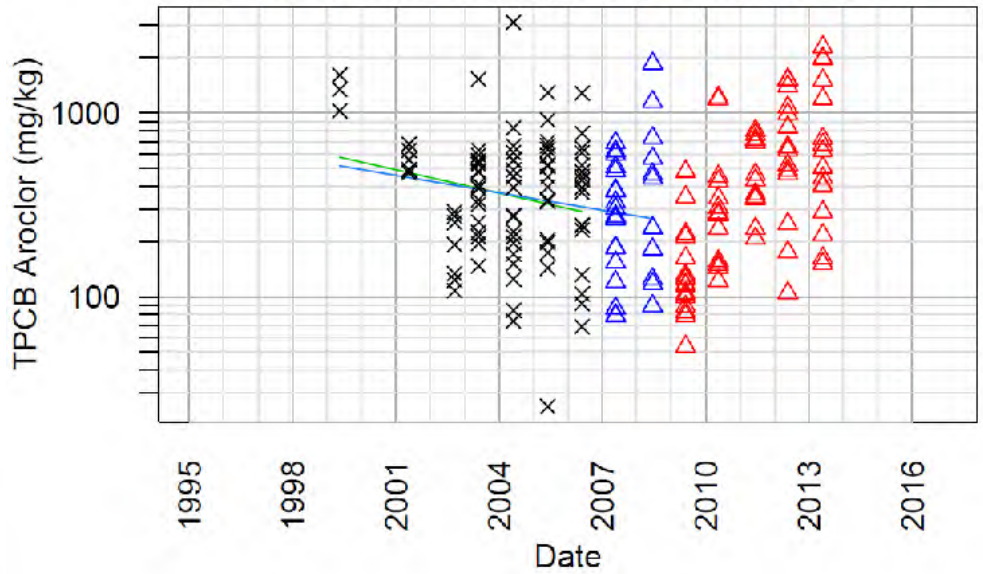


Figure 3D: River Section 1 Smallmouth Bass
Coefficients: A −9.692 %, B −7.29 %
Half Life: A 7.152 years, B 9.508 years



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 3E: River Section 1 Pumpkinseed
Coefficient: −5.505 %
Half life: 12.591

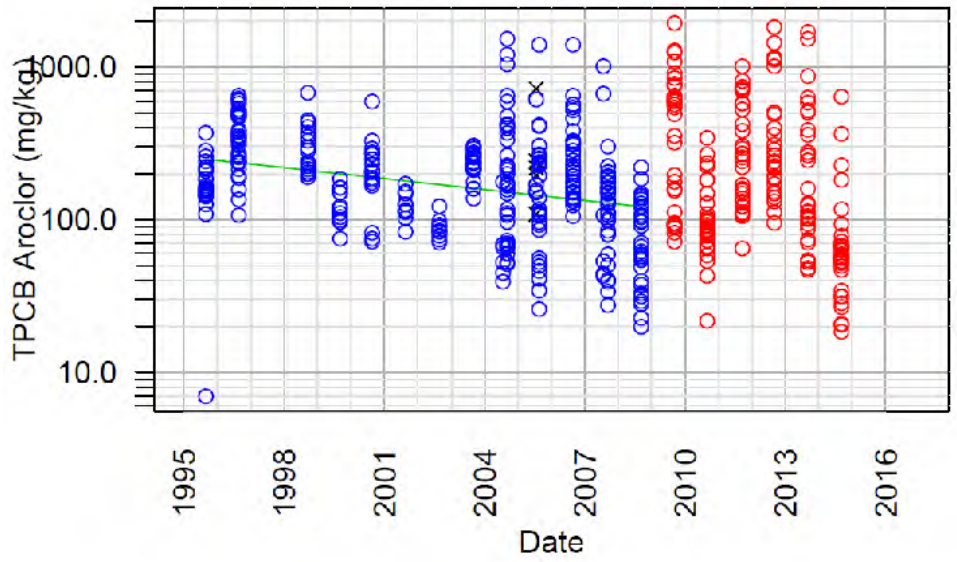


Figure 3F: River Section 2 Largemouth Bass
Coefficients: A −2.437 %, B −10.783 %
Half Life: A 28.443 years, B 6.428 years

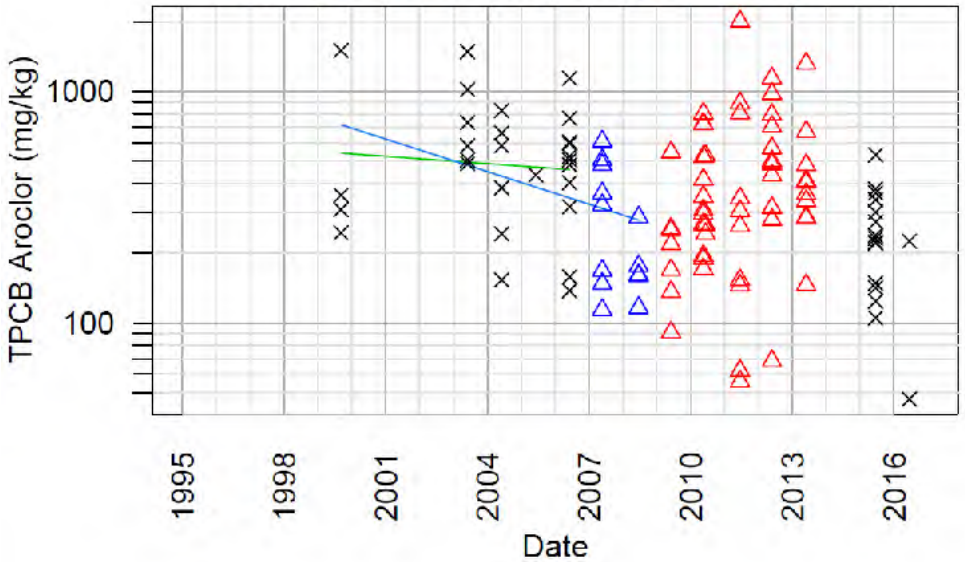


Figure 3G: River Section 2 Brown Bullhead
Coefficients: A 3.338 %, B −3.344 %
Half Life: A −20.764 years, B 20.73 years

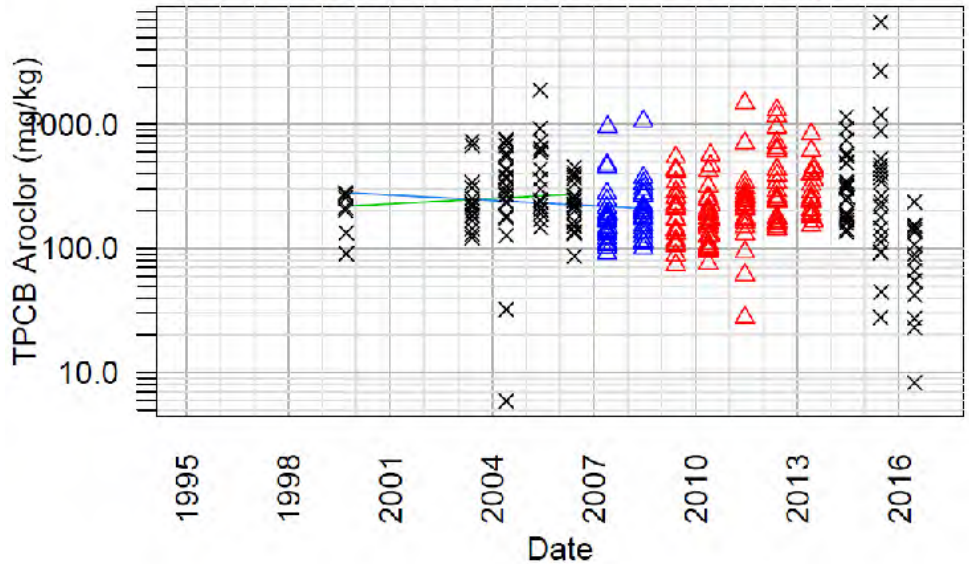
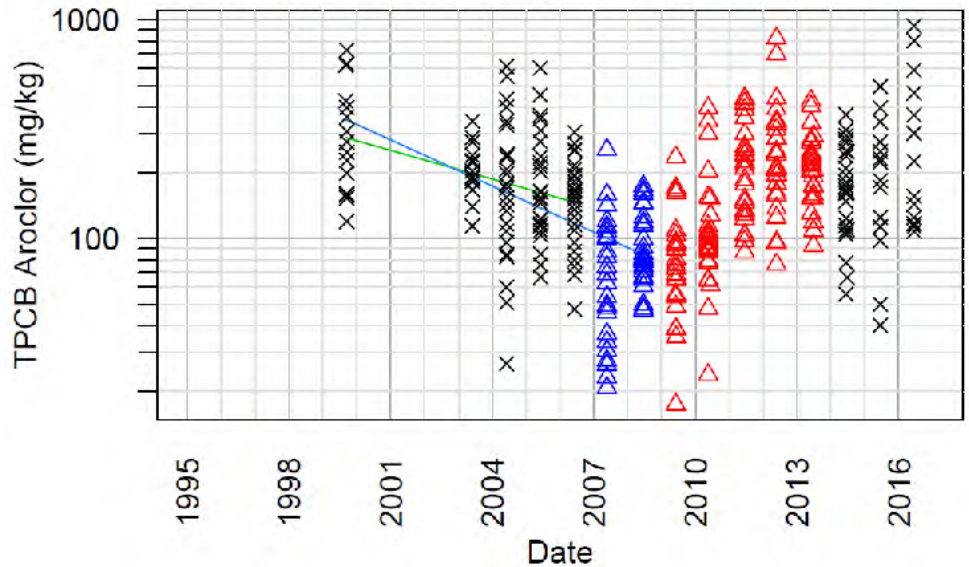


Figure 3H: River Section 2 Yellow Perch
Coefficients: A −10.059 %, B −16.203 %
Half Life: A 6.891 years, B 4.278 years



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 3I: River Section 2 Smallmouth Bass
Coefficients: A −7.421 %, B −10.814 %
Half Life: A 9.341 years, B 6.41 years

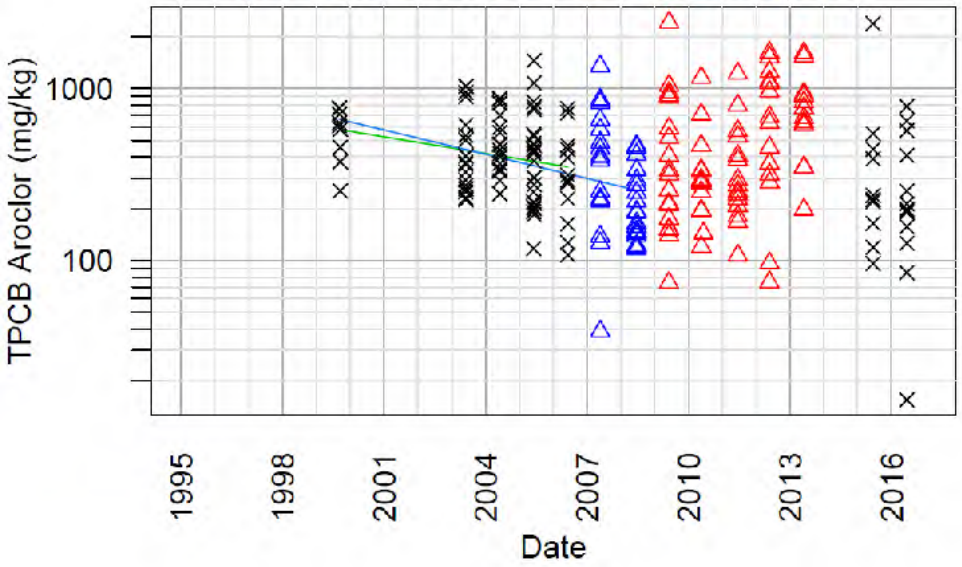


Figure 3J: River Section 2 Pumpkinseed
Coefficient: −17.968 %
Half life: 3.858

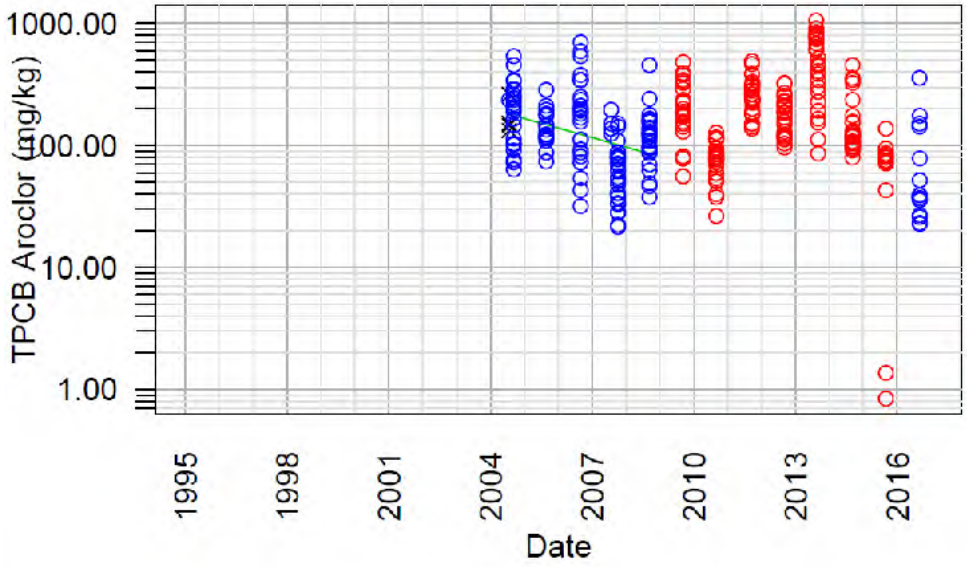


Figure 3K: River Section 3 Largemouth Bass
Coefficients: A −6.275 %, B −9.538 %
Half Life: A 11.046 years, B 7.267 years

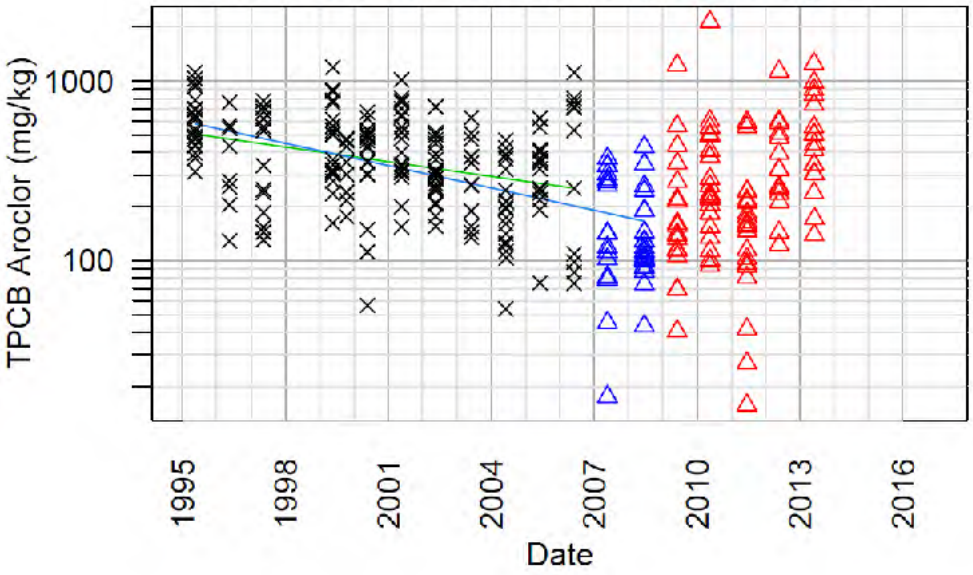
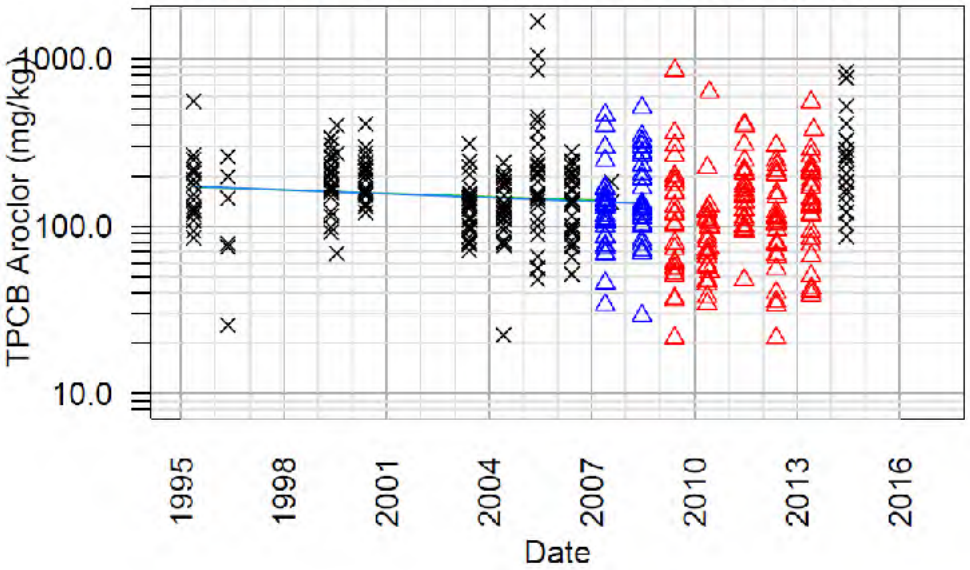


Figure 3L: River Section 3 Brown Bullhead
Coefficients: A −1.539 %, B −1.79 %
Half Life: A 45.035 years, B 38.723 years



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 3M: River Section 3 Yellow Perch
Coefficients: A −13.033 %, B −12.339 %
Half Life: A 5.318 years, B 5.617 years

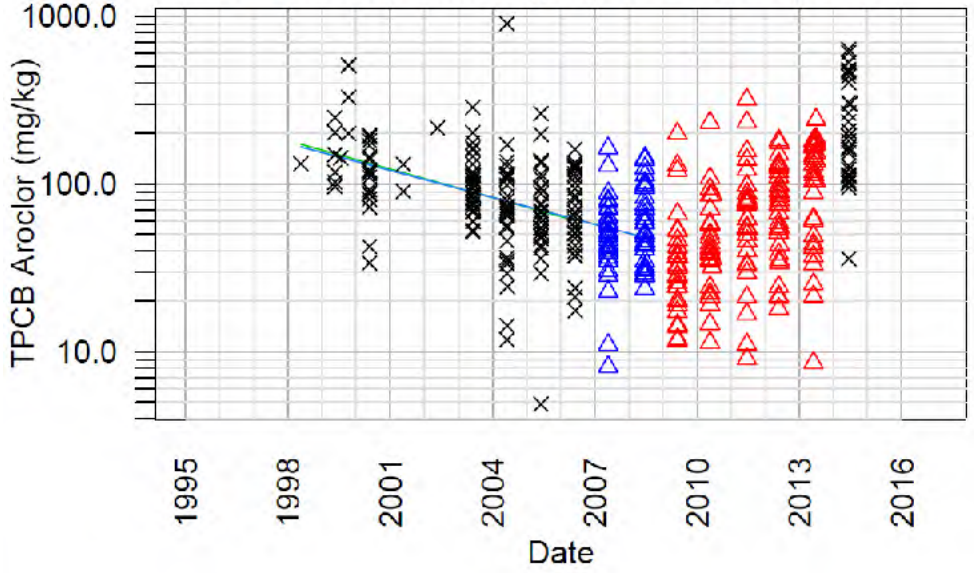


Figure 3N: River Section 3 Smallmouth Bass
Coefficients: A 4.16 %, B 0.376 %
Half Life: A −16.662 years, B −184.468 years

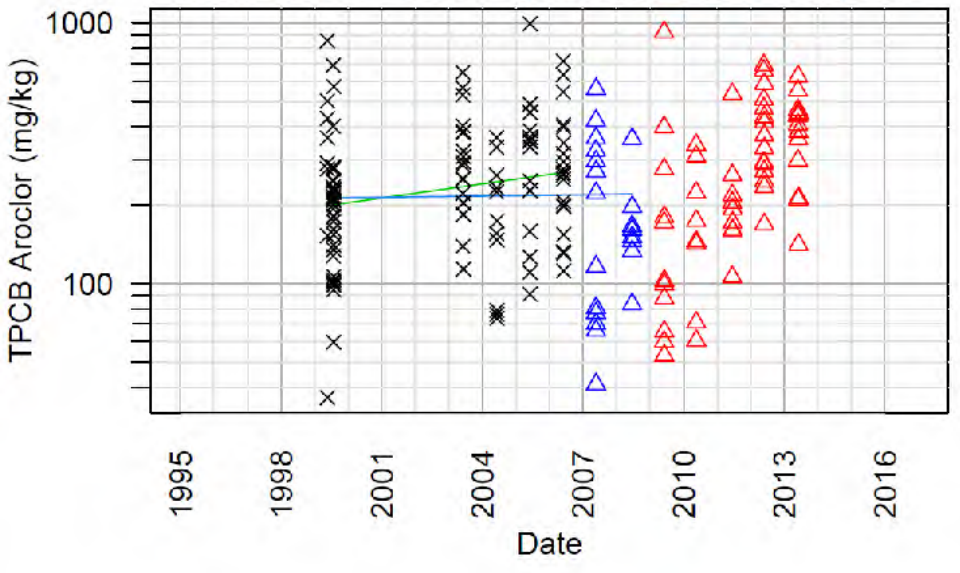
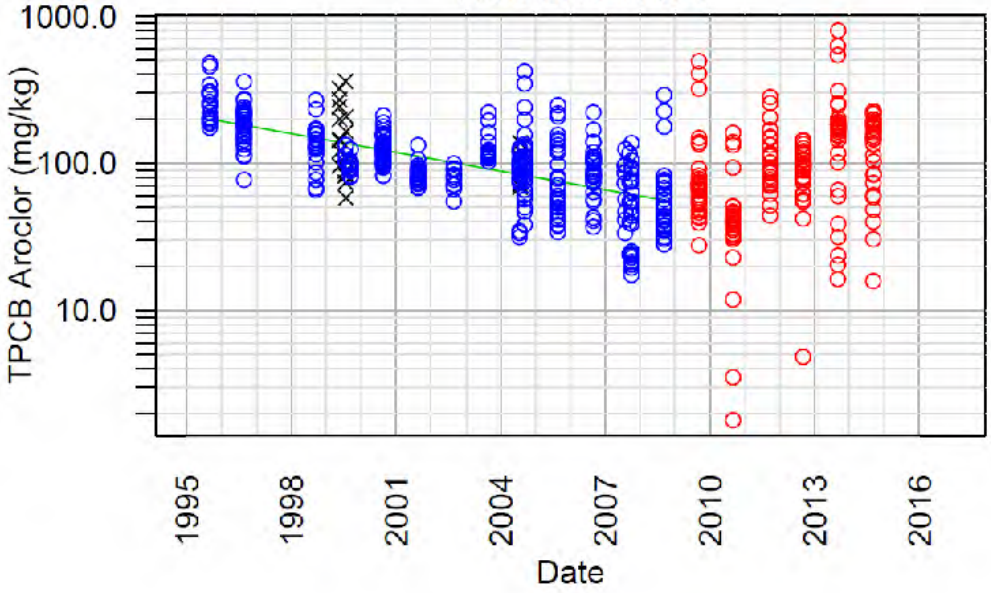


Figure 3O: River Section 3 Pumpkinseed
Coefficient: −9.761 %
Half life: 7.101



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 4A: RS 1 Largemouth Bass

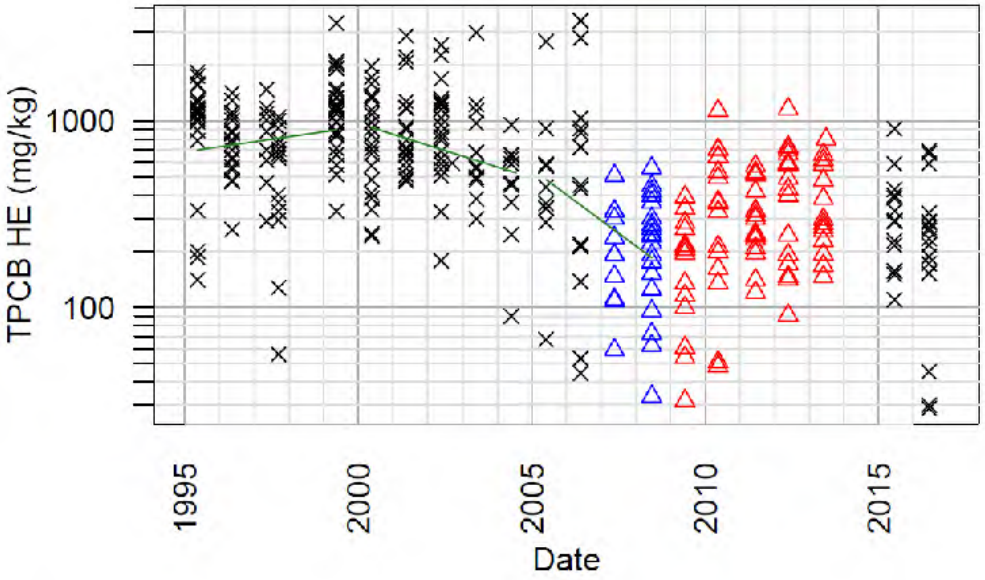


Figure 4B: RS 1 Brown Bullhead

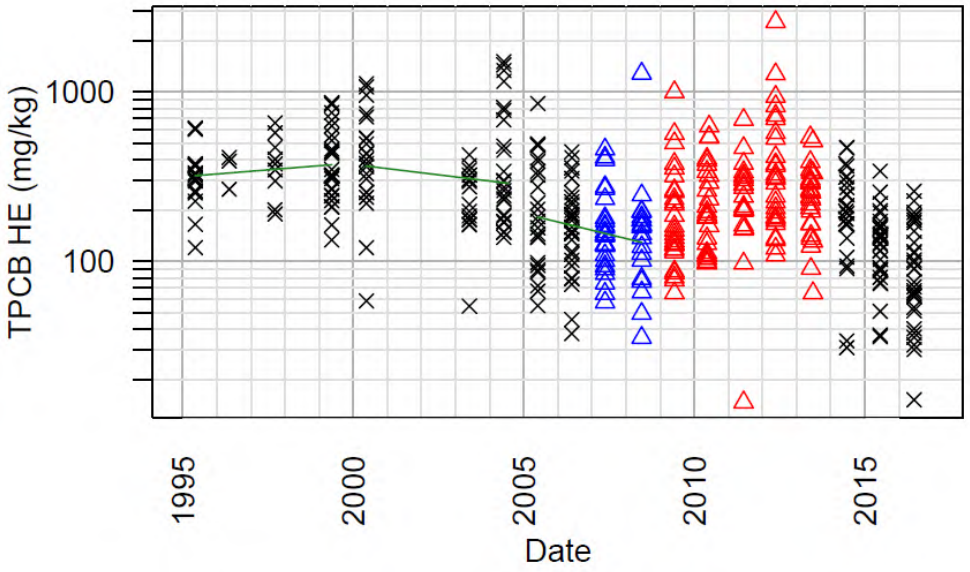


Figure 4C: RS 1 Yellow Perch

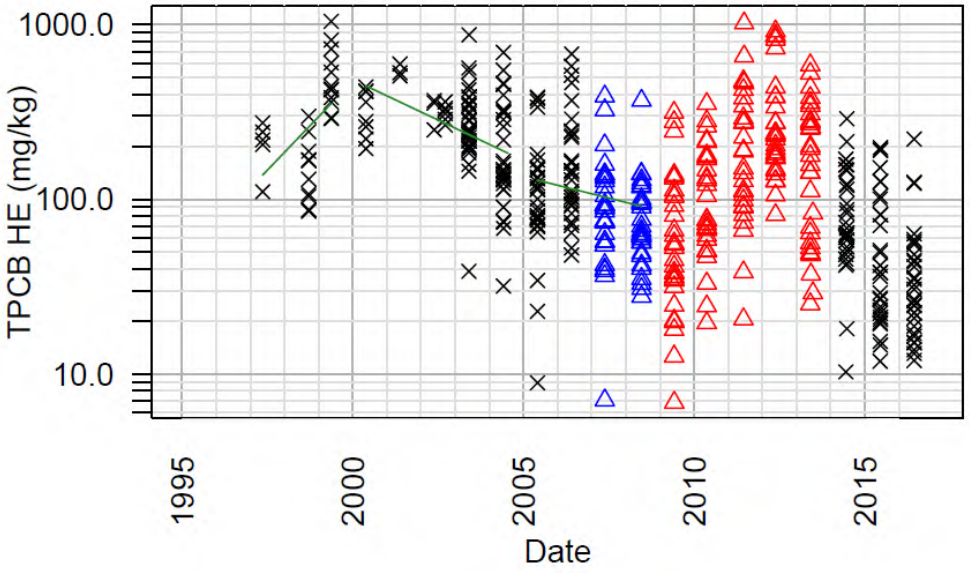
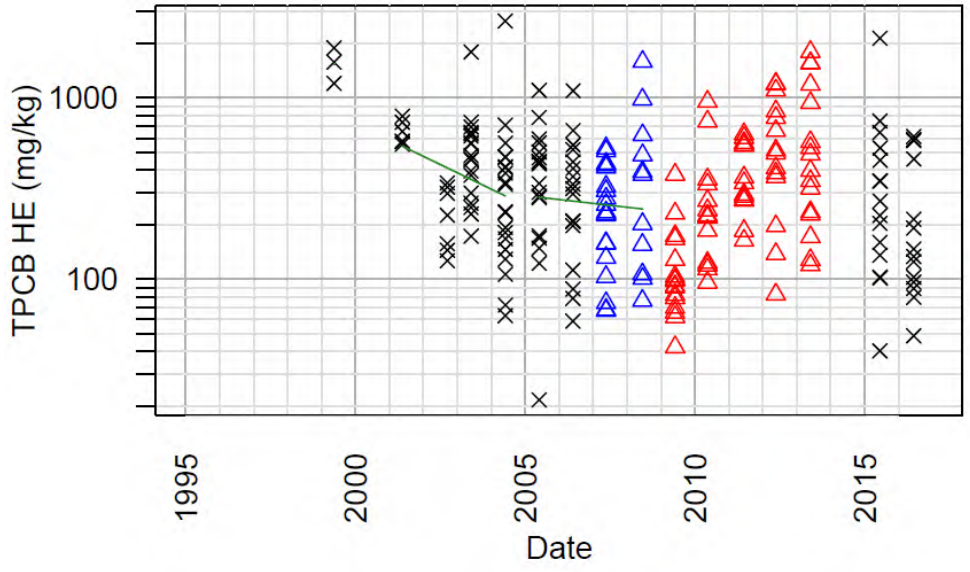


Figure 4D: RS 1 Smallmouth Bass



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging

Figure 4E: RS 1 Pumpkinseed

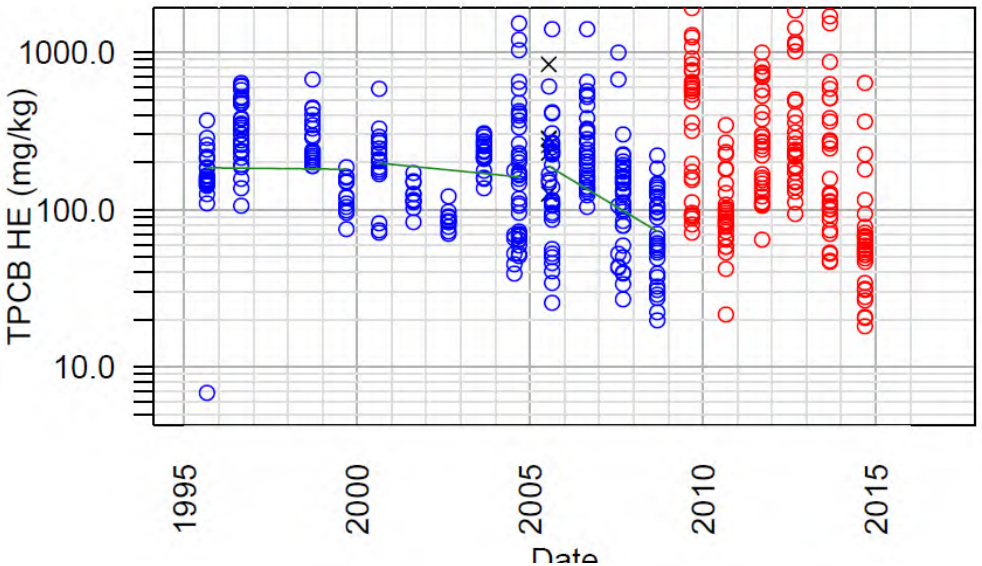


Figure 4F: RS 1 Spottail Shiner

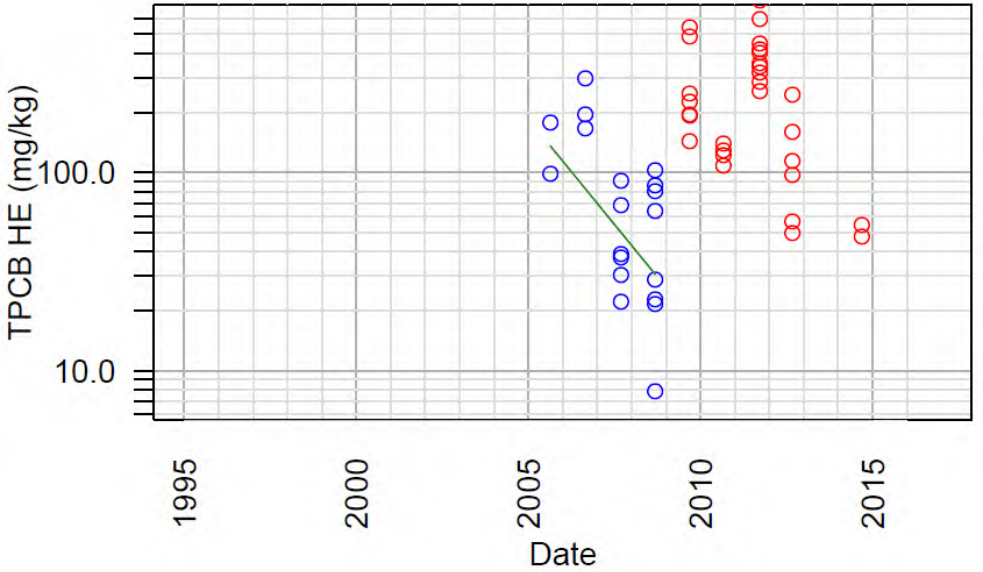


Figure 4G: RS 2 Largemouth Bass

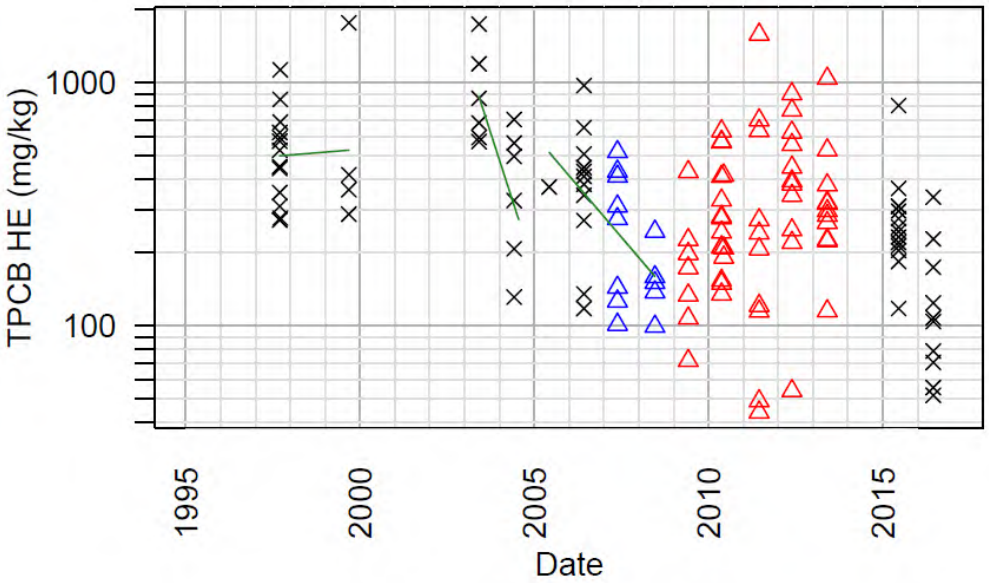
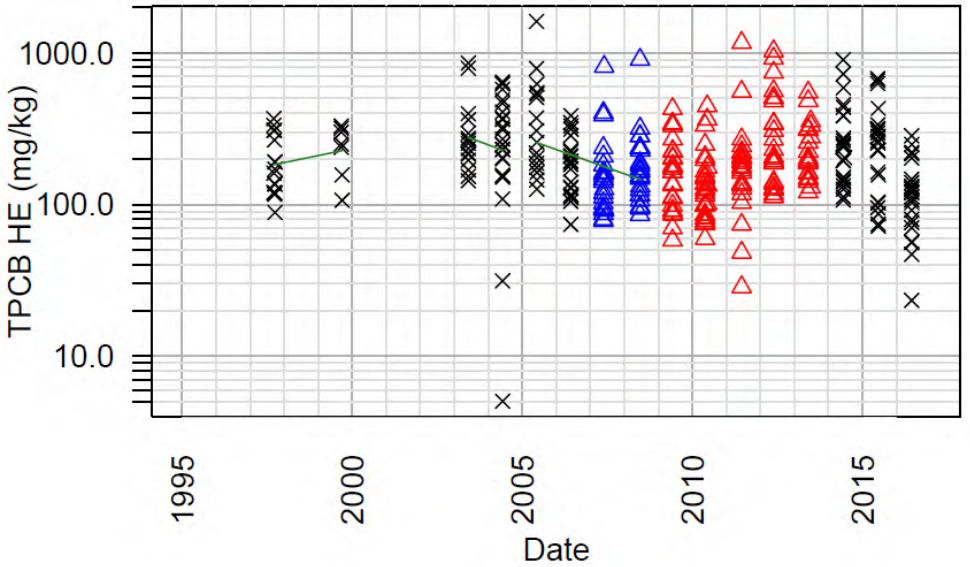


Figure 4H: RS 2 Brown Bullhead



Sample Types: Standard Rib Out: MNA Rib Out: Dredging Whole Body: MNA Whole Body: Dredging

Figure 4I: RS 2 Yellow Perch

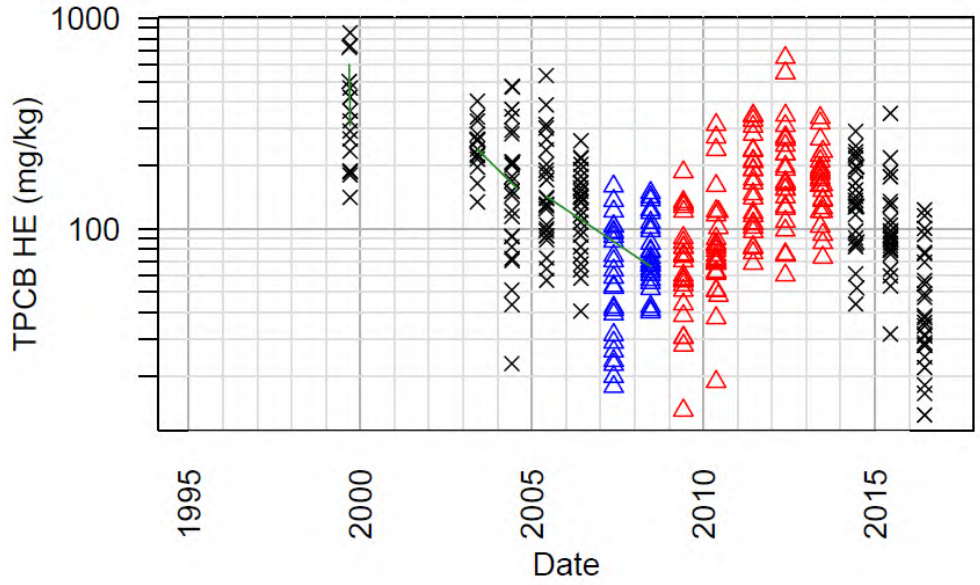


Figure 4J: RS 2 Smallmouth Bass

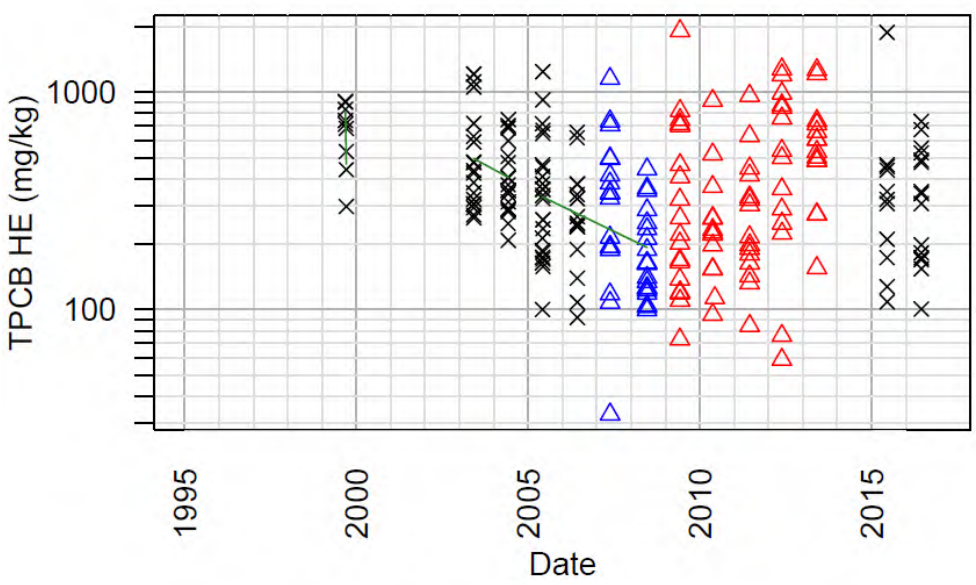


Figure 4K: RS 2 Pumpkinseed

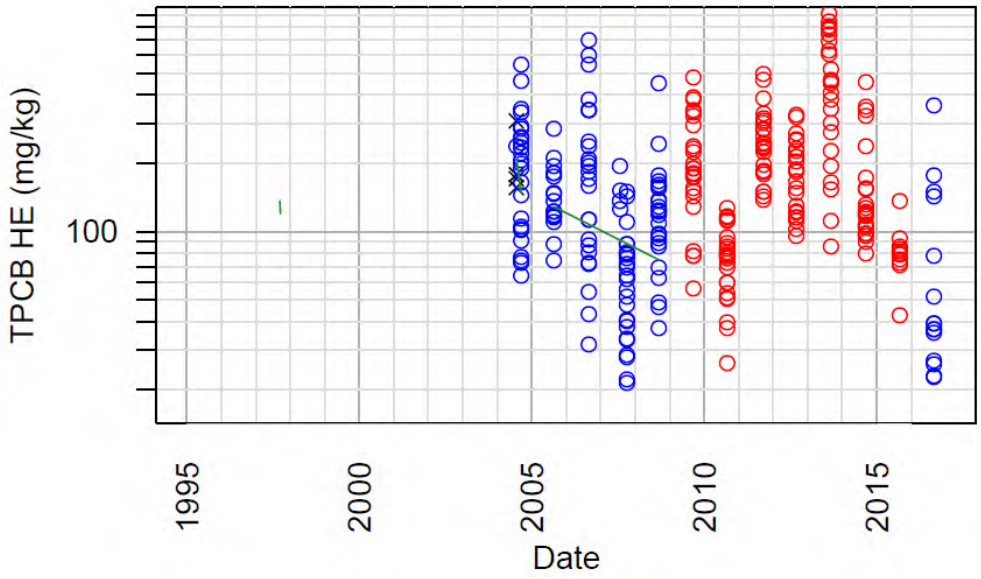
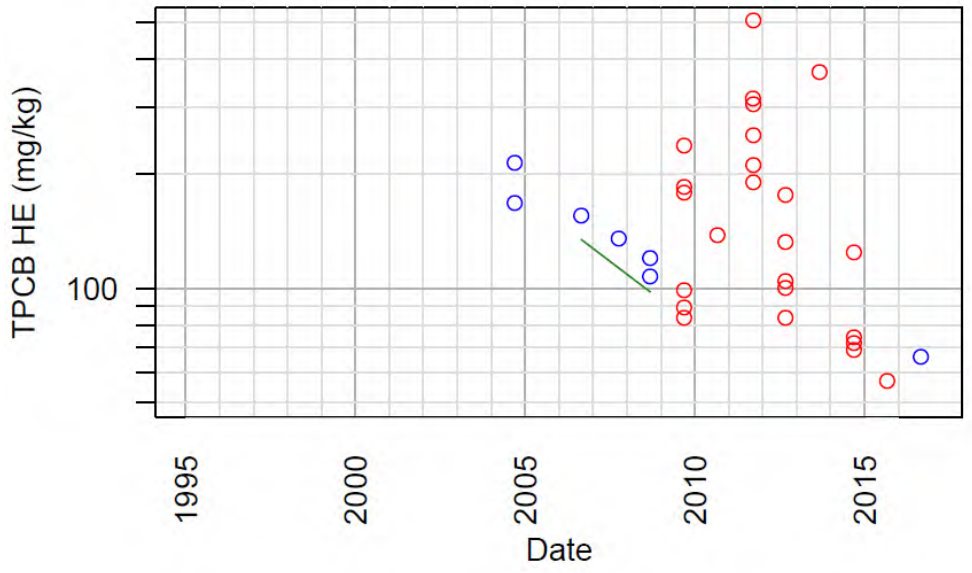


Figure 4L: RS 2 Spottail Shiner



Sample Types: Standard | Rib Out: MNA | Rib Out: Dredging | Whole Body: MNA | Whole Body: Dredging

Figure 4M: RS 3 Largemouth Bass

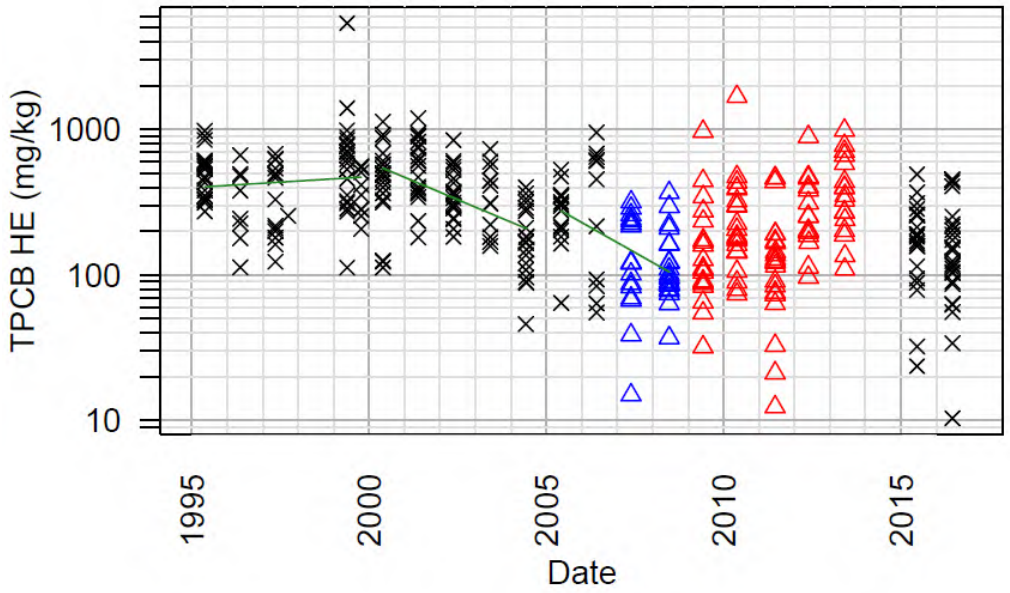


Figure 4N: RS 3 Brown Bullhead

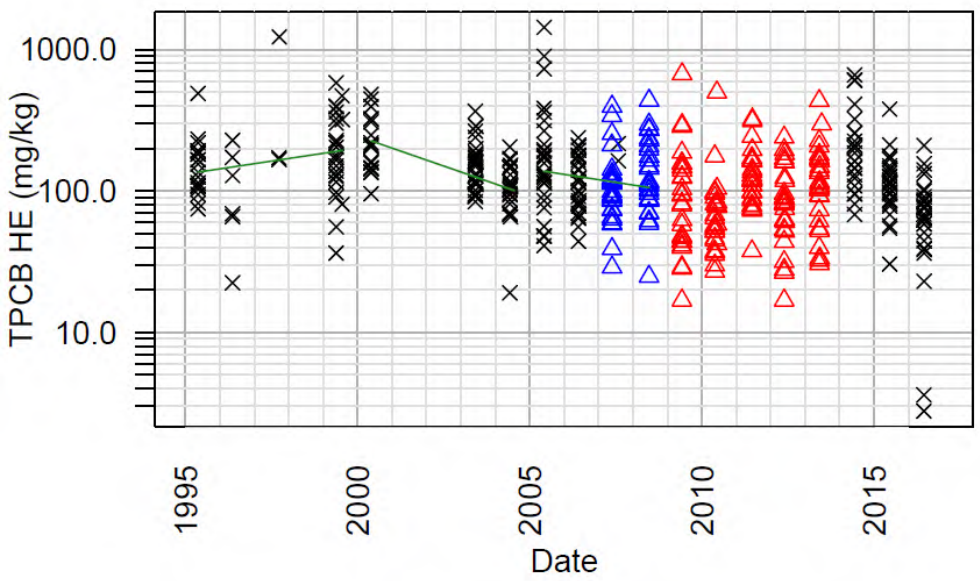


Figure 4O: RS 3 Yellow Perch

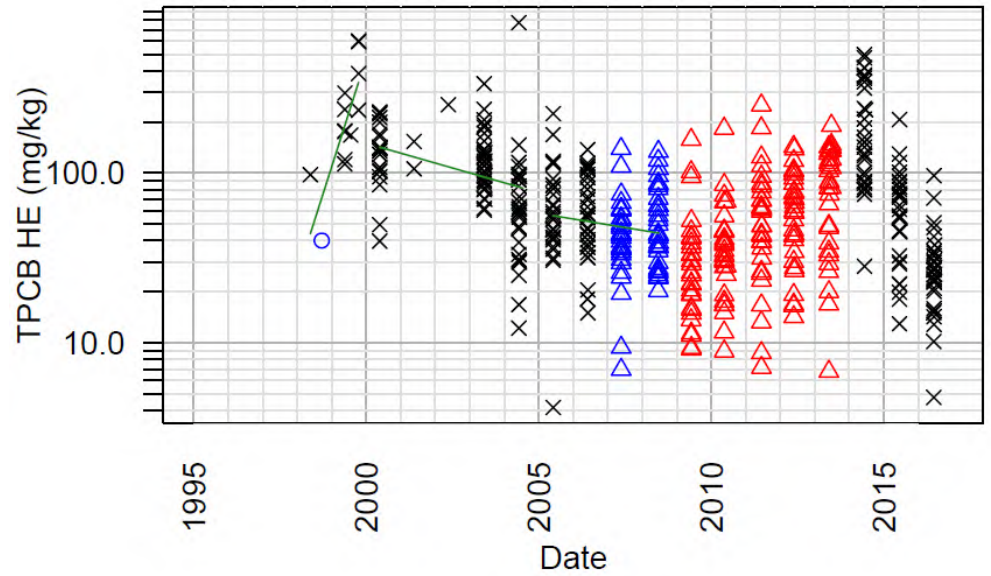
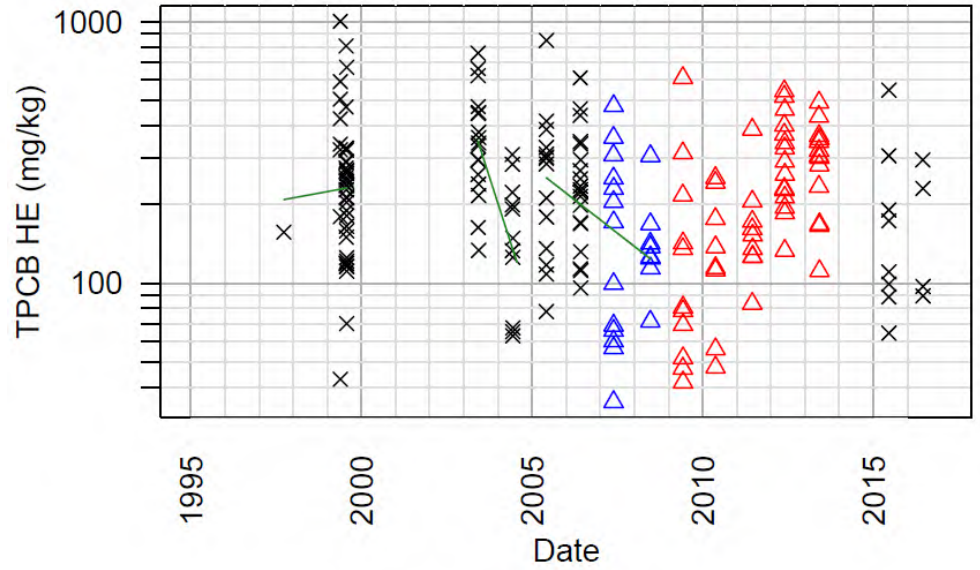


Figure 4P: RS 3 Smallmouth Bass



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging

Figure 4Q: RS 3 Pumpkinseed

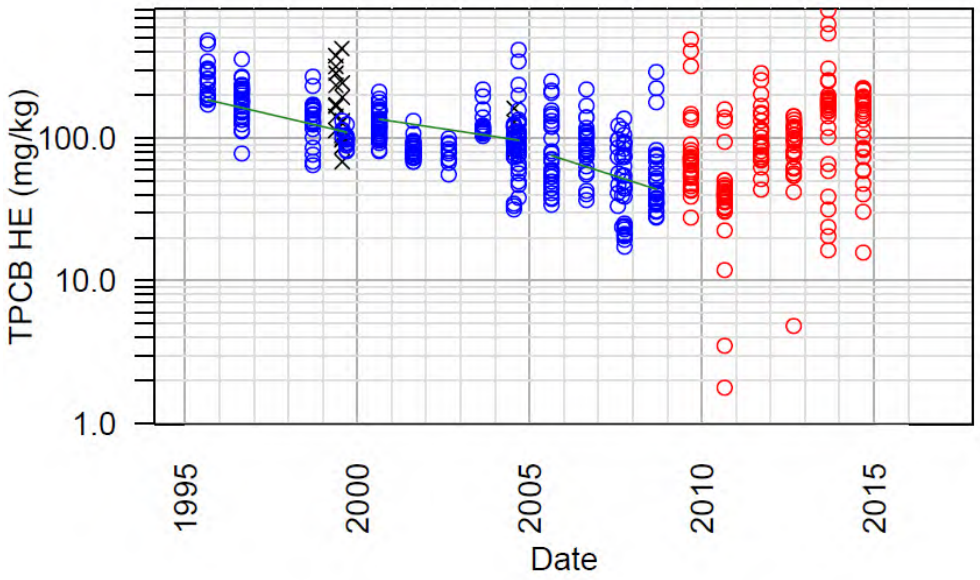
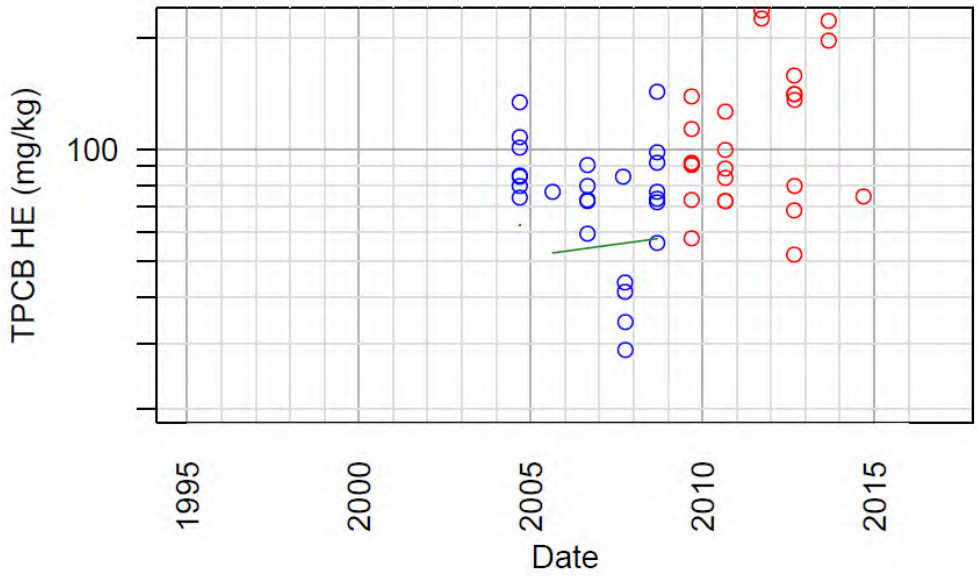


Figure 4R: RS 3 Spottail Shiner



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging

Figure 5A: Annual Mean TPCB Concentration, River Section 1 Largemouth Bass

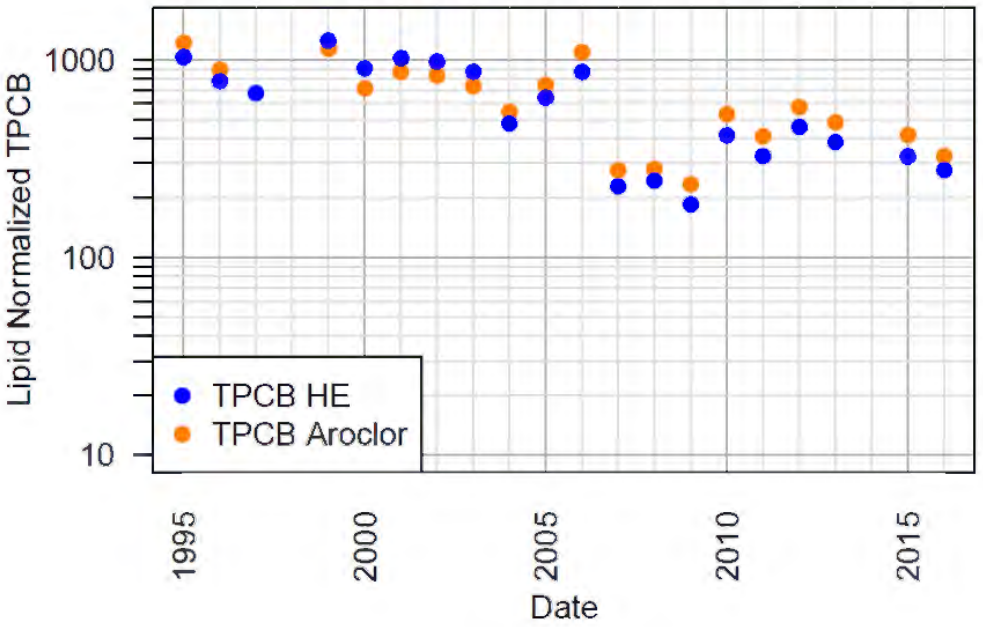


Figure 5B: Annual Mean TPCB Concentration, River Section 1 Brown Bullhead

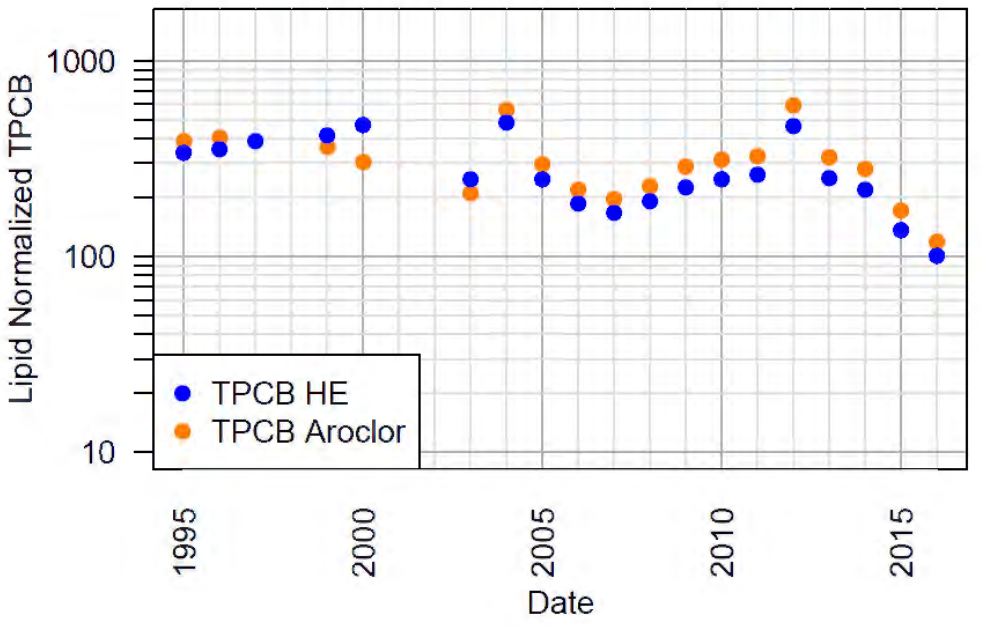


Figure 5C: Annual Mean TPCB Concentration, River Section 1 Yellow Perch

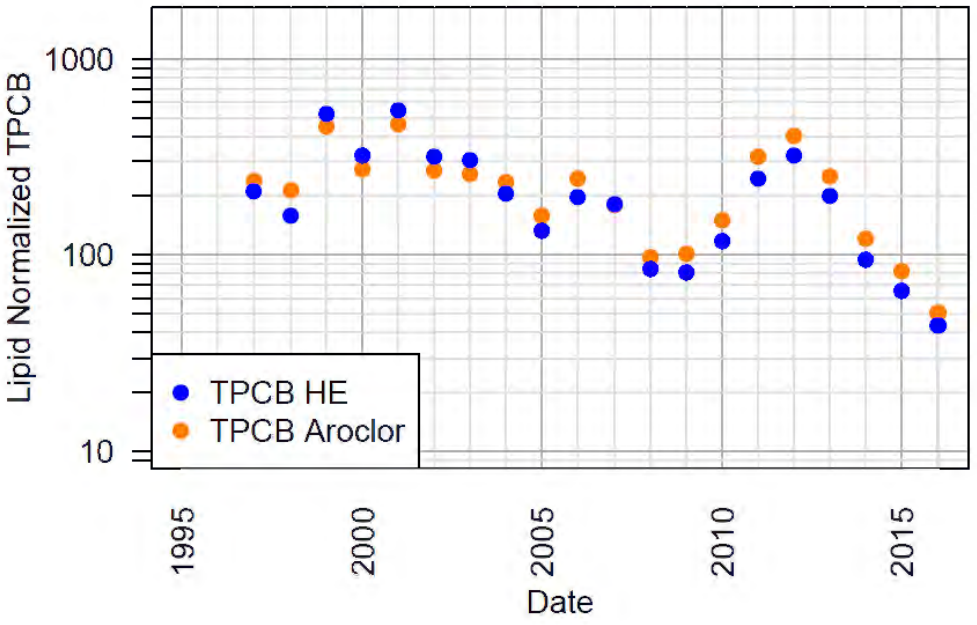


Figure 5D: Annual Mean TPCB Concentration, River Section 1 Smallmouth Bass

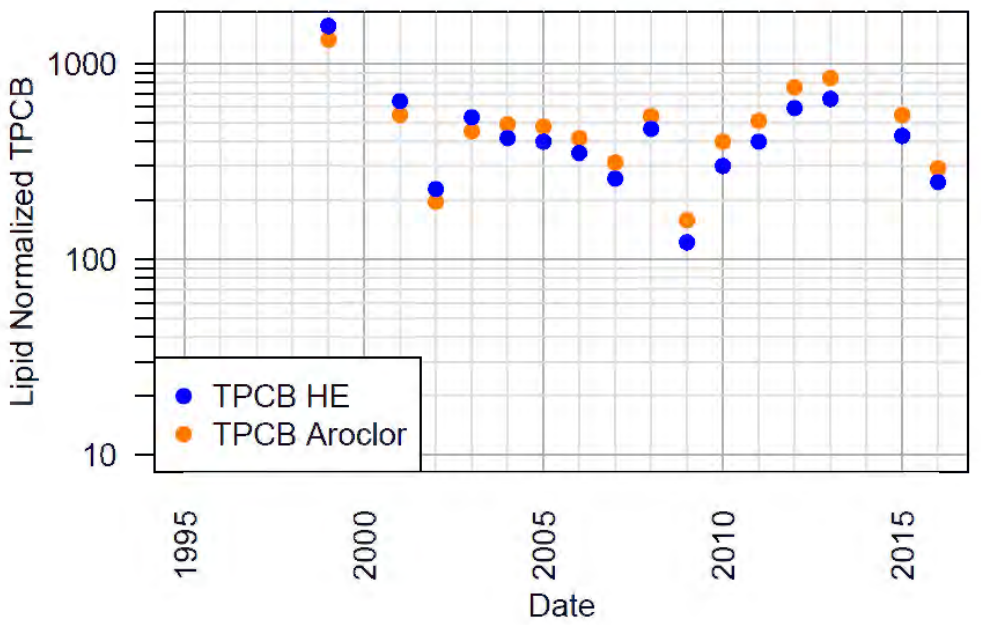


Figure 5E: Annual Mean TPCB Concentration, River Section 1 Pumpkinseed

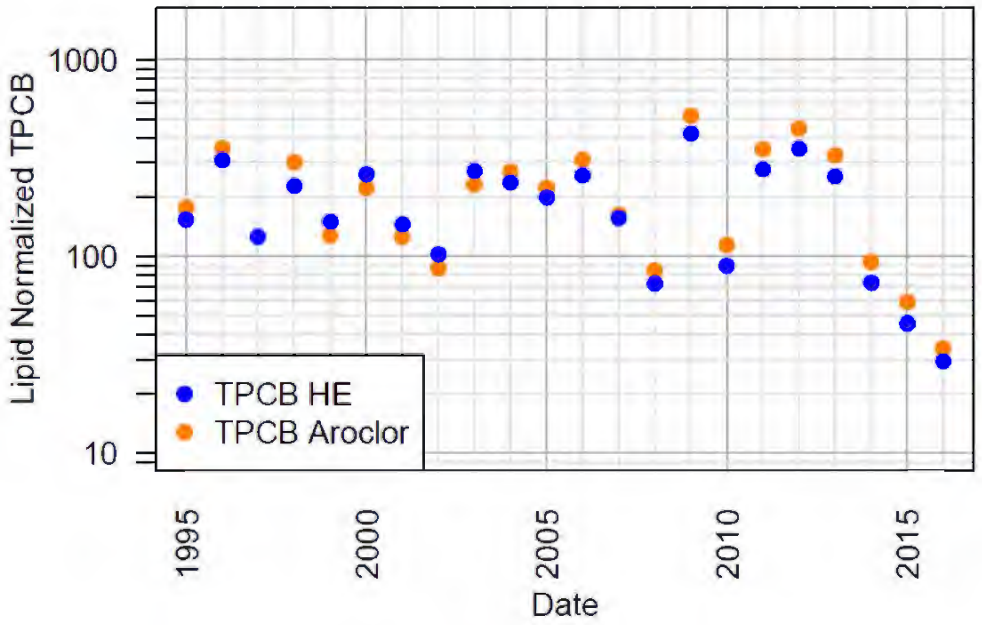


Figure 5F: Annual Mean TPCB Concentration, River Section 1 Spottail Shiner

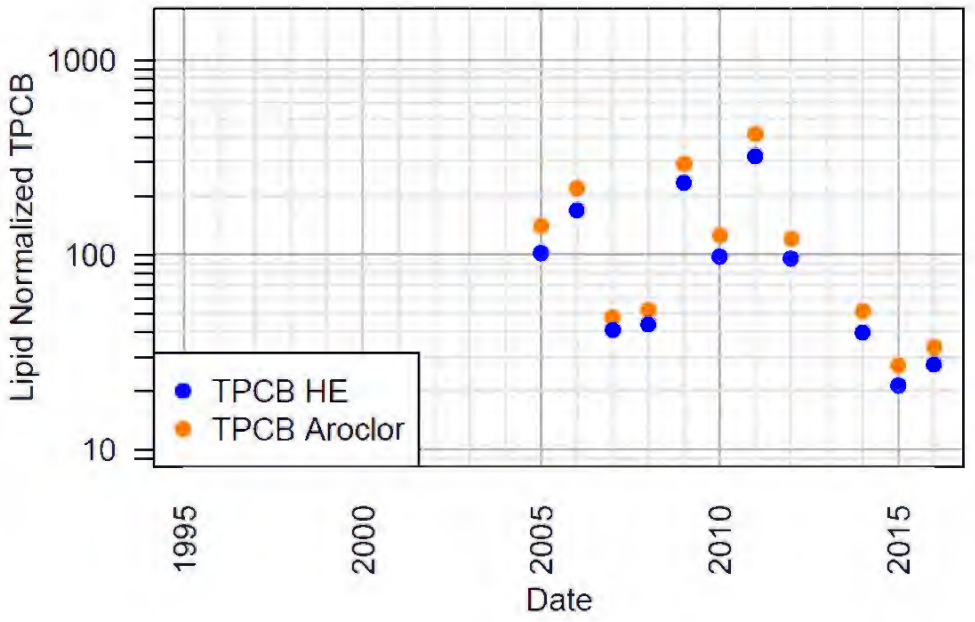


Figure 5G: Annual Mean TPCB Concentration, River Section 2 Largemouth Bass

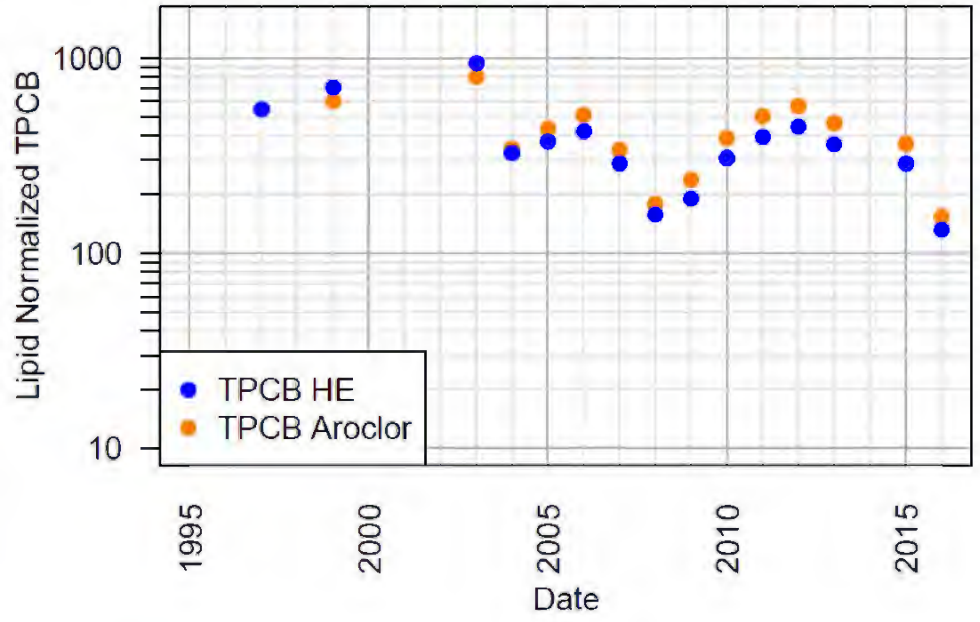


Figure 5H: Annual Mean TPCB Concentration, River Section 2 Brown Bullhead

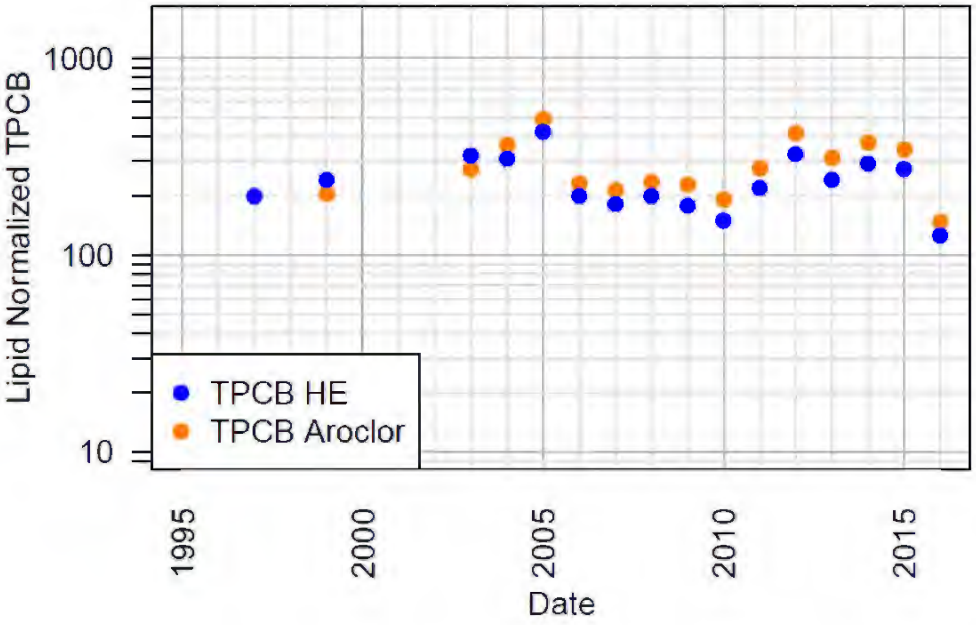


Figure 5I: Annual Mean TPCB Concentration, River Section 2 Yellow Perch

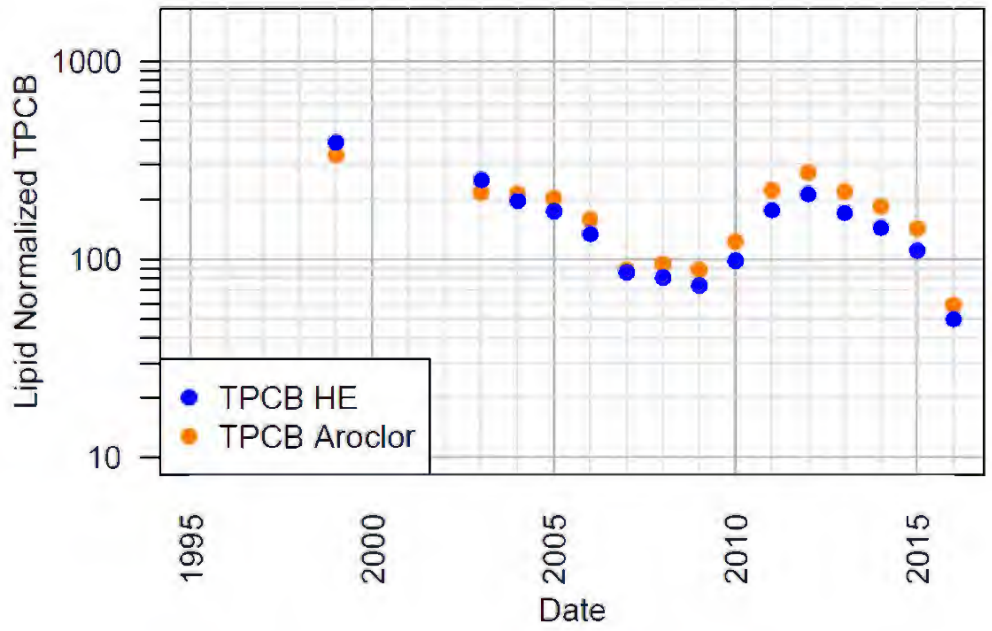


Figure 5J: Annual Mean TPCB Concentration, River Section 2 Smallmouth Bass

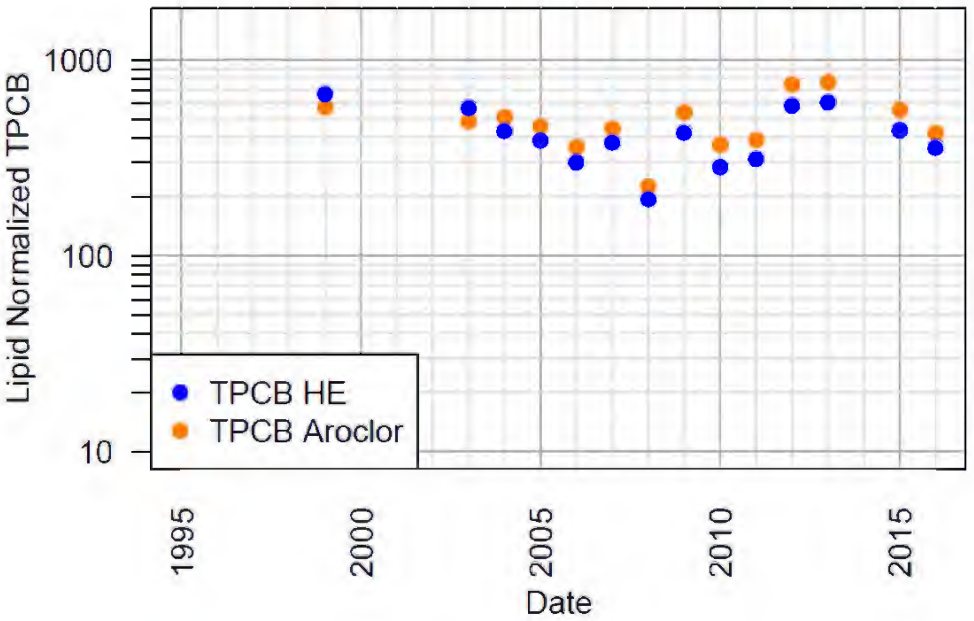


Figure 5K: Annual Mean TPCB Concentration, River Section 2 Pumpkinseed

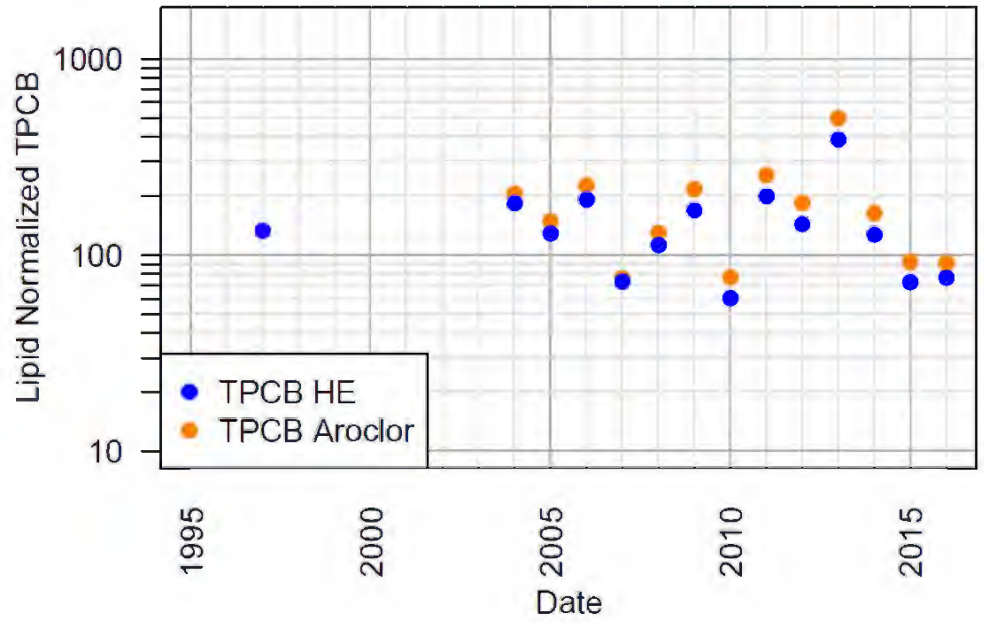


Figure 5L: Annual Mean TPCB Concentration, River Section 2 Spottail Shiner

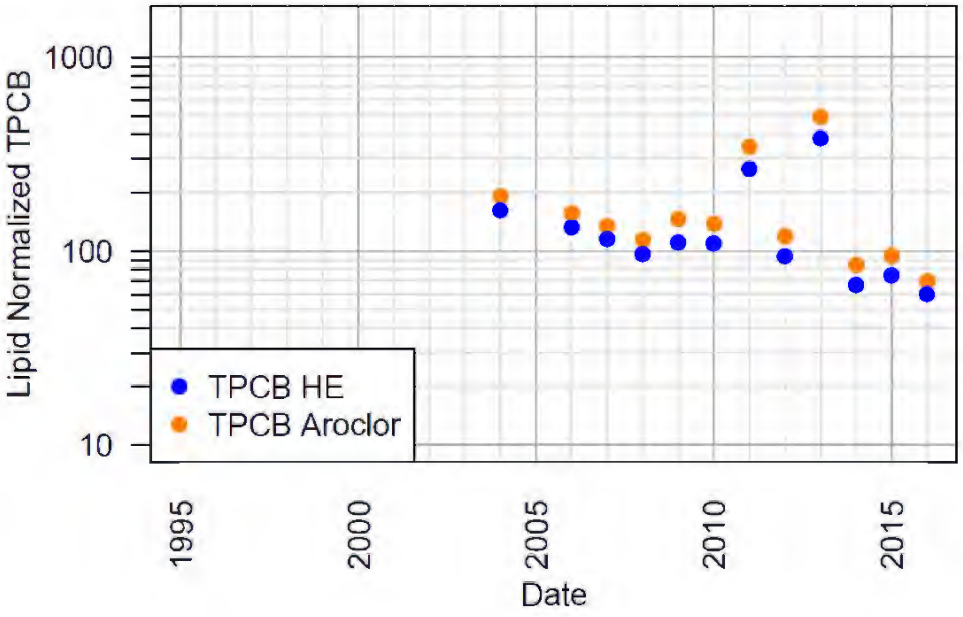


Figure 5M: Annual Mean TPCB Concentration, River Section 3 Largemouth Bass

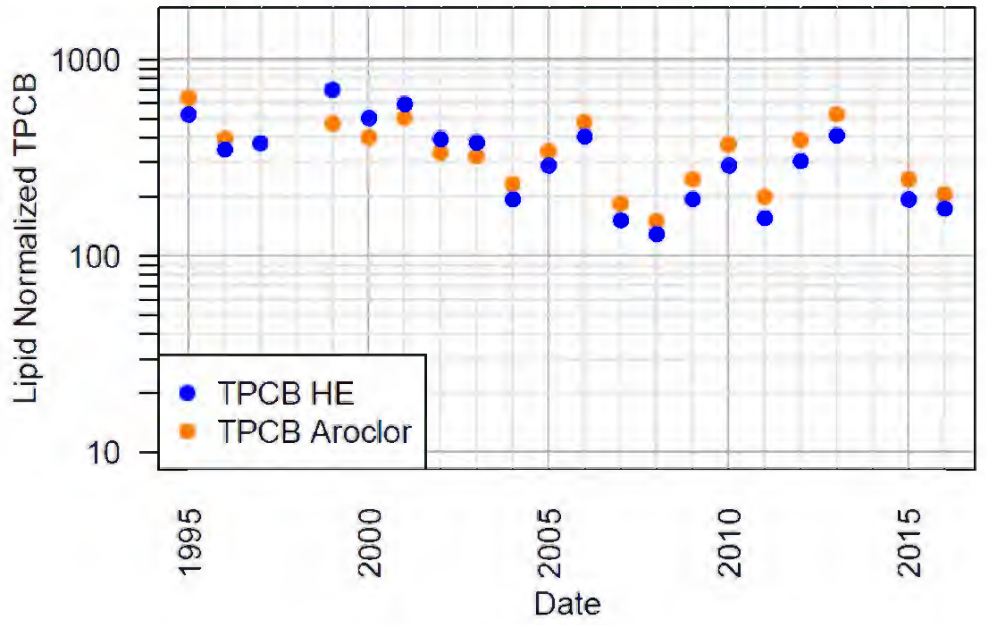


Figure 5N: Annual Mean TPCB Concentration, River Section 3 Brown Bullhead

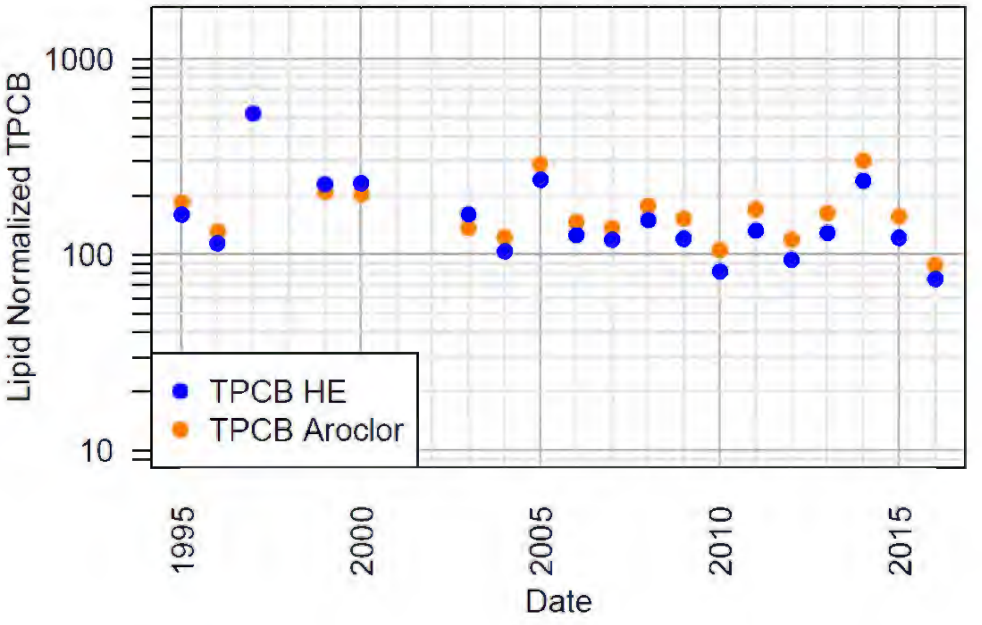


Figure 5O: Annual Mean TPCB Concentration, River Section 3 Yellow Perch

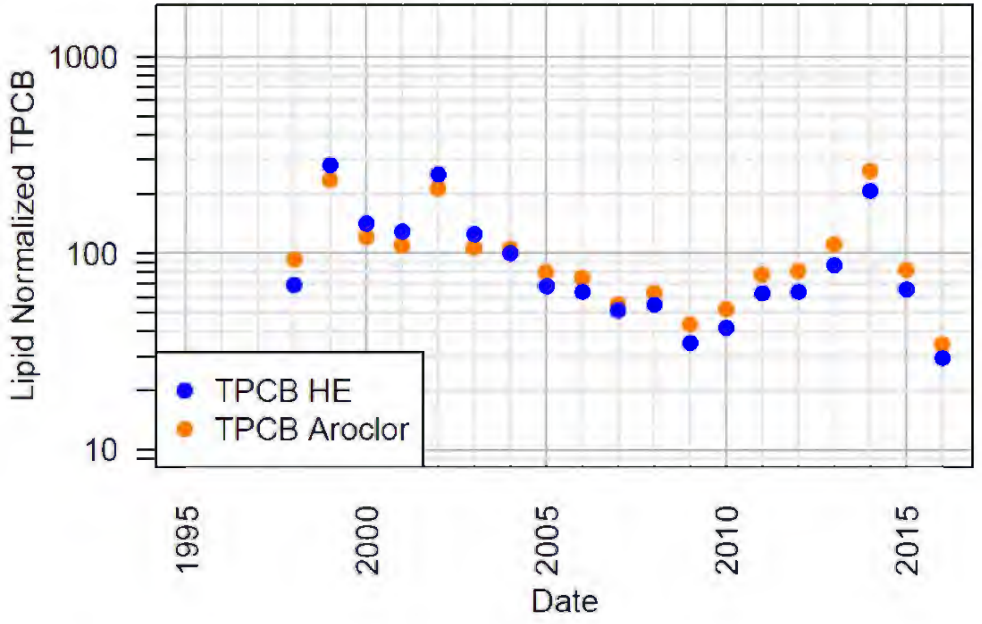


Figure 5P: Annual Mean TPCB Concentration, River Section 3 Smallmouth Bass

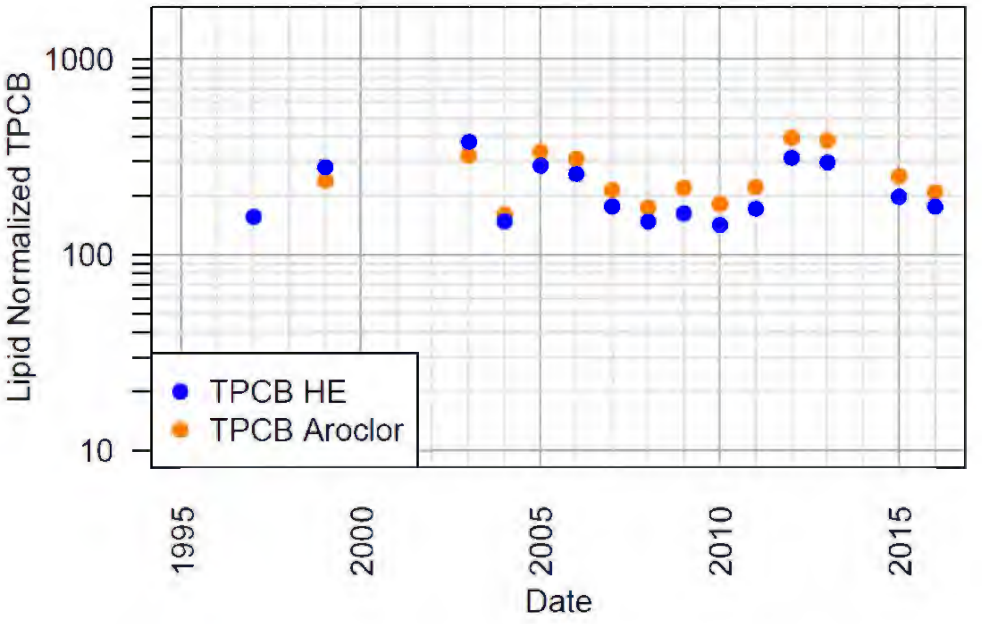


Figure 5Q: Annual Mean TPCB Concentration, River Section 3 Pumpkinseed

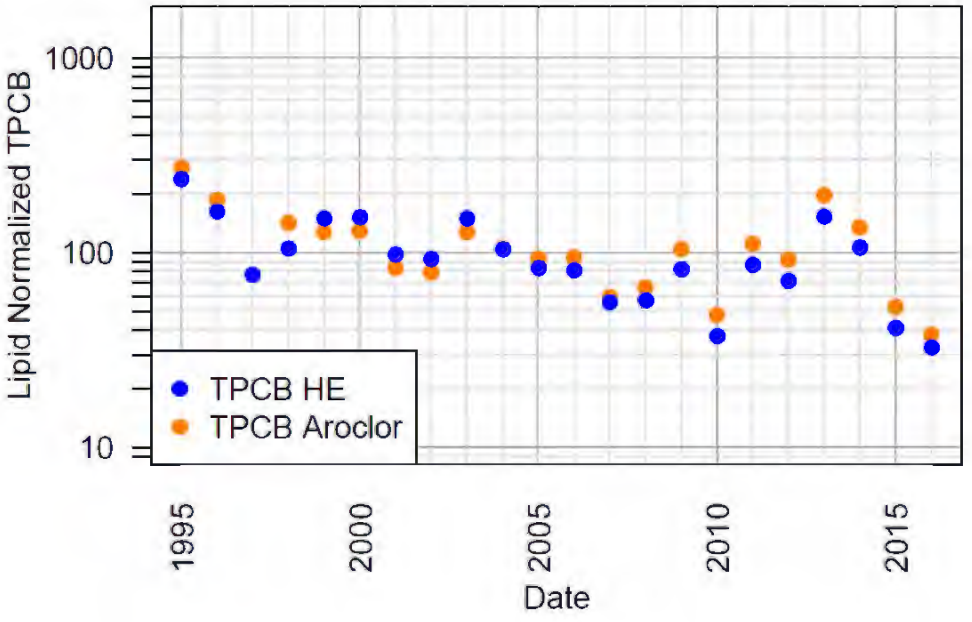
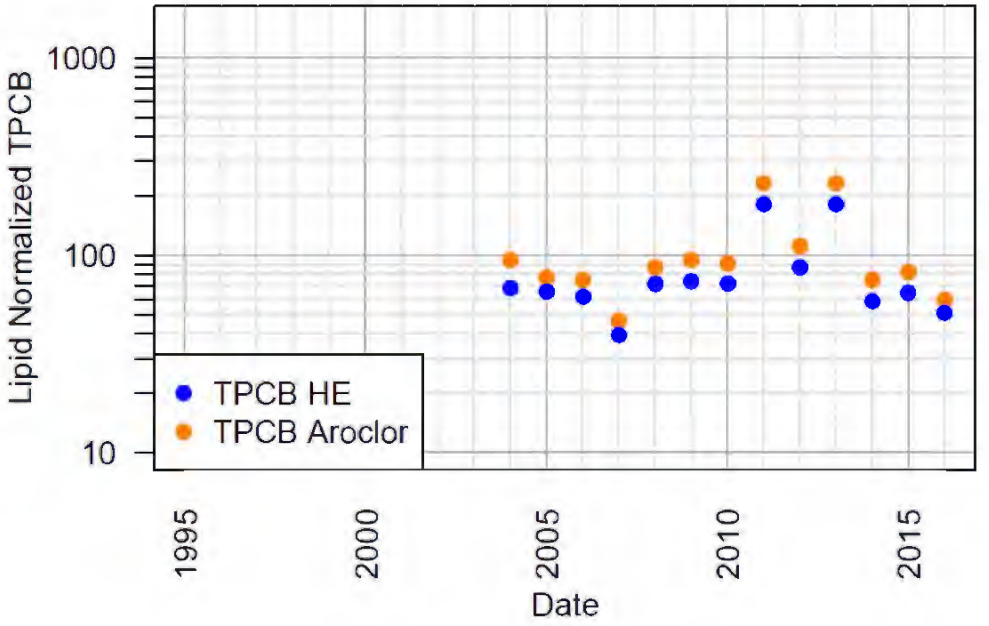


Figure 5R: Annual Mean TPCB Concentration, River Section 3 Spottail Shiner



Attachment P

NOAA Powerpoint: Re-visiting Model Projections of Lower Hudson River Fish PCB

NOAA: Re-visiting Model Projections of Lower Hudson River Fish PCB

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Re-visiting projections of PCBs in Lower Hudson River fish using model emulation

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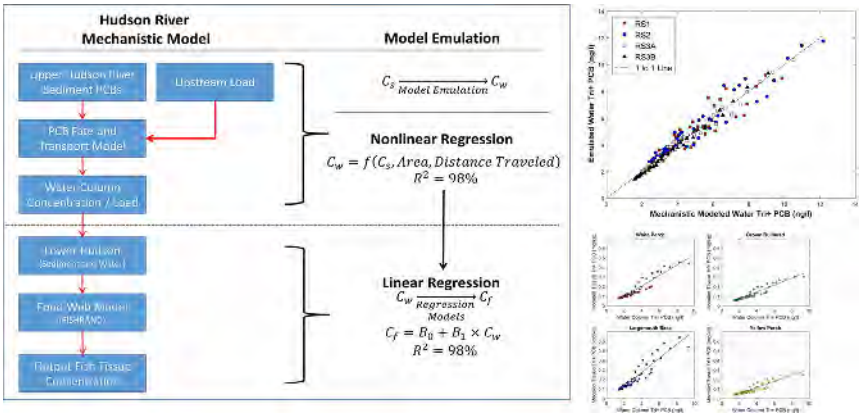
HIGHLIGHTS

- We emulated mechanistic model projections of fish PCBs in the lower Hudson River.
- Emulated models used updated sediment PCBs and recovery rate to revisit original predictions.
- Revised forecasts imply much longer time to recovery in lower Hudson River fish PCBs.
- Overestimating sediment recovery rates minimizes differences in remedial scenarios.
- Model emulation provides a mechanism to evaluate both bias and precision of models.

GRAPHICAL ABSTRACT



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ABSTRACT

Remedial decision making at large contaminated sediment sites with bioaccumulative contaminants often relies on complex mechanistic models to forecast future concentrations and compare remedial alternatives. Remedial decision-making for the Hudson River PCBs Superfund site involved predictions of future levels of PCBs in Upper Hudson River (UHR) and Lower Hudson River (LHR) fish. This study applied model emulation to evaluate the impact of updated sediment concentrations on the original mechanistic model projections of time to reach risk-based target thresholds in fish in the LHR under Monitored Natural Attenuation (MNA) and the selected dredging remedy.

The model emulation approach used a combination of nonlinear and linear regression models to estimate UHR water PCBs as a function of UHR sediment PCBs and to estimate fish concentrations in the LHR as a function of UHR water PCBs, respectively. Model emulation captured temporal changes in sediment, water, and fish PCBs predicted by the mechanistic model over the emulation period. The emulated model, using updated sediment concentrations and a revised estimate of recovery rate, matched the trend in annual monitoring data for white perch and largemouth bass in the LHR between 1997 and 2014.

Our best predictions based on the emulated model indicate that the projected time to reach fish tissue risk-based thresholds in the LHR will take decades longer than the original mechanistic model projections.

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1. Introduction

Remediation decisions at large contaminated sediment sites with bioaccumulative contaminants often rely on highly parameterized mechanistic models to make long-range temporal projections comparing natural recovery and active remedial alternatives. At the Hudson River PCBs Superfund site in New York (Fig. 1), the U.S. Environmental Protection Agency (USEPA) used mechanistic contaminant fate and transport models linked to bioaccumulation models to predict future concentrations in fish (USEPA, 2000a, 2002). Model projections of temporal changes in fish concentrations played an important role in the comparative evaluation of remedial alternatives (USEPA, 2000b).

After USEPA's Record of Decision (ROD) (USEPA, 2002), extensive remedial design sediment sampling revealed that concentrations of PCBs in surface sediments were higher and more widespread than the models had predicted (Field et al., 2009; USEPA, 2010, 2012). Additionally, USEPA observed that PCB loads from the Upper Hudson River (UHR) to the Lower Hudson River (LHR) prior to the start of dredging in 2009 were substantially greater than predicted by the models and showed little evidence of decline (USEPA, 2010). Because modeled fish tissue PCB concentrations in the LHR are a function of PCB loads from the UHR, these findings imply that time to reach target thresholds for human consumption in fish in the LHR was underestimated by the original mechanistic model projections.

In this study, we used statistical model emulation to condense relationships between inputs and outputs of USEPA's linked mechanistic models to investigate sensitivity of model predictions to this new information. Model emulation reduces complex mechanistic models into computationally-efficient equations, dramatically reducing computational demands and time and effort to recalibrate and rerun the mechanistic models, while also maintaining a relevant and consistent representation of the underlying relationships within them (Logemann et al., 2004). The model emulator developed in this study was used to estimate new outputs associated with modified and updated inputs defining a range of remedial scenarios. The model emulator was also used to evaluate the sensitivity of model predictions to variation and uncertainty in initial sediment concentrations and different rates of natural recovery of surface sediment concentrations.

2. Methods

2.1. Study area

The Hudson River PCBs Superfund site extends approximately 321 km (200 miles) downstream from two General Electric (GE) capacitor manufacturing plants adjacent to the UHR to New York Harbor (Fig. 1). USEPA's ROD in 2002 (USEPA, 2002) called for dredging and monitored natural recovery (MNA) of PCB contaminated UHR sediments extending 64 km upstream from the Federal Dam at Troy. This area was divided into three main sections, River Sections (RS) 1 (Thompson Island Pool), RS2 (Schuylerville), and RS3. Because of its overall length, RS3 was subdivided into three modeling subsections RS3A (Stillwater), RS3B (Waterford) and RS3C (Troy). USEPA did not evaluate or select a remedy for the LHR tidal estuary (245 km between the Federal Dam and the Battery in New York City).

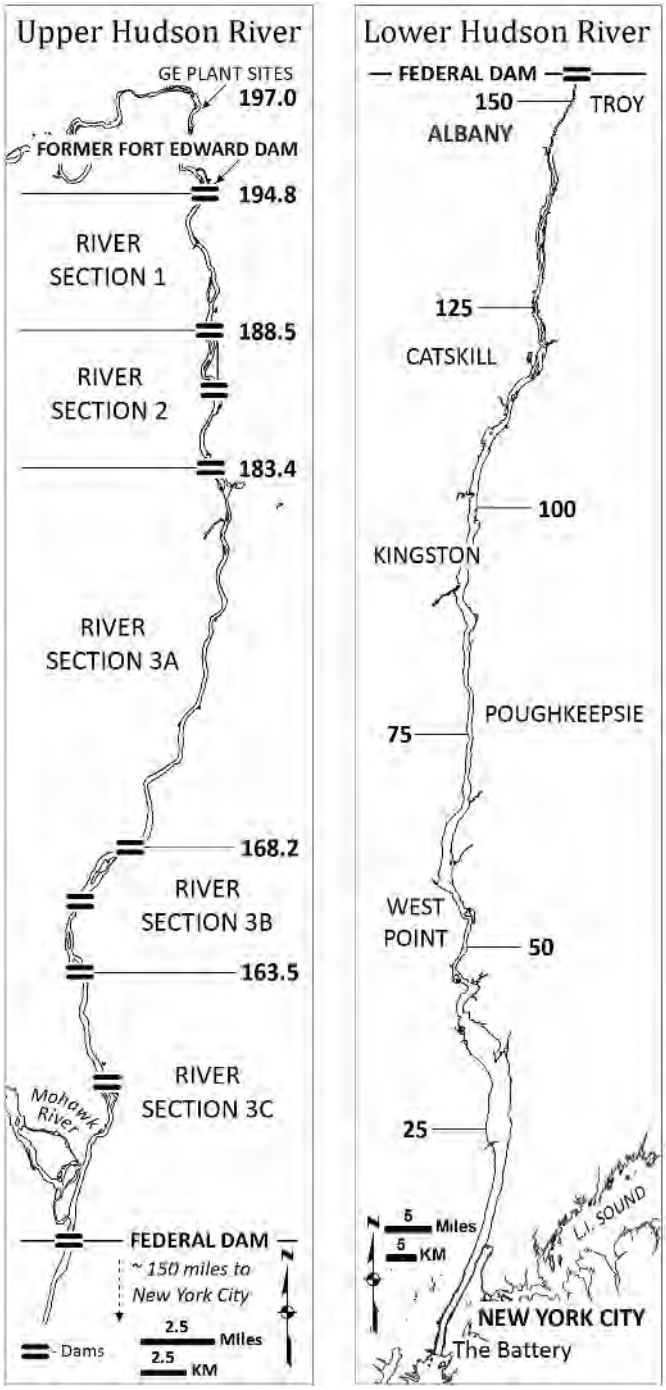

Fig. 1. Map showing the 321 km (200 mile) extent of the Hudson River PCBs Superfund site from Hudson Falls (above the GE plant sites) to The Battery in New York City. The left panel for the Upper Hudson shows the River Sections (RS) for the approximate 64 km (40 mile) remedial action area. The right panel for the Lower Hudson shows the 241 km (150 mile) tidal estuary with the fish model locations.

2.2. Sample sediment data

Sediment samples collected for PCB analysis between 1976 and 1999 by USEPA, GE and New York State were used during the Remedial Investigation and Feasibility Study (RI/FS) to assess risk and to predict future concentrations under various remedial scenarios (USEPA, 2000b, 2000c). Surface sediments were generally collected from the top 5 cm, although some penetrated as deep as 15 cm. Tri+ PCBs in water (average annual whole water concentrations), sediment and fish, the sum of trichlorobiphenyl and higher chlorinated homologues, were used for modeling because historic total PCB data did not effectively quantify mono- and di-chlorobiphenyl PCBs (USEPA, 2000a; Connolly et al., 2000). PCBs in fish tissue are primarily composed of Tri+ PCBs (USEPA, 2000a, 2002).

Subsequent to USEPA's ROD, GE collected sediment samples (mostly cores with some grab samples) from over 8000 locations throughout the UHR supporting design and implementation of the selected remedy. In RS1, most cores were collected on a triangular 24-meter (80-foot) grid from the entire pool. In RS2 and RS3, cores were collected almost exclusively within fine-grained sediments on triangular 24- or 50-

meter grids (QEA, 2002, 2005, 2007). This sampling design is considered approximately unbiased in RS1 and unbiased to fine-grained sediments in RS2 and RS3.

2.2.1. Estimated pre-dredge concentrations

We averaged surface Tri+ PCB concentrations from design sampling conducted from 2002 through 2005 representing pre-dredge surface sediment concentrations in 2003. We used these averages for initial conditions comparing updated MNA and remedial (REM) scenarios.

Most (94%) of the samples represented the top 5 cm and the remainder were from the top 15 cm or less. Average concentrations from samples including intervals up to 15 cm in depth differed inconsequentially from samples composed of the 0–5 cm interval. The USEPA mechanistic model simulated PCB fate and transport in the top 4 cm.

2.2.2. Estimated post-dredge surface sediment concentrations

Evaluating the change in surface sediment concentration following remediation required an estimate of expected post-dredging Tri+ PCB concentrations in sediment. Samples within the remedial design dredge footprints (Arcadis, 2013) were assigned a post-dredge surface sediment Tri+ PCB concentration of 0.25 mg/kg (USEPA, 2002) and arithmetic averages for each river subsection were recalculated to represent the concentration in 2003, the year USEPA expected dredging to commence.

2.2.3. Estimated surface sediment concentration decay rate

Field et al. (2009) found that the exponential temporal decrease in sediment PCBs (exponential decay rates) estimated from USEPA's mechanistic models overstated the rate of natural recovery of surface sediments. GE conducted large-scale sediment surveys throughout the UHR in 1991 (O'Brien and Gere Engineers, Inc., 1993) and in 2002 through 2005 as part of remedial design (QEA, 2005, 2007). We compared average surface concentrations from these two surveys and calculated an exponential decay rate for each river section (Table 1). The average surface sediment Tri+ PCB concentration representing 2003 in each modeled river subsection was calculated, using only samples from the top 5 cm matching the top 5 cm sampling interval collected in 1991. In RS2 and RS3, these samples from 2003 can be considered representative of cohesive sediment deposits and directly comparable to samples from the cohesive sediment transects from 1991. By necessity, decay rate estimates for RS1 were based on comparison of remedial design samples, representing both cohesive and non-cohesive sediments, with samples representing cohesive sediments collected in 1991. Because cohesive sediments tended to have higher than average Tri+ PCB concentrations, the estimated decay rate is likely to overstate the actual rate. The overall average decay rate and confidence interval (CI) was used to guide selection of model emulation scenarios.

Table 1
Average surface (top 5 cm) sediment Tri+ PCB concentration (mg/kg) in 1991 and 2003 and estimated exponential decay rate.

Model subsection	Cohesive sediment 1991[a]	Updated sediment 2003[b]	Exponential decay
1	20 (227)[c]	16.9 (3414)[c]	1.4%
2	18 (33)	14.7 (1539)	1.7%
3A	4.3 (103)	3.4 (2129)	2.0%
3B	5.7 (30)	5.6 (682)	0.1%
Average 95% confidence interval (CI)			1.3% (−0.1% to 2.6%)

[a] O'Brien and Gere Engineers, Inc. (1993).
[b] Includes cohesive and non-cohesive sediments in River Section 1 and cohesive only in River Sections 2 and 3.
[c] Number of samples.

2.3. Selected remedy

The selected remedy, initiated in 2009, included both MNA and active remediation (dredging and backfill or capping followed by MNA) in the UHR. Sediment remediation areas were defined primarily on two criteria: surface concentrations (defined by USEPA as the top 30 cm) and mass-per-unit area (MPA), a measure of PCB inventory. Remediation areas were defined as follows: for RS1, a surface concentration of 10 mg/kg Tri+ PCBs in the surface or an MPA of 3 g/m^2 Tri+ PCBs; for RS2 and RS3, a surface concentration of 30 mg/kg Tri+ PCBs or an MPA of 10 g/m^2 Tri+ PCBs. Source control near GE plant sites, approximately 3 km upstream of the modeled area, was assumed under both MNA and active remediation scenarios.

2.4. Mechanistic model framework

The mechanistic numerical models developed by USEPA predicted sediment, water and fish Tri+ PCB concentrations in the RS1, RS2, RS3A, and RS3B reaches of the UHR (USEPA, 2000a). GE also developed similar mechanistic models that were generally consistent with those developed by USEPA (QEA, 1999a). USEPA used the projections of PCB load from the UHR (RS3B) to the LHR from the Upper Hudson River Toxic Chemical Model (HUDTOX) as input to the Farley model (Farley, 1999; USEPA, 1999) to calculate sediment and water concentrations in the LHR. Output from the Farley model was then used as input to USEPA's FISHRAND model, a mechanistic food web model, to predict Tri+ PCB concentrations in four species of fish (white perch, brown bullhead, largemouth bass, and yellow perch) at four LHR locations downstream of the Federal Dam at Troy (RM152 (Albany/Troy) (river kilometer [RK] 245), RM113 (Catskill) (RK 182), RM90 (Kingston) (RK 145), and RM50 (West Point) (RK 80) USEPA, 2002). While PCB-contaminated sediment in the UHR was the primary focus for remedial alternatives, reduction in PCB load to the LHR was a major remedial action objective and was expected to result in a reduction of PCB concentrations in lower river fish. Because initial PCB concentrations in LHR fish were lower than UHR fish, model projections indicated that LHR fish would reach human health risk management objectives (thresholds) much sooner than UHR fish.

We captured mechanistic model output by digitizing Tri+ PCB time series from the USEPA mechanistic model output for MNA and the selected remedy, including sediment (USEPA, 2000b: Figures 6-24, 6-26, 6-28, and 6-30; USEPA, 2002: Figures 363150-1, 3, 5, and 7) and water (USEPA, 2002: Figures 363150-10, -11, -12, and -13) for four model subsections in the UHR and fish at four locations in the LHR (USEPA, 2002: Figures 313787-2, 3, 4, and 5). Digitizing was accomplished using Plot Digitizer, a shareware Java program used to digitize scanned plots. Digitized sediment, water, and fish Tri+ PCBs time series were interpolated to equally-spaced annual time steps so that modeled values for each media could be paired temporally. Interpolation was conducted using linear interpolation using MATLAB© software (MATLAB 8.6, Release 2015b, The MathWorks Inc., Natick, MA, 2000). These time series simulated scenarios assumed dredging would begin in 2003 or 2004 and end by 2010 for the selected remedy.

2.5. Model emulation

Digitized input and output from mechanistic model projections provided a basis for using nonlinear optimization to fit a simplified mathematical model of water concentrations (C_w) in each UHR subsection as a function of 1) original and updated sediment Tri+ PCBs (C_s), 2) upstream source input (2 ng/L or 0 ng/L), 3) area of subsection, and 4) distance from the downstream dam in each subsection (see Supplementary Fig. 1). The emulated model structure is a simplified parameter version of the USEPA mechanistic model including four one-dimensional model compartments representing each river subsection.

2.5.1. Model emulator

The model emulator represented each of the four river subsections with one model compartment composed of three terms representing PCB transfer to or from the water column: 1) upstream source minus deposition; 2) release/resuspension minus deposition of a fraction of these resuspended solids; and 3) post-dredge resuspension of disturbed residuals. The general form of the emulator within the *i*th subsection is:

$$\begin{aligned} \textit{Water Column Load}_i = &(\textit{Water Column Load}_{i-1} - \textit{Deposition}_i) \\ &+ (\textit{Resuspension}_i - \textit{Deposition}_i) + \textit{Post Dredge Resuspension}_i. \end{aligned} \quad (1)$$

Each model compartment (i.e. river subsection) represents an impounded pool within which flows are generally laminar. Deposition of PCBs from the water column to the sediment bed was assumed proportional to distance traveled within each subsection with constant deposition rate per unit distance (g_i, $i = 1,2,3,4$) within river segments.

Release/resuspension of sediment PCBs to the water column was assumed to be directly proportional to average PCB concentration and area of PCB-containing cohesive sediments per river subsection with net sediment to water transfer coefficients (γ_i; $i = 1,2,3,4$) assumed constant through time.

Post-dredging sediment residuals were assumed to be more susceptible to resuspension with sediment to water transfer coefficients (β_i; $i = 1,2,3,4$) proportional to pre-dredge PCB concentrations and area dredged. These lower density disturbed residuals were assumed to decline with time at an 8% rate as they either flushed downstream, or became more consolidated and less susceptible to erosion.

Lower Hudson River fish Tri+ PCBs (C_f) were predicted from modeled water column Tri+ PCB concentrations (C_w) from the mechanistic model output for RS3B using linear regression.

2.5.2. Emulator calibration

Net contaminant transfer coefficients were estimated by minimizing root mean squared error between temporally paired emulated and mechanistic modeled Tri+ PCB concentrations in water. The paired sediment and water time series for each of the 4 river sections spanned 30 years (2005–2034) for MNA and 25 years (2010–2034) for REM1 (the selected remedy) and each remedial scenario was modeled assuming: 1) partial source control with Tri+ PCB load decreasing from 0.16 kg/d to 0.0256 kg/d by the year 2005; and 2) complete source control, assuming upstream Tri+ PCB load would decrease from 0.16 kg/d to 0.0 kg/d (USEPA, 2000b). These 55 time steps and 4 river sections and 2 upstream load scenarios resulted in a system of 440 simultaneous nonlinear equations with 12 unknown net transfer coefficients which were solved using nonlinear optimization using MATLAB© scientific software (The MathWorks 2015). Full mathematical detail is provided in Appendix A. The estimated coefficients are summarized in Table S-1. Mechanistic water column Tri+ PCB concentrations from RS3B were treated as predictors of LHR fish Tri+ PCB concentrations and were calibrated by linear regression. Projections of LHR fish tissue Tri+ PCBs were calculated by applying this regression model to emulated water Tri+ PCB concentrations at the downstream end of RS3B.

Although we calibrated the model emulation to both upstream load scenarios, we found only small differences in future model projections of primary interest, so we focused on scenarios with average upstream source concentrations of 0.0256 kg/d (approximately 2 ng/L Tri+ PCB). This is reasonable because measured water column Tri+ PCB concentrations upstream of RS1 have been approximately 2 ng/L Tri+ PCB since 2004 (Farrar, 2011; USEPA, 2010). For the calibration step, we selected 2005 as the initial year for MNA because mechanistic model projections reached baseline concentrations of 2 ng/L Tri+ PCBs in that year. Initial year 2010 was selected for REM1 because dredging was anticipated to be completed by that time.

2.5.3. Uncertainty

Analytical statistical theory for mechanistic simulation models is generally intractable due to their complexity, so statistical inference to model predictions is often limited. In situations where computer runtime for simulation models is relatively short, statistical inference may be available through Monte Carlo simulation or Bayesian Markov Chain Monte Carlo Methods (Raftery et al., 1995; Smith, 1994; USEPA, 1994). These examples have the commonality that mechanistic model equations are relatively simple and can be run repeatedly, a necessity for both Bayesian and Monte Carlo methods. Because linked fate and transport models often require extremely long run-times (Glaser and Bridges, 2007), Monte Carlo or Bayesian simulation is not directly applicable. Model emulation provides a solution to this computational problem by providing a surrogate model that can be run repeatedly within a reasonable period of time, while maintaining essential elements of the physical processes embodied in the mechanistic model. This advancement provides a mechanism to evaluate both bias and precision of models, providing risk managers with a more complete description of the reliability of predictions.

2.5.3.1. Bias. Our primary objective was to apply model emulation deterministically to evaluate bias in modeled forecasts associated with change in initial sediment bed Tri+ PCB concentrations. Future Tri+ PCB concentrations in sediment, water, and fish tissue were estimated using updated sediment Tri+ PCB concentrations reflecting averages from comprehensive remedial design sampling. Changes in these values associated with updated estimates of temporal decay rates in sediments were also considered. Using these modified model inputs, future Tri+ PCB concentrations in LHR fish were re-calculated and compared to human health total PCB risk thresholds of 0.05 mg/kg, 0.2 mg/kg and 0.4 mg/kg, representing levels protective of fish consumers eating one meal per week, one meal per month, and one meal every two months respectively (USEPA, 2002). USEPA considered Tri+ PCB and total PCB concentrations interchangeable in fish (USEPA, 2002).

These estimates representing central tendency or best estimates updated for new sediment surface and decay rates were compared with the original mechanistic model estimates.

2.5.3.2. Precision. We also estimated precision of model forecasts using parametric Monte Carlo simulation for auto-correlated time series of sediment Tri+ PCB concentrations. Synthetic sediment time series were generated that reproduced temporal autocorrelation patterns and between river section cross correlations similar to those in original EPA mechanistic modeled sediment time series. Each sediment Tri+ PCB concentration time series was simulated from a lognormal distribution with mean concentration

$$C_i(t) = C_{0i} e^{-kt + \varepsilon_i(t)}$$

where C_{0i} is the initial sediment Tri+ PCB concentration in the *i*th subsection, and *k* is the PCB concentration decay rate. Because the sediment decay rate was estimated from just two points in time (1991 and 2003), we viewed this as a relatively uncertain parameter and as such investigated a relatively wide range of plausible decay rates uniformly distributed on the interval from 0.02 to 0.05. The residual time series $\varepsilon_i(t)$ was simulated as a normally distributed mean zero correlated random variable with autocorrelation and variance estimated from the residuals of an exponential fit to the mechanistic model time series. [The mathematical details of this probability model are summarized in Appendix B.]

This Monte Carlo simulation procedure involved four steps; 1) simulating four normally distributed auto-correlated sediment time series ($\varepsilon_i(t)$, $i = 1,2,3,4$), 2) randomly selecting a uniformly distributed decay coefficient between 0.02 and 0.05, 3) calculating $C_i(t)$ and 4) applying the model emulator, to these four sediment time series, producing four corresponding Tri+ PCB time series for water and finally a synthetic fish tissue Tri+ PCB time series. These four steps were

repeated 1000 times, and the fish Tri+ PCB time series were plotted, and the time to reach risk thresholds was calculated for each of the 1000 synthetic time series.

2.6. Remedial scenarios evaluated

Model emulation was used to evaluate the following remedial scenarios: (1) Mechanistic model projections for sediment PCB concentrations under Monitored Natural Attenuation (MNA1) and the selected remedy (REM1); (2) MNA (MNA2) and the selected remedy with updated sediment PCBs (REM2); and (3) An alternative remedial scenario (REM3), not considered in the ROD, that applies the RS1 cleanup target levels to RS2 and RS3 with updated sediment PCBs. For each of these scenarios, we applied both the original (8%) and the updated (3%) rate of exponential decrease in surface sediment PCBs.

3. Results

3.1. Model emulator

3.1.1. UHR sediment to water

Fitting a set of nonlinear and linear regression models using inputs and outputs from the original mechanistic models provided a computationally simple means to reproduce the USEPA water column model Tri+ PCB results under MNA and selected remedy scenarios. The mechanistic model developed by USEPA predicted sediment and water Tri+ PCB concentrations in RS1, RS2, RS3A and RS3B that were used to compare remedial alternatives.

The four-compartment nonlinear model emulator with twelve parameters linking PCB transfer from sediment to water explained 98% ($R^2 = 0.98$) of the variation in mechanistic modeled water column concentration over the 30 year projection for MNA and the 25 year projection for REM1 (Fig. 2). This demonstrates that the model emulator successfully captures the changes in sediment and water concentrations predicted by the mechanistic model for MNA and for the selected remedy in the UHR model sections over the emulation period.

3.1.2. UHR water to LHR fish

The mechanistic model predicted Tri+ PCB concentrations in four species of fish (white perch, brown bullhead, largemouth bass, and yellow perch) at four locations in the LHR (USEPA, 2002). Fish tissue Tri+ PCB concentrations in the LHR below the Federal Dam (RM152) had a strong linear relationship to water column Tri+ PCB at Waterford (RS3B) in the UHR for all four modeled species ($R^2 \geq 0.90$, Fig. 3). This linear relationship between water Tri+ PCB at RS3B and LHR fish concentrations in the mechanistic model output provided the basis for the model emulation of fish PCBs.

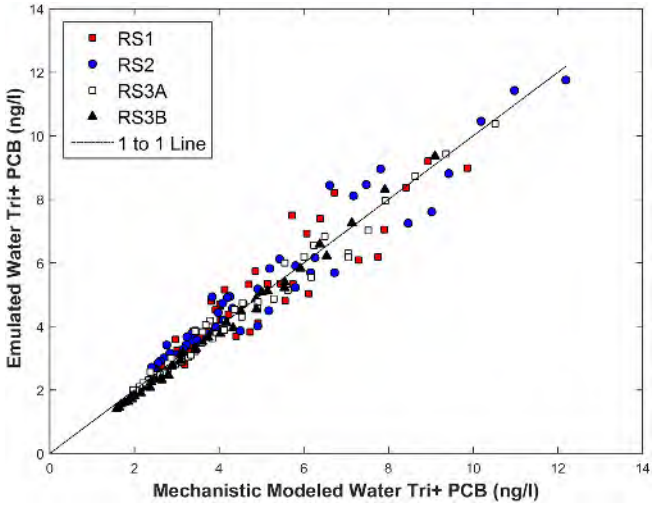

Fig. 2. Emulated vs original mechanistic model projected Tri+ PCB (ng/l) water concentrations by river subsections on the Upper Hudson River for MNA and the selected remedy.

Modeled fish tissue Tri+ PCBs for all four species at the other three LHR locations (RM113, RM90 and RM50) were also strongly linearly related to Tri+ PCB concentrations at Waterford, showing that the mechanistic model linking water to fish was effectively linear [Supplementary Table S-1 lists the regression coefficients and standard errors for white perch, brown bullhead, largemouth bass, and yellow perch at all four LHR locations].

Mechanistic food web model predictions of fish tissue concentrations for all four species at RM152 are strongly linearly related ($R^2 > 0.99$; Supplementary Fig. 2). Largemouth bass are predicted to have higher PCB concentrations than white perch, while brown bullhead and yellow perch are predicted to have lower concentrations.

Mechanistic model projections of white perch Tri+ PCB concentrations at RM113, 90, and 50 are also proportional to white perch Tri+ PCB concentrations at RM152 ($R^2 > 0.96$) and decrease with distance from the Federal Dam (Supplementary Fig. 3). The other three species had similar proportional relationships (not shown).

Emulation equations, with estimated coefficients, were applied to new model inputs such as new average PCB concentrations and decay rates in sediment (see Table A.2 for nonlinear regression coefficients).

The model emulation combined the nonlinear regression model between sediment and water with these linear regressions linking fish tissue and water column Tri+ PCBs to predict fish tissue Tri+ PCBs in the LHR from sediment Tri+ PCB concentrations in the four upper river sections. A comparison between the mechanistic model projections of Tri+ PCBs for all four species at RM152 and the emulation results are shown in Fig. 4 ($R^2 = 0.92$). Emulated model concentrations for largemouth bass and white perch tended to underestimate the mechanistic model at the higher concentrations (early in the time period).

3.1.3. Updated surface sediment concentrations

The average Tri+ PCB concentration in sediment samples from the top 5 cm in 2003, exceeded the upper bound of the mechanistic model predictions (representing the top 4 cm) under MNA (MNA1) and were more than twice the mean concentration predicted for cohesive sediments in all four model subsections of the UHR (Table 2; Fig. 5). The GE mechanistic model for RS1 similarly understated average measured sediment PCBs in 2003 (QEA, 1999a).

The projected Tri+ PCBs concentrations in surface sediment under USEPA's natural recovery scenarios declined with an approximate 8% annualized exponential decay rate (USEPA, 2000a). Using the cohesive sediment data from the 1991 transect survey and the sediment data collected in 2003, we estimated the decay rate over the twelve year period to be 2% or lower in all four model sections (Table 1) with an average decay rate of 1.3% (95% CI = −0.1% to 2.6%). The 3% rate selected for simulated scenarios was a round number representing a reasonable upper bound for calculated decay rates shown in Table 1.

Dredging was expected to begin in 2003 and require 6 years to complete (USEPA, 2000b). In the emulation, we treated 2010 as the first post-dredging year. We assumed that natural recovery would continue outside the dredging footprint while dredging occurred. To estimate surface sediment concentrations in the initial post-dredging year, needed for simulating post-dredging scenarios, exponential decay rates of 8% and 3% were applied to the average surface concentration estimated from pre-design sampling in 2003. Post-dredging river-subsection averages were then calculated accounting for reduced concentrations due to dredging and backfilling (Table 2).

The post-dredging surface Tri+ PCB concentrations estimated for 2010 were also considerably higher than predicted by the USEPA models. In RS2 and RS3, where the target cleanup levels were at least a factor of 3 higher than for RS1, estimated post-dredging surface

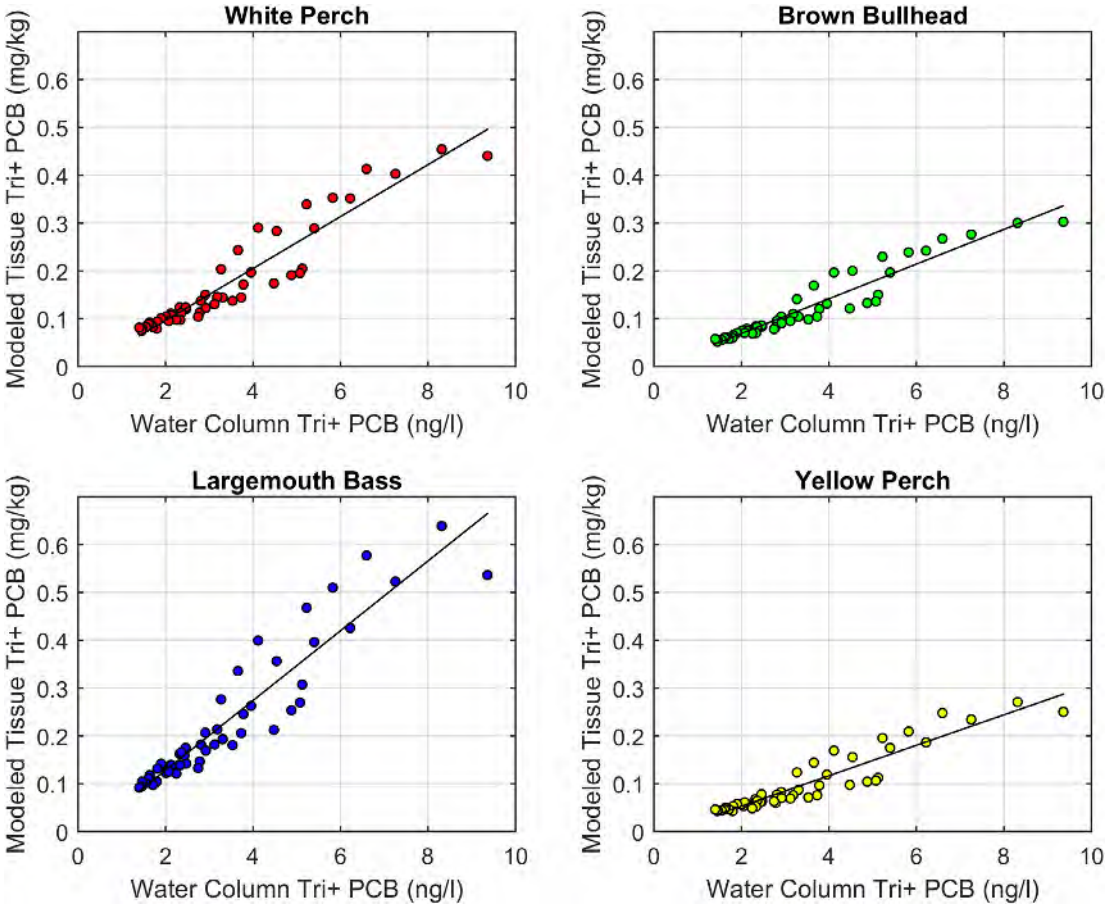

Fig. 3. Mechanistic model Tri+ PCB water concentrations (ng/l) at Waterford (RS3B) vs tissue concentrations (mg/kg) for white perch, brown bullhead, largemouth bass, and yellow perch from RM152 for MNA and the selected remedy.

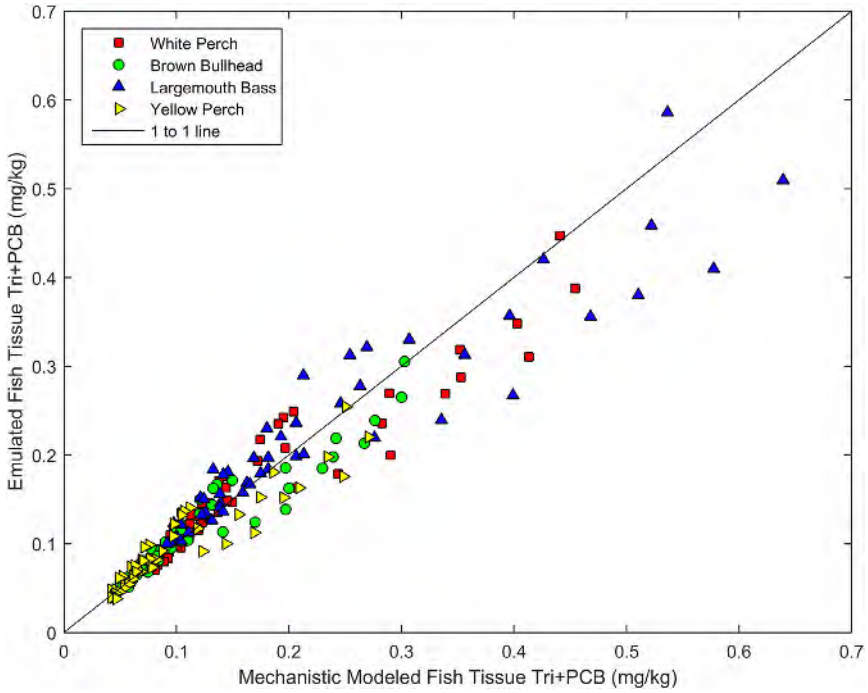

Fig. 4. Emulated vs original mechanistic model projected Tri+ PCB (mg/kg) fish concentrations for white perch, brown bullhead, largemouth bass, and yellow perch from RM152 for MNA and the selected remedy.

Table 2
Average Tri+ PCB concentrations (mg/kg) in surface sediment by river subsection under different remedial scenarios and rate of exponential decay in concentration between 2003 and 2010.

River subsection	Reach	Remedial scenario					
		MNA1[a]	MNA2[b]	REM1[c]	REM2[d]	REM2[e]	REM3[f]
	Year	2003	2003	2010	2010	2010	2010
	Decay				8%	3%	3%
RS1	Thompson Island Pool	8.5	16.9	0.5	0.8	1.1	1.1
RS2	Schuylerville	6.5	14.7	1.0	2.8	3.9	1.0
RS3A	Stillwater	1.3	3.7	0.5	1.4	2.0	1.0
RS3B	Waterford	1.0	6.0	0.4	1.9	2.7	0.9

[a] MNA1: Mechanistic model predictions for Monitored Natural Attenuation for sediment concentrations in 2003.
[b] MNA2: Measured sediment concentrations in 2003 based on updated data.
[c] REM1: Mechanistic model predictions for the selected remedy for sediment concentrations post-remediation (2010).
[d] REM2: Estimated concentrations for the selected remedy post-remediation (2010) based on updated data, assuming 8% exponential decay since 2003.
[e] REM2: Estimated concentrations for selected remedy post-remediation (2010) based on updated data, assuming 3% exponential decay since 2003.
[f] REM3: Estimated post-remediation (2010) concentrations for hypothetical remedial scenario that applies RS1 cleanup levels to RS2 and RS3, based on updated data and assuming 3% exponential decay since 2003.

concentrations, based on updated data, are about 5 times higher than previously predicted based on the mechanistic model.

3.1.4. Emulated models with updated surface sediment concentrations pre- and post-removal

The effect of a lower natural recovery rate (3%) in sediment was also evaluated in combination with updated sediment surface Tri+ PCBs concentration. This updated decay rate is more consistent with the observed changes in surface concentrations during the 12 year period between the 1991 transect survey and the remedial design data collected in 2003, while not being overly conservative with respect to anticipated decay rates. The mechanistic model profile using USEPA's original projections of sediment concentrations under MNA (MNA1) and the selected remedy (REM1) was compared to the emulated model projections using an exponential decay rate of 8%. The computed exponential decay function closely matches the original model projections (Fig. 6), supporting the use of an exponential decay model for emulated results representing other decay rates (e.g., 3%) for surface sediment concentrations under MNA2, REM2 and REM3.

The emulated models projected LHR fish Tri+ PCBs using updated surface sediment concentrations (i.e., based on the 2003 pre-design sampling) as input. Estimates of pre- and post-removal surface sediment concentrations derived from the extensive remedial design sediment dataset (Table 2) provided more accurate characterization of surface Tri+ PCB concentrations prior to initiation of remediation.

Fig. 7 illustrates the difference between USEPA's original scenarios (MNA1 and REM1 with 8% decay rates) and updated scenarios (MNA2, REM2 and REM3 with updated sediment and 3% decay rates) for Tri+ PCB concentrations in white perch at RM152. The emulated LHR fish Tri+ PCB concentrations (MNA2, REM2, REM3) were substantively higher than USEPA's original mechanistic model predictions for MNA1 and REM1 and remain elevated over a much longer period. The updated sediment surface and decay rates for MNA2, REM2, and REM3 provide greater discrimination between remedial alternatives than in the evaluation of remedial alternatives prior to remedy selection.

The model emulator was used to estimate the number of years necessary to reach USEPA risk thresholds in white perch at RM152 under original modeled scenarios (MNA1, REM1) with the number of years to reach thresholds based on updated scenarios (MNA2, REM2, REM3) using two sediment exponential decay rates: 8% (mechanistic model) and 3% (upper bound of empirical estimate). Fig. 8 displays the number of years predicted to attain the 0.4 and 0.2 mg/kg Tri+ PCB thresholds for white perch at RM152 under remedial scenarios REM1, REM2 and REM3, each with 3% and 8% exponential decay rates. For all scenarios, using the updated sediment concentrations the time for fish tissue Tri+ PCB concentrations to reach remedial action objectives of 0.4 and 0.2 mg/kg is estimated to be substantively longer than originally predicted. For the original selected remedy (REM1) under either 8% or 3% decay assumptions, white perch at RM152 were projected to reach the 0.4 mg/kg threshold before or immediately after dredging was completed. With updated sediment concentrations (REM2) and 3% decay, white perch at RM152 were estimated to reach 0.2 mg/kg more than six decades longer than the original mechanistic model projections. The REM3 scenario greatly reduced the time to thresholds compared to REM2, but still longer than the original model predictions (REM1) [see Supplementary Tables S-2 and S-3 for time to 0.2 and 0.4 mg/kg thresholds for all scenarios, species, and locations].

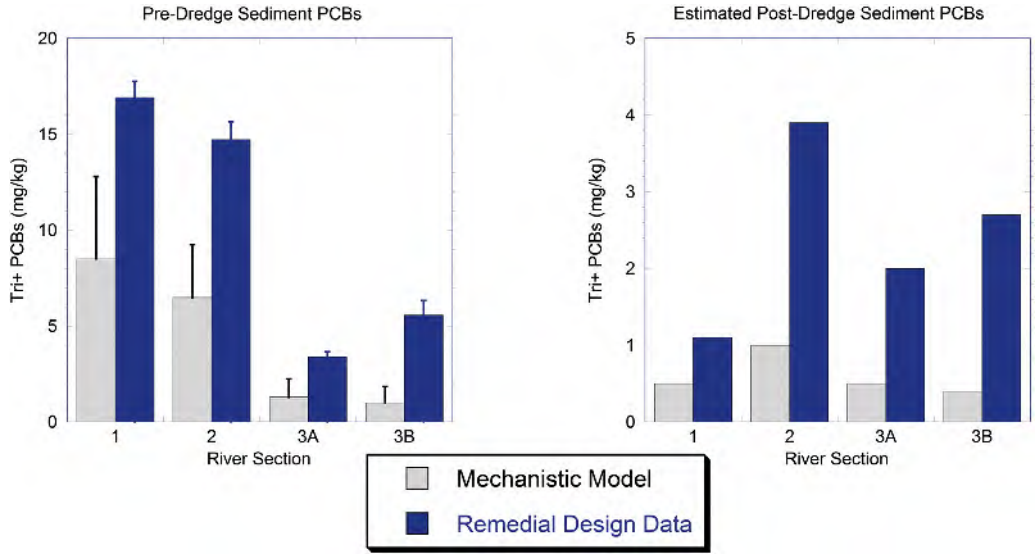

Fig. 5. Mechanistic model predictions of average and upper bound (error bars) surface sediment (top 4 cm) Tri+ PCB concentrations for 2003 pre-dredging (left panel) and post-dredge concentrations (right panel) compared to estimated river subsection average pre- and post-dredge sediment (top 5 cm) concentrations from remedial design sampling between 2002 and 2005 (approximately 2003).

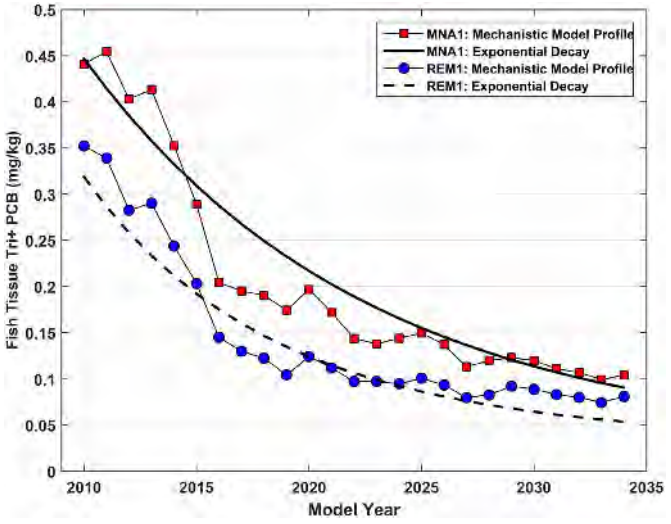

Fig. 6. Emulated model projections for white perch Tri+ PCB concentrations (mg/kg) from RM152 under MNA (MNA1) and the selected remedy (REM1) comparing the original mechanistic model (square and circle) results with simulated exponential decay rate of 8% (solid and dashed line).

3.2. Precision

Precision of emulator-based Tri+ PCB concentration in fish tissue was estimated using Monte Carlo simulation of equally likely sediment time-series with a range of decay rates (2% to 5%) and with statistical properties matching original mechanistic model sediment time series. The emulator was applied to these time-series, propagating uncertainty in sediment Tri+ PCB concentrations through to corresponding uncertainty in output Tri+ PCB concentrations in white perch at RM152. Fig. 9 shows the Monte Carlo distribution of future trajectories of fish tissue Tri+ PCB concentration, illustrating the uncertainty in estimates of the number of years needed to reach risk thresholds. The estimated number of years to thresholds were estimated to be 27 (95% CI: 19, 43), 49 (95% CI: 35, 77) and 102 (95% CI: 73, 162) for the 0.4 mg/kg, 0.2 mg/kg and 0.05 mg/kg risk based thresholds respectively.

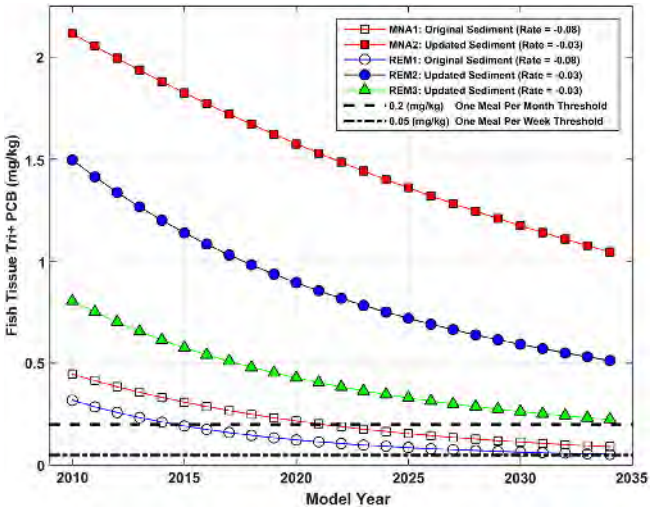

Fig. 7. Emulated model projections for white perch Tri+ PCB concentrations (mg/kg, wet weight) from RM152 for MNA (squares) and the selected remedy (REM) (circles) comparing the time to reach risk thresholds of 0.2 and 0.05 mg/kg at 8% (open symbols) and 3% (filled symbols) exponential decay rates for original mechanistic model concentrations (MNA1, REM1), updated sediment concentrations from remedial design sampling (MNA2, REM2), and hypothetical scenario that applies the RS1 target cleanup levels to RS2 and RS3 using updated sediment concentrations (REM3) (triangles).

4. Discussion

4.1. Interpretation of key findings

4.1.1. Model emulation

Model emulation provides a fast and inexpensive way to efficiently calculate outputs from inputs for complex mechanistic models, while retaining underlying physics-based properties. The method of model emulation is relatively new, with recent developments in global climate modeling stimulating the need to quantify uncertainty in complex mechanistic simulation models (Castruccio et al., 2014). An approach similar to ours was proposed by Margvelashvili et al. (2010) emulating a linked one-dimensional sediment/contaminant and three dimensional sediment transport model in the South-East Tasmanian coast of Australia.

For the Hudson River, sediment fate and transport model emulation successfully reproduced mechanistic model projections of sediment and water Tri+ PCB concentrations in the UHR and fish Tri+ PCB concentrations in the LHR. These results demonstrate that essential elements of the mechanistic mass balance model were captured by the emulator and support its validity for re-visiting temporal projections of fish tissue concentrations in the LHR with updated model inputs. Use of the emulator allowed us to update original predictions without necessitating access to computer codes that are often not readily available to third party investigators. Model emulation may also reduce the time to update complicated simulation models, because recalibration procedures may also entail re-evaluation of the physical mechanisms of the model itself. We believe these features of model emulation could enhance the transparency and accountability of the comparisons of alternative remedial scenarios.

4.1.2. Surface sediment concentrations and natural recovery

Extensive systematic remedial design sampling of surface sediment conducted to delineate dredge areas showed that the mechanistic model predictions of surface sediment concentrations underestimated surface PCBs under MNA and post-remediation scenarios and overestimated the rate of decrease in surface sediment PCBs. The higher than predicted post-remediation concentrations primarily resulted from high concentrations of PCBs in surface sediment adjacent to the planned dredge areas (Field et al., 2011).

Multiple reasons are possible for the mechanistic model underestimating surface sediment Tri+ PCBs, but processes that resulted in an overstated effective recovery rate (8%, MNA1 scenario) (as compared to our empirical estimate of <3% from data only available after the original model was developed) should be considered. Overestimated natural recovery rates are not unique to this model or this situation. For example, models developed by GE for the UHR had a similar effective decay rate (QEA, 1999a). Rates of recovery derived from data collected in the 1970s to mid-1980s have also led to overly optimistic estimates of rates of decline. Consistent with our findings, PCB concentrations in Great Lakes salmonids declined at high double digit rates in the 1970s and 1980s, but the inclusion of more recent data showed that declines have slowed to the low single digits in the 1990s and later (Rasmussen et al., 2014). Examination of PCB data from the 1970s to 2000s in several species of Great Lakes fish suggest that the estimates of contaminant decline were overly optimistic and responses to mitigation weaker than anticipated (Carlson et al., 2010; Sadraddini et al., 2011).

4.1.3. Estimated rate of recovery and fish concentrations

Monitoring data for adult white perch collected annually at RM152 in the late spring between 1997 and 2014 (NOAA, 2015) were normalized to 3% lipid for consistency with the USEPA FISHRAND model and overlaid on updated emulated model predictions for MNA (MNA2) at 3% and MNA1 at 8% decay. The original mechanistic model understates the measured tissue concentrations, whereas the updated predictions using 3% decay are more consistent with the measured data (Fig. 10).

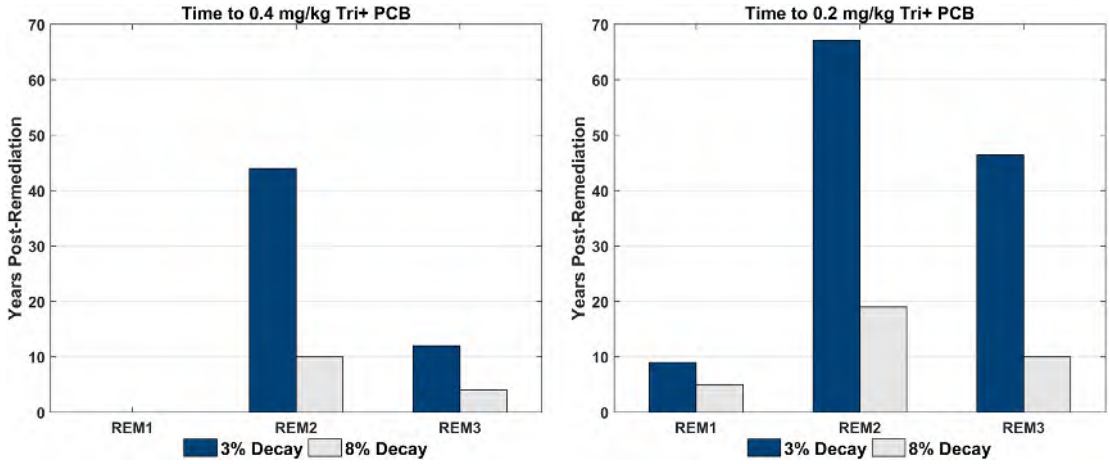

Fig. 8. Emulated model projections of the number of years to reach 0.4 and 0.2 mg/kg Tri+ PCB thresholds for white perch at RM152 under three remedial scenarios and two exponential decay rates, 3% and 8%: the selected remedy with original initial sediment concentrations (REM1), the selected remedy with updated initial sediment concentrations (REM2), and a hypothetical scenario that applies the RS1 target cleanup levels to RS2 and RS3 using updated sediment concentrations (REM3).

It could be argued that this apparently lower than expected decay rate in LHR white perch tissue concentrations is an artifact of Tri+ PCB releases from UHR dredging which began in 2009. However, the updated predictions equally describe trends in monitoring data collected between 1997 and 2009 (Fig. 10), supporting the lower than anticipated 3% recovery rate. It should also be noted that, due to a change in fish processing protocol between 2004 and 2013 (USEPA, 2015), lipid-adjusted Tri+ PCBs shown in Fig. 10 may understate actual concentrations during that time period. Adjusting these data for this change in protocol would shift Tri+ PCBs upward, suggesting even slower recovery rates, again supporting our finding that recovery rates are <8%. Similar results were observed for largemouth bass (Supplementary Fig. S-4). The monitoring data do not definitively identify the correct decay rate, but 3% is a demonstrably better fit to the data than 8%.

4.2. Use of model emulation to evaluate uncertainty

Resource managers need to account for uncertainty in modeled forecasts to avoid selecting overly optimistic, or pessimistic, remedial options. For relatively simple measurement endpoints, statistical analyses are regularly used to quantify uncertainty. For example, uncertainty in exposure estimates is generally quantified using 95% confidence limits. When more complicated functions of the data are involved, the statistical methods of bootstrapping (Efron, 1979) and Monte Carlo simulation (Manly, 1991; USEPA, 1997) are used to describe uncertainty distributions. Bootstrap and Monte Carlo methods involve selecting equation inputs from statistical distributions to which model equations are applied, producing distributions of model outputs. Traditional metrics of uncertainty, such as confidence intervals or percentiles, are calculated directly from the output distributions. The time required to run linked sediment fate and transport models precludes direct application of bootstrap and Monte Carlo methods, because the model runs must be repeated many times to develop statistical distributions of output parameters.

Our model emulation provides a novel approach to extend the utility of complex linked sediment transport, contaminant fate and transport, and bioaccumulation models for the Hudson River by creating a computational shortcut that reliably predicts mechanistic model outputs from imperfectly known model inputs. By varying inputs to the model emulator (i.e. sediment concentrations and decay rates) within reasonably constrained ranges, the uncertainty distributions of emulated outputs were developed, simulating the uncertainty distributions of the mechanistic model. Importantly, because the mechanistic model is based on linked physical processes thought to be predictive, the model emulator can also be considered to be similarly predictive. The use of model emulation allowed for the investigation of the sensitivity of model outputs to uncertainty in model inputs, including both bias and precision.

4.2.1. Bias

The emulator was used in a deterministic way by modifying model inputs. The resulting mechanistic model forecasts were highly sensitive

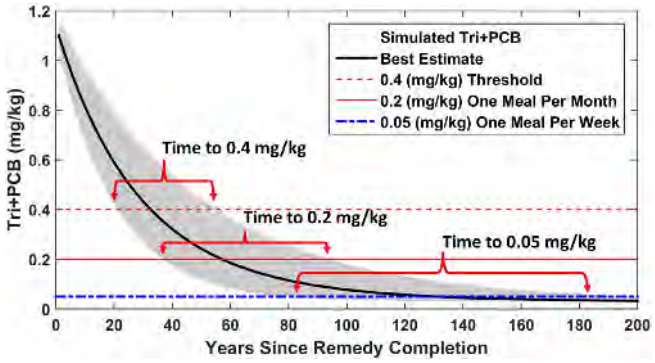

Fig. 9. Monte Carlo distribution of Tri+ PCB concentrations (mg/kg) in white perch at RM152 using the emulated model for the selected remedy with updated sediment concentrations and exponential decay rates in sediment Tri+ PCBs between 2 and 5%.

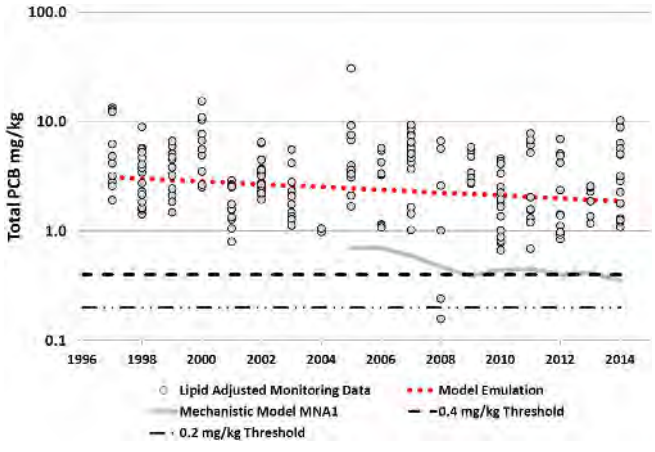

Fig. 10. Emulated model (dotted line) for white perch Tri+ PCB (mg/kg; normalized to 3.0% lipid) from RM152 with 3% exponential decay compared to monitoring data for white perch between 1997 and 2014 (circles) and risk thresholds (0.2 and 0.4 mg/kg PCBs) (horizontal dashed lines). Dredging began in 2009 and was completed in 2015.

to changes (e.g., bias) in initial sediment bed Tri+ PCB concentrations and temporal trend rates, but less so to variation in loads from upstream sources. This paper focuses on the scenario with upstream input concentration decaying to 2 ng/L Tri+ PCBs by 2005, which is consistent with recent monitoring data (USEPA, 2010). The mechanistic models indicated that recovery eventually would be limited with a 2 ng/L upstream baseline load compared to complete source control (upstream load = 0 ng/L). However, the emulated model for 0 upstream load (results not shown) did not differ much from the 2 ng/L model during the emulation period, possibly because initial higher than expected sediment concentrations and lower than expected decay rates mask relatively small differences due to upstream loads.

Updating input sediment Tri+ PCB concentrations to reflect more comprehensive, recent sampling led to the realization that concentrations observed in 2003 sample data exceeded the deterministic upper bound developed from the mechanistic model. Updating input bed sediment concentrations with this new information led to longer estimated recovery times for LHR fish, indicating reduced apparent benefit forecasted for the selected remedy.

The rate of natural recovery is more uncertain than surface sediment concentrations in 2003 because recovery estimates require comparisons with data from older sampling programs, which were based on subjective sampling designs and much smaller sample sizes. Although no completely unbiased sediment sampling program had been conducted prior to 2003, the 1991 UHR transect survey (O'Brien and Gere Engineers, Inc., 1993) was closest to an unbiased systematic sampling study with spatially extensive coverage and many sampling locations distributed throughout the UHR. Lack of unbiased estimates of mean surface concentration at multiple points in time limit the potential to accurately estimate the natural recovery rate. For our study of bias in the decay rates, we used 3% because, while we believe that our sediment decay rate estimate is the best available, the fact that it is based on just two time steps and because only one time step is based on a completely unbiased sampling design, the estimate of 1.3% exponential decay is highly uncertain. Therefore, for evaluating bias, we used 3% as a value that is meaningfully <8%, yet not overly pessimistic. Such subjectivity about sediment recovery rates, at one of the most heavily studied Superfund sites in the United States, is disconcerting and should stimulate a focus on improving the estimate of the rate of recovery at other contaminated sites where remedial alternatives are being evaluated.

4.2.2. Precision

The precision of model forecasts was estimated using a parametric Monte Carlo approach to simulate autocorrelated time series of bed sediment Tri+ PCB concentrations. Sediment concentration inputs were modeled as a first order (i.e. exponential) decay function with temporally correlated residual errors. Application of the model emulator to the 1000 sets of simulated sediment time series resulted in corresponding ensembles of water and fish tissue time series. As discussed above, temporal recovery rates at the Hudson River site are highly uncertain, so the effects of this uncertainty were incorporated into this analysis by simulating first order decay rates as a range of values uniformly distributed from 2% to 5%. This range was chosen subjectively, but nonetheless the analysis illustrated that even modest uncertainty in decay rates can translate into a wide range of estimated times to recovery (Fig. 8). This result indicates that reliable estimates of exponential decay rates in contaminated media are required for reliable remedial alternatives comparisons.

Each of the 1000 simulated time series varies through time around its selected exponential decay rate. When data are strongly correlated temporally, concentration time series may wander far from the exponential decay curve for significant periods of time, leading to greater uncertainty in estimates of time to threshold values. Although results were not shown, the Monte-Carlo procedure was used to evaluate effects of temporal autocorrelation by holding the exponential decay rate fixed across all 1000 simulations. This analysis showed that times to reach threshold concentrations were insensitive to these types of excursions of sediment concentrations due to autocorrelation.

If large linked contaminant fate and transport models are to be used for remedial alternatives evaluation, supporting sediment data appropriate for estimating temporal decay rates are necessary. Frequently, high resolution geochronology sediment cores are used to deduce sedimentation rates and indirectly extrapolate natural recovery rates that are often extrapolated over large spatial regions. However, exposures to biotic receptors are generally assumed proportional to spatial averages, which may not be adequately represented by a small number of high resolution cores. This problem is likely exacerbated by the tendency for investigators to rely on high resolution cores with interpretable geochronology, which typically are collected in low energy areas with continuous deposition and greater than average sedimentation rates that are not representative of site conditions (USEPA, 1998; QEA, 1999b). Those rates, which could be considered to represent an upper bound on sedimentation rates, are then extrapolated over large areas with varying energy regimes and less interpretable geochronologies.

The model emulation approach was useful for quantifying bias and precision of mechanistic model forecasts of fish tissue Tri+ PCB concentrations at the Hudson River. Further application of the method is recommended at contaminated sediment sites where large contaminant fate and transport models have been developed for use in remedial decision-making. Model emulation at other large sites should provide further support for utilizing this approach when additional site data become available to evaluate model projections.

4.3. Improving model calibration and validation

Following the approach used by Castruccio et al. (2014), model emulation can also improve the objectivity and efficiency of model calibration and validation by using a mechanistic model to "pre-calculate" a relatively wide range of model input and output combinations from which a model emulator can be developed. The emulator is then used to iterate on model inputs until optimal combinations of input parameters minimizing error between outputs and sample data are obtained. The emulator provides a mechanism to efficiently calculate combinations of inputs and outputs, allowing many more combinations of model parameters to be evaluated than would otherwise be possible using the mechanistic model directly.

This approach would provide an understanding of the full range of inputs calibrating to the sample data. Combinations of model parameters resulting in similar model fit to data would be considered to represent similarly likely scenarios. If only a small range of model parameters fit the data well, one would conclude that the available data are adequate to uniquely identify the most likely model. In this situation, one could be confident in model projections, whereas a broad range of model parameter combinations resulting in similar model fit to data, would suggest that the sample data are inadequate to uniquely identify a likely model. In this situation, one would not ascribe a great deal of confidence in modeled projections.

4.4. Implications for remedy selection

The model emulation results demonstrate the importance of generating an accurate estimation of both surficial sediment concentrations and the rate of natural recovery of the sediment surface in order for mechanistic models to provide useful information for decision-makers on the relative comparisons among remedial alternatives. If the model-predicted rate of natural recovery is too high, the magnitude of the difference between MNA and an active remedy or between various active remedies, such as the selected remedy for the Hudson River site and a more comprehensive alternative, will be underestimated. USEPA considered two alternative dredging scenarios: the selected remedy (REM1) and a full section removal. The full section removal scenario essentially doubled the area to be dredged (additional 190 ha). According

to USEPA's review of alternatives, full section removal would have been more protective, but the projected difference in fish concentrations (and risk) between the two remedial scenarios was considered too small to warrant the increased cost (USEPA, 2002). The difference between those two dredging alternatives was understated because of the overly optimistic rate of recovery of the surface sediment considered. This is illustrated in Fig. 7, which clearly discriminates between the different alternatives and shows the large difference in time to reach risk thresholds for emulated fish concentrations for the selected dredging remedy (REM1) and for the updated scenario with a more aggressive (but less than full section removal) remedy (REM3). This hypothetical remedy, which maintained the same target cleanup levels for surface sediment throughout the UHR, would involve removing an estimated additional 71 ha, <50% of the area under the full section removal scenario.

While we estimate risk thresholds would be reached meaningfully sooner under this hypothetical and more aggressive remedy (REM3) than under the selected remedy with updated sediment surface concentrations and decay rate (REM2), the estimated time to thresholds would still be longer than the original mechanistic model projections (REM1). Our analysis suggests that achievement of LHR fish PCB threshold concentrations targeted as remedial action objectives to protect human health will be delayed for up to several decades. Our analysis also implies that the remedial action objectives will not be met in the time frame identified in the 2002 ROD for the Hudson River (USEPA, 2002) without implementing a more comprehensive remedy.

Models are often considered to be most useful for evaluating uncertainty in predictions of the relative, as opposed to absolute, benefits for alternative remedial options (Glaser and Bridges, 2007). In such situations management teams may rationalize potential inaccuracies in model forecasts by assuming that relative comparison of forecast remedial effectiveness is possible even when absolute forecasts may be inaccurate or highly uncertain. Our analyses suggest that when models are biased or imprecise the relative differences between remedial alternatives can be significantly under- or over-estimated. In addition, the model emulation approach can serve to improve precision and reduce bias in model output, therefore more reliably discriminating among remedial alternatives. Box and Draper (1987) stated "All models are wrong, some are useful". The models discussed in this paper rely on accurate surface sediment concentrations and the rate of change to make reliable projections of concentrations in sediment, water, and biota. The best way for resource managers and decision-makers to know if the models used for comparing remedial options are useful is to collect systematic, unbiased data on surface sediment concentrations that can be used to estimate the rate of natural recovery and to regularly monitor fish tissues for bioaccumulative contaminants.

5. Conclusions

Our analyses demonstrate that pre-remedial surface sediment Tri+ PCBs in the Upper Hudson River were two to three times higher and estimated post-remediation Tri+ PCBs averaged about four times higher than predicted by the original mechanistic models used by USEPA in the Hudson River 2002 ROD. The rate of recovery, as measured by the exponential decay rate of Tri+ PCBs in surface sediment, was overestimated by the original mechanistic models. We estimated a mean of 1.3% and a 95% upper CI of ~3% compared to the ~8% derived from the original EPA and GE mechanistic models.

The emulated models successfully reproduced the mechanistic model projections for sediment and water in the UHR and fish in the LHR. The emulated models were used to incorporate the updated information on higher surface sediment concentrations and reduced rate of sediment recovery. Our model projections suggest that the original mechanistic model projections greatly underestimated the time to reach risk thresholds in the LHR fish, thereby extending by decades the time period for the project to reach its fish PCB-based remedial action objectives in the LHR.

The results also demonstrated the adverse impact of over-estimation of the rate of sediment recovery on the potential ability of risk managers to discriminate among alternative remedial scenarios.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.scitotenv.2016.02.072.

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Appendix A. Mathematical formulation for emulator

Table A.1 summarizes the locations of the four dams (*River Mile* = d_i), acres of cohesive sediments (A_i), distances between dams ($\delta_i = d_i - d_{i-1}$), area remediated and average distance between deposits and downstream dams ($\overline{d}_i$). Table 2 lists the Tri+ PCB concentrations (c_{si}) in surface sediment in 2003 and 2010 for each of the five scenarios evaluated in this study. The load at the ith dam is represented by L_i and the transfer coefficients from water to sediment and sediment to water are represented by γ_i and g_i respectively. With this notation, the processes for deposition and resuspension at each model annual timestep were described mathematically in the following set of four equations which are nonlinear in the transfer coefficients

$$L_i = L_{i-1} \times (1 - g_i \times \delta_i) + \left\{ \gamma_i \times (c_{si} \times A_i) \times \left(1 - g_i \times \overline{d}_i\right) + \beta_i \times (R_i \times c_{si} \times A_i) \right\} \times Q_i \quad \text{(A.1)}$$

where $i = 1,2,3,4$ indexes each of the four modeled sections of the river, β_i represents the sediment to water net transfer coefficient for dredged residuals and R_i represents the 8% decay of post-dredge residual concentrations. If discharge at successive dams is similar ($Q_i = Q_{i-1}$), Eq. (A.1) can also be expressed in terms of water column concentrations as opposed to loads by dividing both sides of Eq. (A.1) by Q_i giving the following Eq. (A.2).

$$c_{wi} = c_{wi-1} \times (1 - g_i \times \delta_i) + \left\{ \gamma_i \times (c_{si} \times A_i) \times \left(1 - g_i \times \overline{d}_i\right) + \beta_i \times (R_i \times c_{si} \times A_i) \right\} \quad \text{(A.2)}$$

For the Hudson River, results were similar for Eqs. (A.1) and (A.2) so the simpler Eq. (A.2) was used for these analyses.

Each of the 25 years from 2010 through 2034 provides a different set of modeled sediment bed and water column Tri+ PCB concentrations from which the best estimates of emulator net transfer coefficients (g_i, γ_i and β_i, $i = 1,2,3,4$) can be estimated using constrained nonlinear least squares. These paired inputs and outputs from the EPA mechanistic model were available for two remedial scenarios; 1) natural recovery (MNA1), and 2) the selected remedy (REM1A). Each of these scenarios was also simulated with the assumptions of 0 and 2 ng/l PCBs entering from upstream of RS1. Modeled time series spanning 30 (2005–2034) and 25 (2010–2034) year time frames for MNA and active remediation respectively, under two sets of upstream input assumptions and four river sections provided 440 ($2 \times 25 \times 4 + 2 \times 30 \times 4$) nonlinear equations in 12 unknown net transfer coefficients (i.e., g_i, γ_i and β_i, $i = 1,2,3,4$). The transfer coefficients were estimated

Table A.1
Summary of input parameters and initial conditions for calibrating model emulator.

Reach	River section	Downstream river kilometer	River section length (km)	Area (ha)			
				Cohesive sediment area (ha)[a]	Alternative REM1 remediated area[b]	Alternative REM2 remediated area[c]	Alternative REM3 remediated area[d]
Thompson Island Pool	RS1	303.4	10.1	42	114	124	124
Schuylerville	RS2	295.2	8.2	54	31	35	56
Stillwater	RS3A	270.7	24.5	93	38	29	64
Waterford	RS3B	263.1	7.6	52	17	13	28
Total					200	201	272

[a] Cohesive sediment area from Tables 5.2a–5.2b in USEPA (2000b).
[b] Area for alternative REM1 from Tables 8–9 in USEPA (2000a).
[c] Alternative REM2 area calculated based on delineated dredge area.
[d] Alternative REM3 area based on delineated dredge area for the selected remedy and additional area estimated from number of cores exceeding RS1 target cleanup levels.

Table A.2
Estimated model emulation nonlinear regression coefficients.

Model coefficients	River section			
	RS1	RS2	RS3A	RS3B
Water to sed	0.0000	0.0350	0.0157	0.0641
Sed to water	0.0160	0.0095	0.0078	0.0451
Post dredge resuspension	0.0251	0.0143	0.0283	0.0357

by constrained nonlinear least squares with MATLAB© Release 2011a (The MathWorks 2011).

Appendix B. Probability model for synthetic sediment time series

The residual process $C_i(t) = C_{0i}e^{-kt+\varepsilon_i(t)}$ was simulated by randomly drawing an exponential decay rate (k) from a uniform probability distribution on the interval 0.02–0.05, followed by simulation of $\varepsilon_i(t)$ as a mean zero normally distributed random variable with covariance matrix $\boldsymbol{C}$ with the entries c_{ij} defined as $cov(\varepsilon_i(t),\varepsilon_i(t+h)) = e^{-a_i h2}$, and covariance between subsections i and j given by $cov(\varepsilon_i(t),\varepsilon_j(t)) = c_{ij}$ for $i \neq j$. The constants a_i and c_{ij} were estimated from the four mechanistic modeled sediment Tri+ PCB concentration time series. The expected mean of the simulated sediment series for the ith subsection is $C_{0i}e^{-kt}$. The simulated series are distributed log-normally because $\varepsilon_i(t)$ is a normally distributed random variable.

The estimated coefficient a_i defining the rate of decline in temporal auto correlation was 0.1. The resulting correlation matrix C was a real symmetric banded matrix with diagonal entries $C_{ii} = 1.0$ and with 5 non-zero off diagonal with values $C_{i,i\pm j} = 1$, 0.90, 0.67, 0.41, 0.20, and 0.08; for $j = 1, 2, \ldots, 5$ respectively and $i = 1, 2, 3, \ldots, 200$ years. The remaining values $C_{i,i\pm j} = 0$; for $j > 5$.

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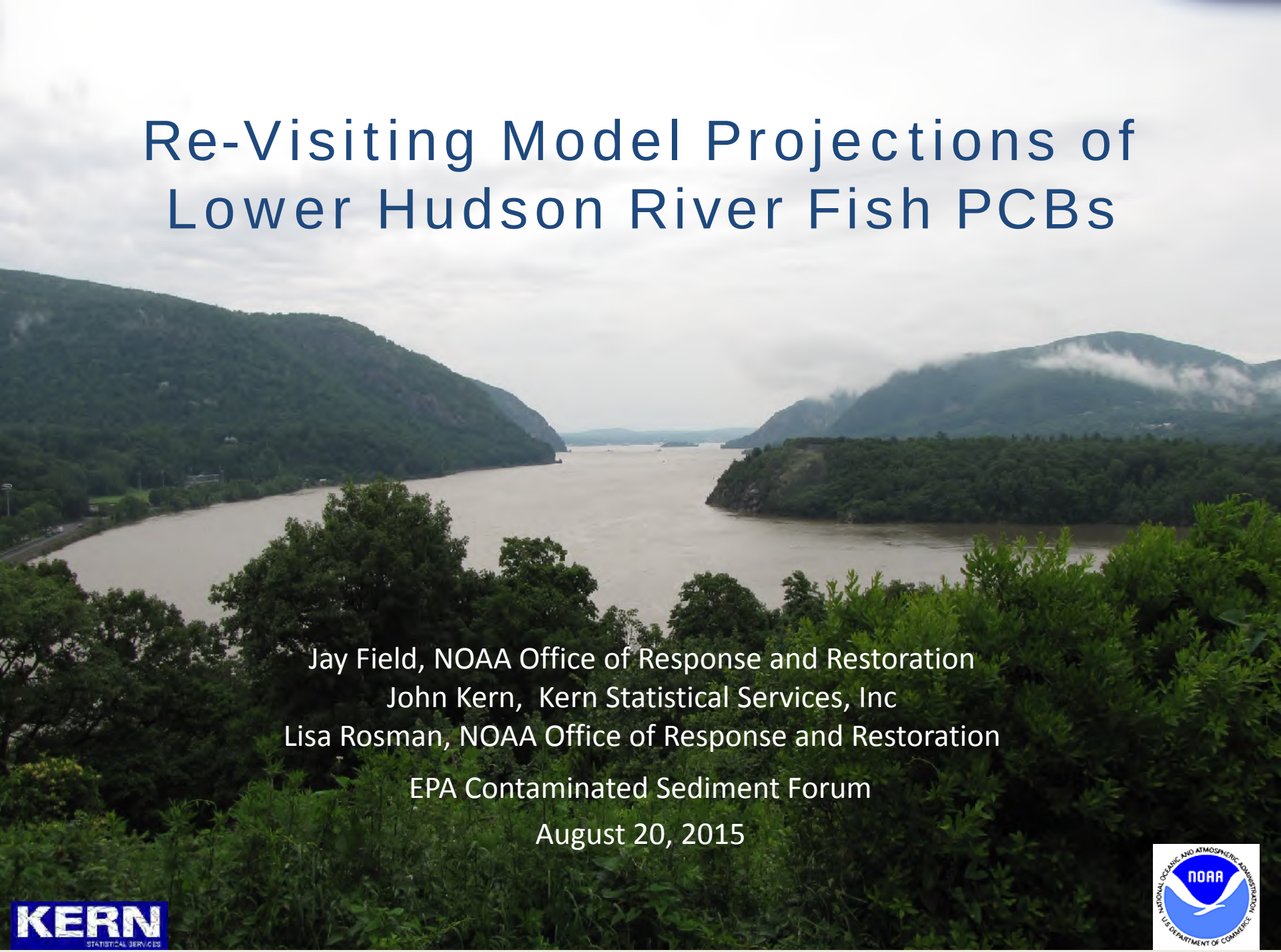
Re-Visiting Model Projections of Lower Hudson River Fish PCBs



Jay Field, NOAA Office of Response and Restoration
John Kern, Kern Statistical Services, Inc
Lisa Rosman, NOAA Office of Response and Restoration

EPA Contaminated Sediment Forum

August 20, 2015

Introduction

- Mechanistic model projections of PCBs in fish played an important role in the comparison of remedial alternatives in the 2002 Record of Decision (ROD) for the Hudson River PCBs Superfund Site
- Post-ROD findings showed that the mechanistic models overestimated the rate of natural recovery in surface sediment
- Model emulation provides a way to update the original mechanistic models with new information

Why Revisit Model Projections?

- Need models to predict the future impact of decisions
- Decisions often difficult, expensive, and controversial
- Similar mechanistic models used to inform decision-making at other Superfund sites
- Rare opportunity to revisit model predictions

Important Questions

- What is the impact of post-ROD data on mechanistic model projections for recovery of fish concentrations in the Lower Hudson River?
- What are the implications for the use of similar models in comparing remedial alternatives?

Overview

- Background (Hudson River, selected remedy)
- Post-ROD findings
- Mechanistic modeling for the Upper Hudson River (UHR) and Lower Hudson River (LHR)
- Emulation of mechanistic model
- Impact of post-ROD findings on mechanistic model projections of recovery of LHR Fish
- Issues and recommendations for estimating temporal trends in sediment

Hudson River PCBs Superfund Site

Hudson Falls/Ft Edward
GE Plant Sites

Former Ft Edward Dam

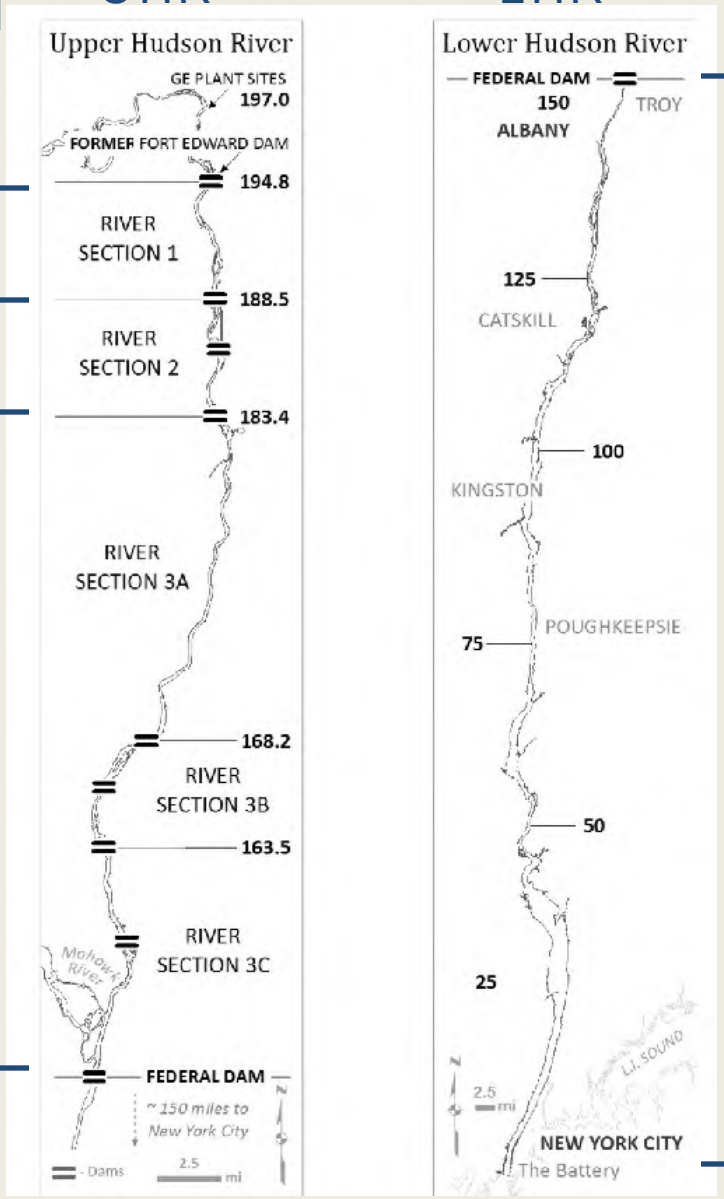
River Section 1

River Section 2

EPA Remedy

River Section 3

Federal Dam



Federal Dam

~ 150 miles

The Battery/NYC

Selected Remedy for the Upper Hudson River (UHR)

- REM 3/10/Select: Dredging and Monitored Natural Recovery
  - Upstream source control (NY State remedial process)
  - Target Cleanup Levels
    - River Section 1 (Thompson Island Pool) ~ 6 miles
      - **3 g/m^2 Tri+ PCBs mass per unit area (MPA)**
      - **10 mg/kg Tri+ PCBs in surface sediment** (~ 25-30 mg/kg total PCBs in top 12 inches)
    - River Sections 2 & 3 (multiple reaches/pools) ~ 35 miles
      - **10 g/m^2 Tri+ PCBs MPA**
      - **30 mg/kg Tri+ PCBs in surface sediment** (~ 60-90 mg/kg total PCBs in top 12 inches)

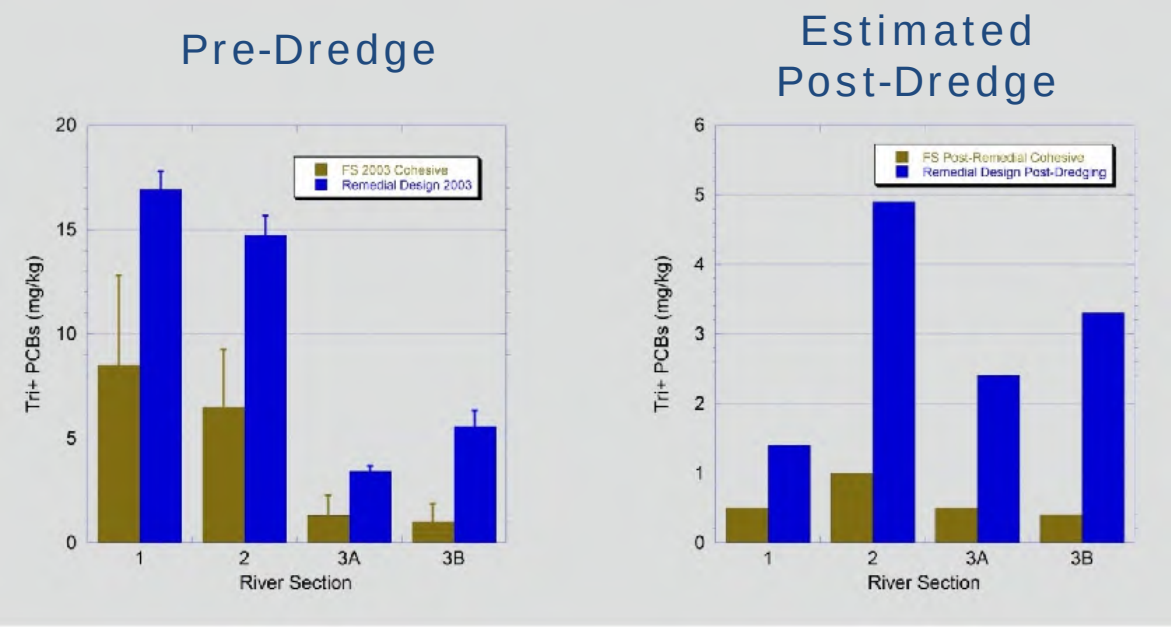
Tri+ PCBs: Trichloro-biphenyl and higher chlorinated PCBs

- Consistent with historical analytical data
- PCBs in HR fish 98-100% Tri+ (USEPA 2002)

Post-ROD Data

- Sediment Data collected for Remedial Design
  - Systematic (unbiased) sampling for UHR (2002-5)
    - RS1: all sediment (cohesive and non-cohesive)
    - RS2 & RS3: cohesive sediment only
  - >8000 cores collected from UHR with PCBs measured in the top 2 inches (5 cm)
  - Mean PCBs assumed to represent 2003 and comparable to 4 cm surface PCBs in mechanistic model output

Surface Sediment PCBs: Mechanistic Model Predicted vs Measured Post-ROD



Tri+ PCBs in surface sediments exceeded the mean by a factor of 2-3 and the upper bound of model predictions

Estimated post-remediation PCBs for the selected remedy were 3-5X higher than model predictions

Surface sediment represents top 4 cm for model and top 2 inches (5 cm) for remedial design data

Empirical Estimate of Natural Recovery Rate

	Average Tri+PCB (mg/kg) in Surface Sediment		
Model Subsection	GE 1991 UHR Survey[1] (Cohesive Sediment)	Remedial Design Data 2002-2005[2]	Exponential Decay Rate
1	20	16.9 (3414)	1.4%
2	18	14.7 (1540)	1.7%
3A	4.3	3.4 (2129)	2.0%
3B	5.7	5.6 (685)	0.1%
Mean			1.3%
95% CI			-0.1% - 2.6%

< 3%

[1] O'Brien & Gere Engineers, Inc. 1991 Data Summary Report, Hudson River Project

[2] Includes cohesive and non-cohesive sediments from top 2 inches in River Section 1 and cohesive only in Sections 2 and 3. Data collected 2002-2005, considered to represent concentrations in 2003.

Summary of Post-ROD Findings

- Measured surface sediment PCBs higher than predicted by the mechanistic model throughout UHR
- Rate of sediment recovery slower than mechanistic models predicted
- PCB loads from the UHR to the LHR prior to 2009 greater than predicted by EPA's mechanistic models and showed little evidence of decline[1]

[1] USEPA 2010. Hudson River PCBs Site EPA Phase 1 Evaluation Report

Importance of Incorporating New Data into Mechanistic Model Framework

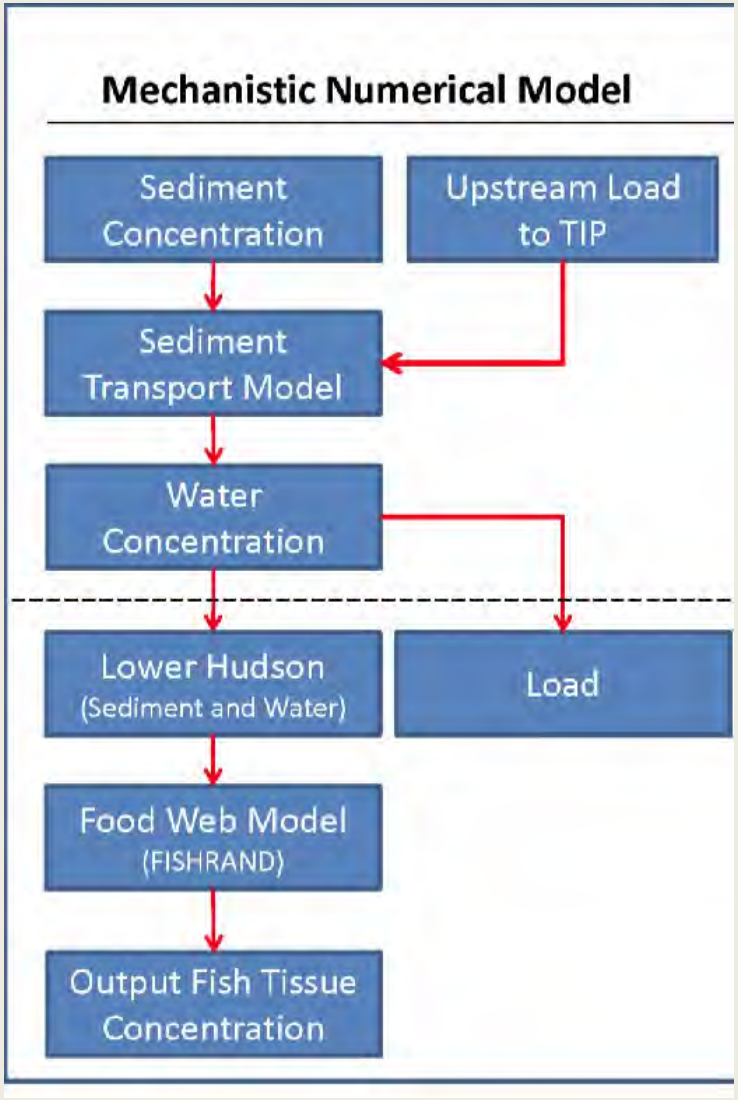
- Post-ROD findings in UHR sediment and estimates of load to LHR likely impact projected declines in LHR fish PCBs

- Re-running the original mechanistic models with new data was not an option because of the cost and effort involved

Why Use Model Emulation?

- Provides alternative approach to efficiently condense complex integrated models into a simple, easy-to-use model
- Maintains the underlying relationships within the mechanistic model
- Enables use of updated data and evaluation of alternative scenarios
- Used effectively for large numerical ocean and climate change models

Mechanistic Model Schematic



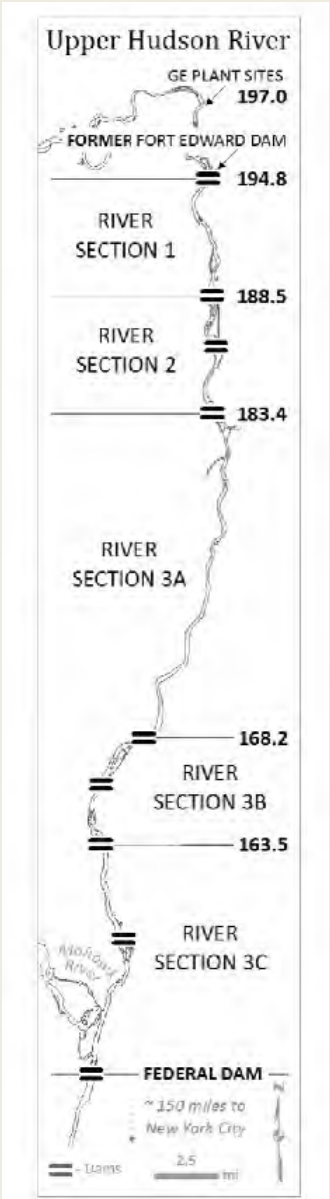
Model boundary conditions: Upstream PCB input into RS1 (Thompson Island Pool)

Surface sediment PCBs projected for UHR model subsections

PCBs in water projected for UHR model subsections

Output from UHR models used to predict fish PCBs at 4 LHR locations between RM152 and RM50 for 4 species of fish

Upper Hudson River (UHR)



Boundary Condition

RS1

RS2

RS3A

RS3B

RS3C

Surface sediment & water PCBs projected for UHR

PCB Load from Waterford (RS3B) used as input to LHR models

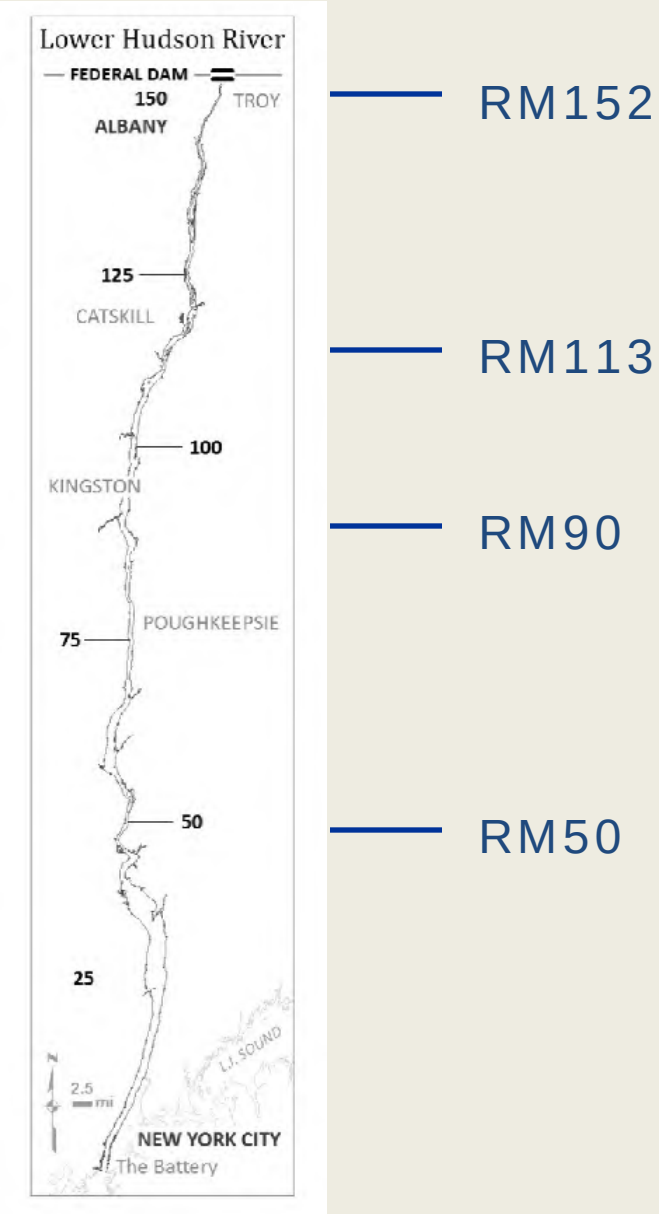
Lower Hudson River (LHR)

Output from mechanistic model PCB Load from Waterford (RS3B) used as input to LHR models

Farley[1] model used to project LHR water and sediment PCBs

FISHRAND Food Web model used Farley model output to project PCBs in 4 species of fish at 4 LHR locations

- White Perch
- Largemouth Bass
- Brown Bullhead
- Yellow Perch



[1]Farley KJ 1999. An integrated model of organic chemical fate and bioaccumulation in the Hudson River Estuary

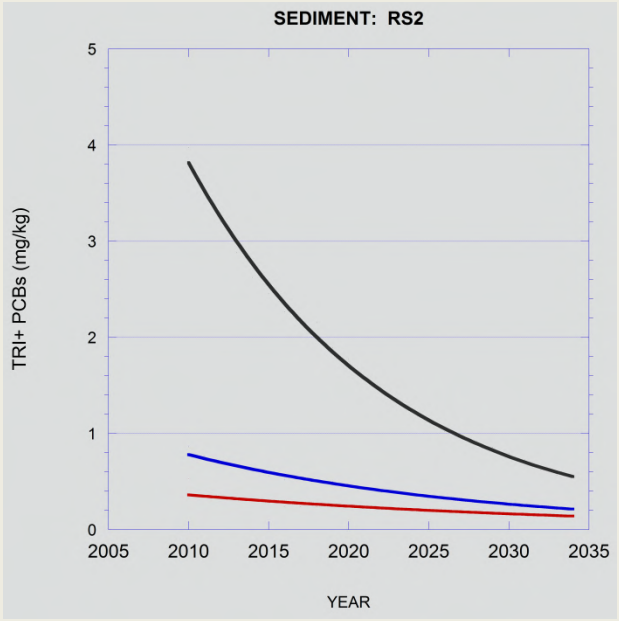
Mechanistic Model Remedial Scenarios

- MNA: Monitored Natural Attenuation with source control (assumes upstream boundary conditions of 2 ng/L PCBs by 2005)
- REM-3/10/Select: Selected Remedy
- REM-0/0/3: Full section removal in RS1 & RS2

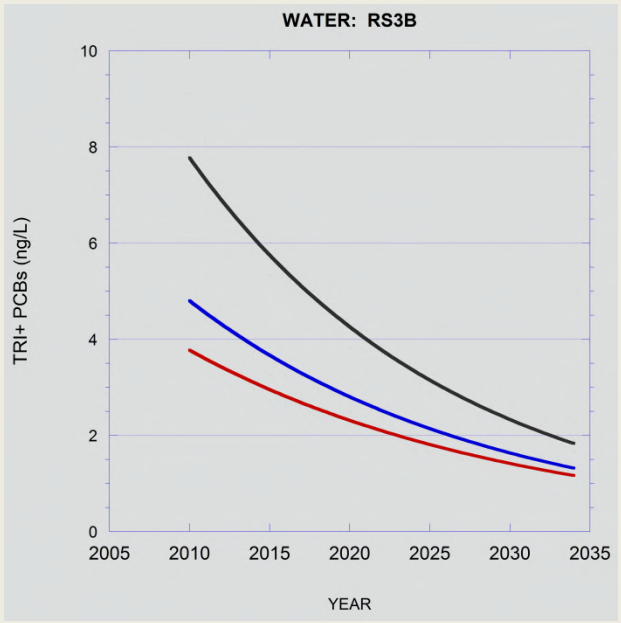
Models assumed active remediation began in 2003 and completed by 2010

EPA Mechanistic Model Projections for 3 Remedial Alternatives

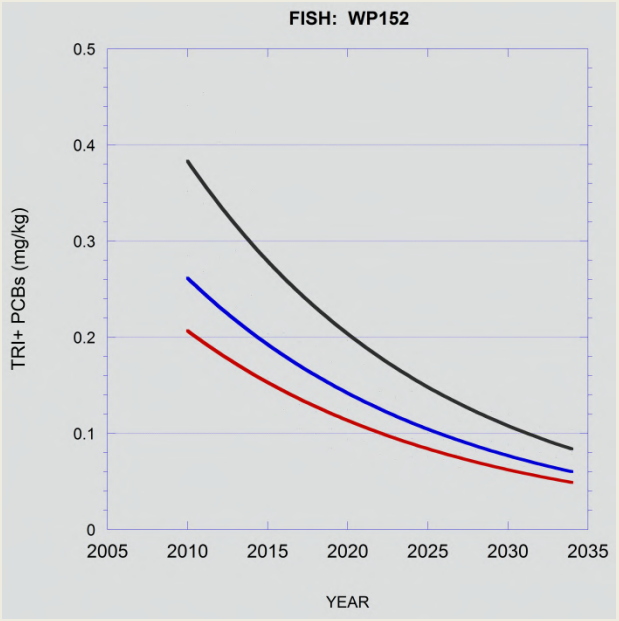
SEDIMENT (RS2) [1]

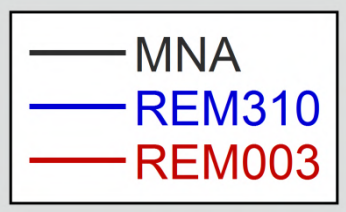


WATER (RS3B) [1]



FISH (LHR) [2]





[1] USEPA 2000. Hudson River PCBs Reassessment RI/FS Phase 3 Report. Feasibility Study.

[2] USEPA. 2002. Hudson River PCBs Site Record of Decision and Responsiveness Summary.

Model Emulation Approach

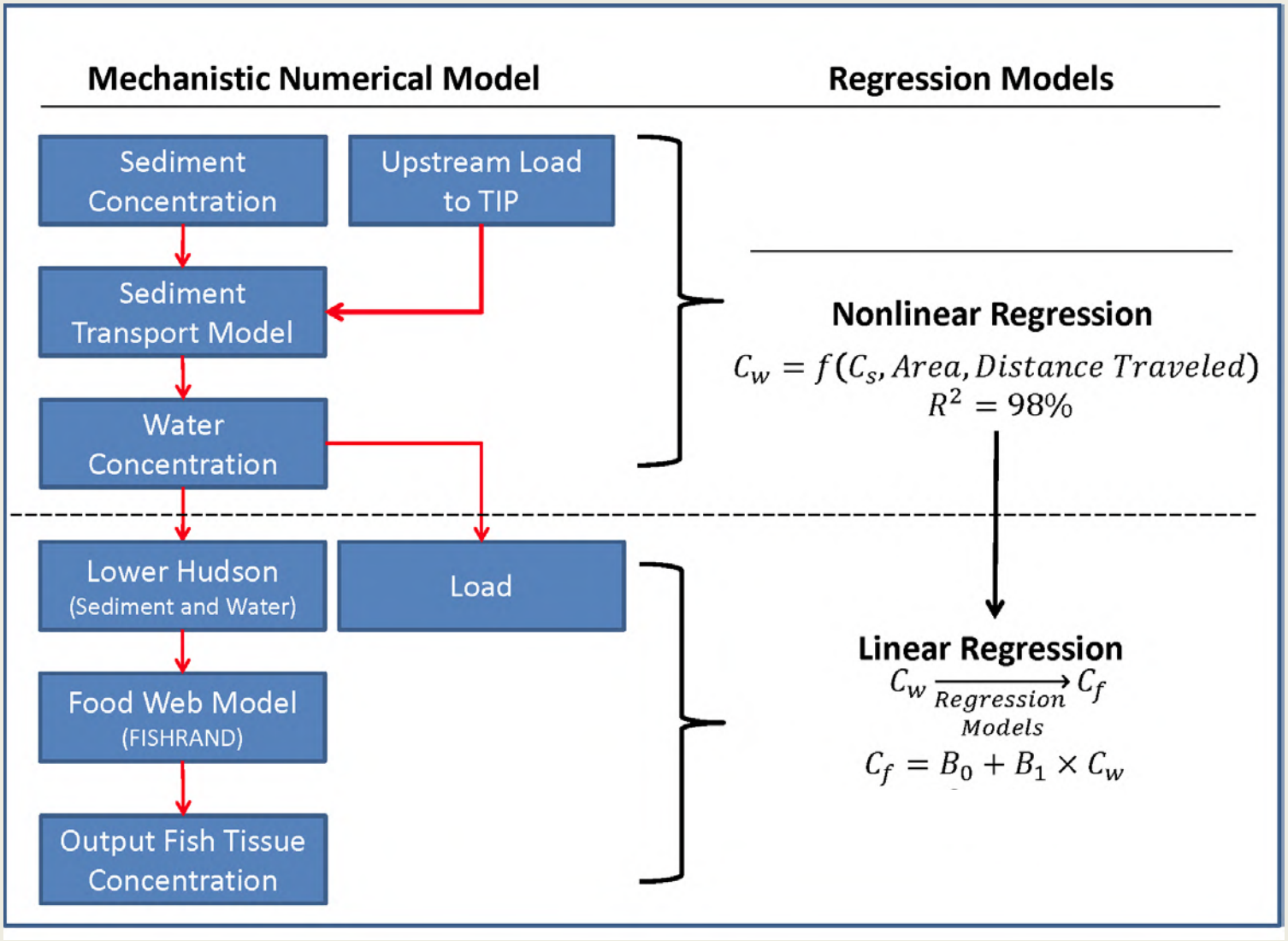
- Develop statistical models to reproduce mechanistic model projections for PCBs in UHR surface sediment and water and LHR fish for Monitored Natural Attenuation (MNA) and the selected remedy (REM)

- Use updated surface sediment PCBs and rate of decrease in sediment PCBs to assess the impact of the post-ROD findings on predictions of LHR fish PCBs

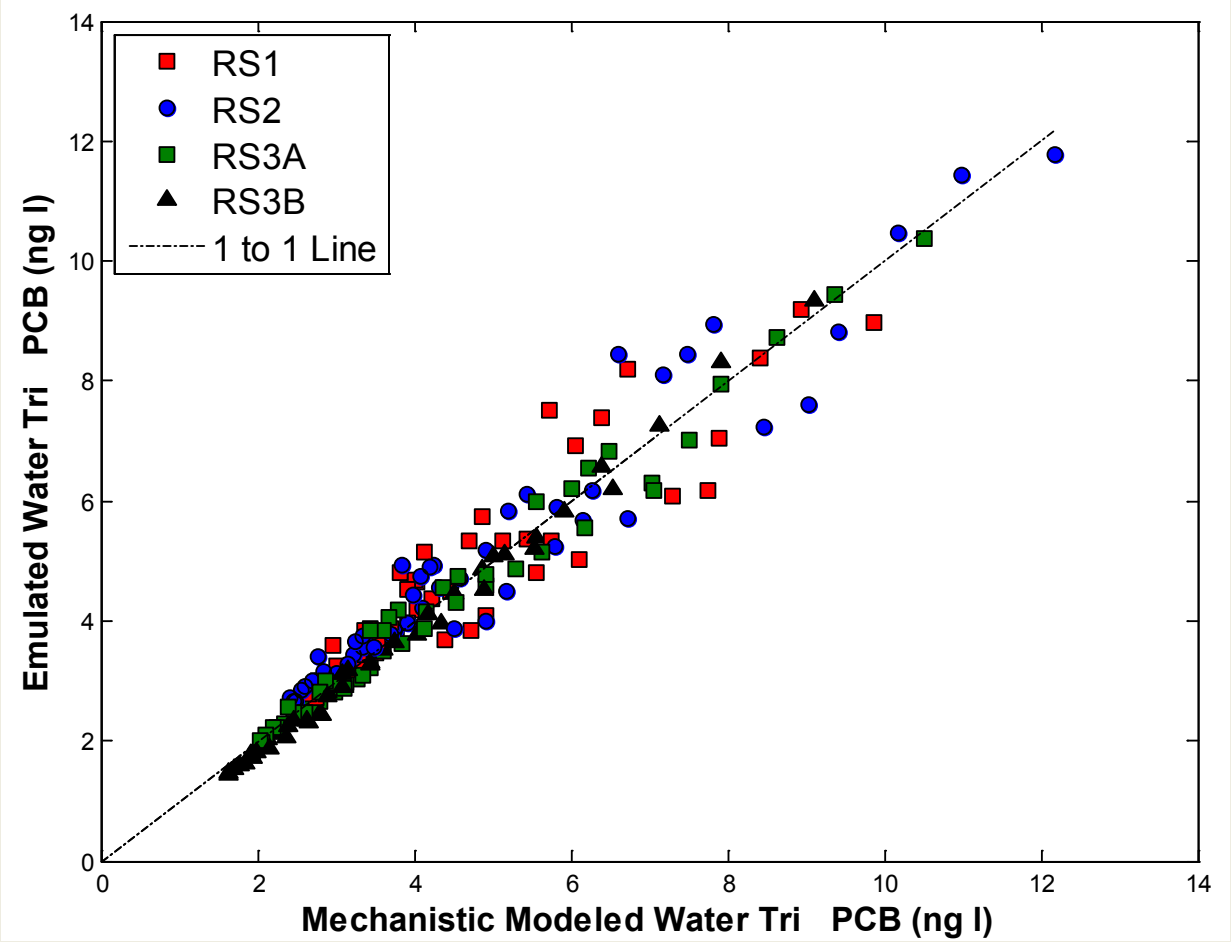
Overview of Model Emulation

- UHR Sediment: Reproduce mechanistic model projections for cohesive sediment PCBs in 4 UHR subsections for MNA and the selected remedy
- UHR Water: Use non-linear regression to predict water PCBs in 4 UHR subsections from sediment PCBs
- LHR Fish: Use linear regression to predict fish PCBs in 4 species of fish at 4 locations in the LHR from water PCBs at Waterford (RS3B)

Model Emulation Schematic



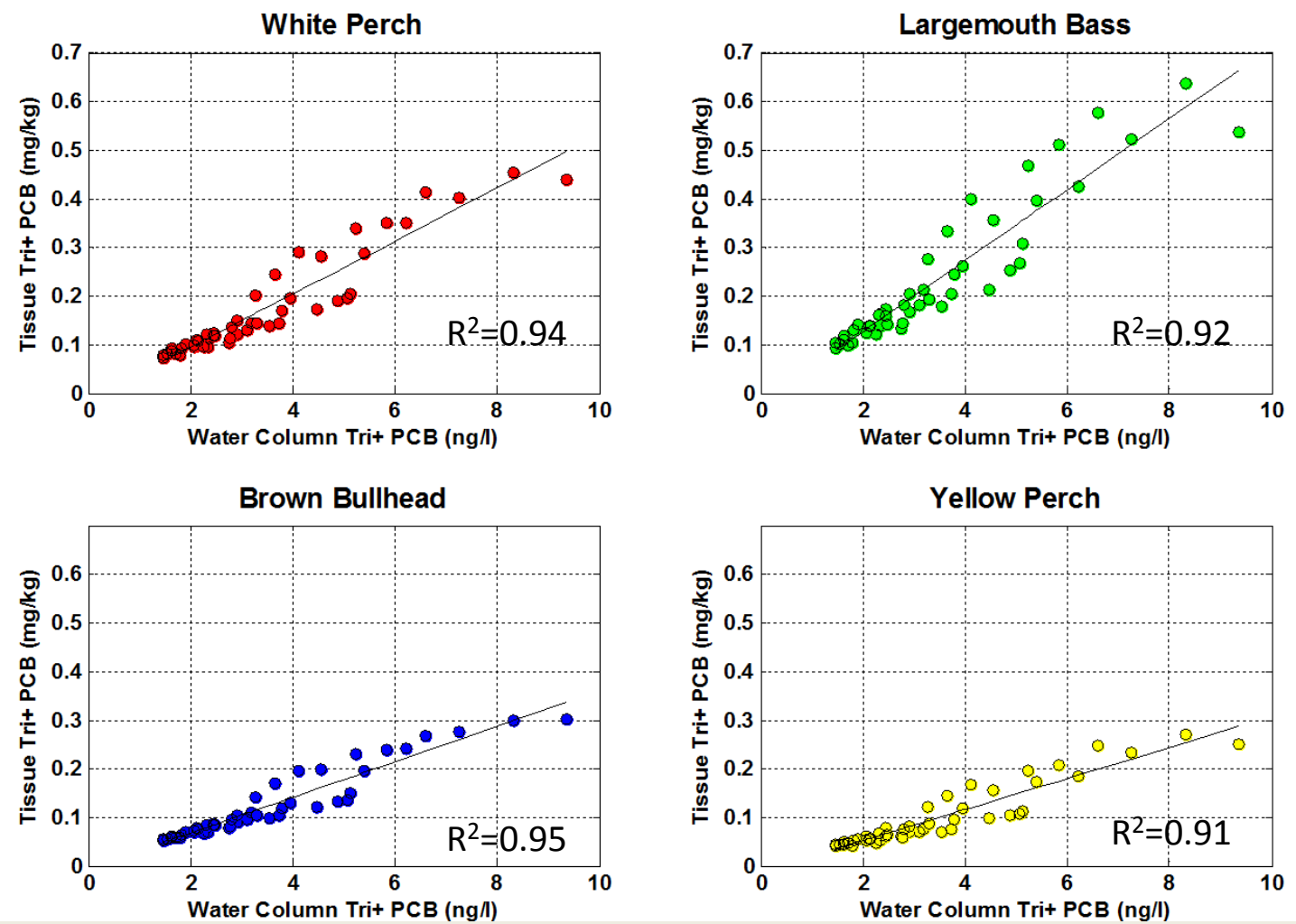
Emulated vs Mechanistic Model Water Concentrations (Tri+ PCB, ng/L)



Data Source: USEPA. 2002. Hudson River PCBs Site Record of Decision and Responsiveness Summary.

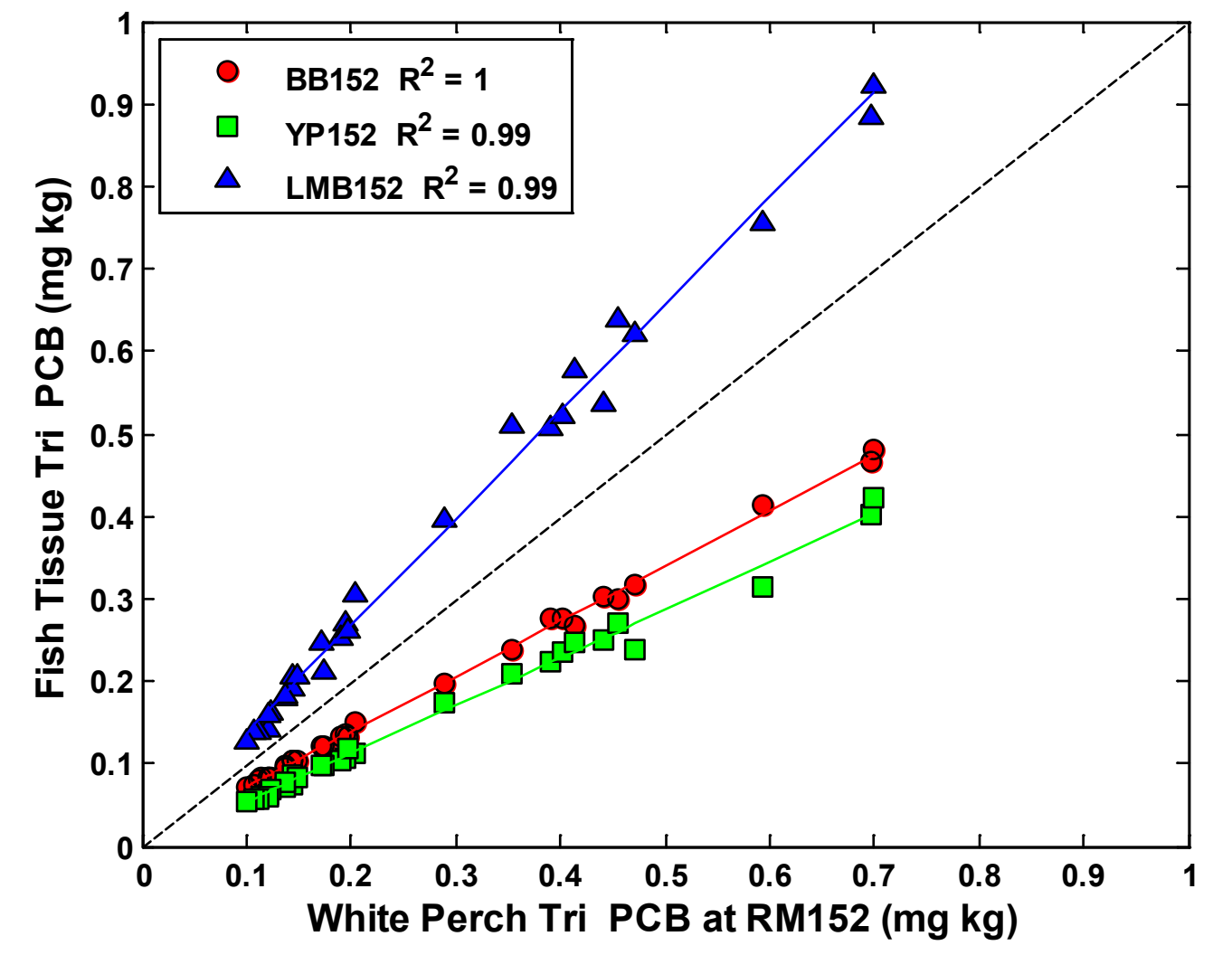
Emulation of LHR Fish PCBs Mechanistic Model Output

Water (RS3B) vs Fish PCBs at RM152



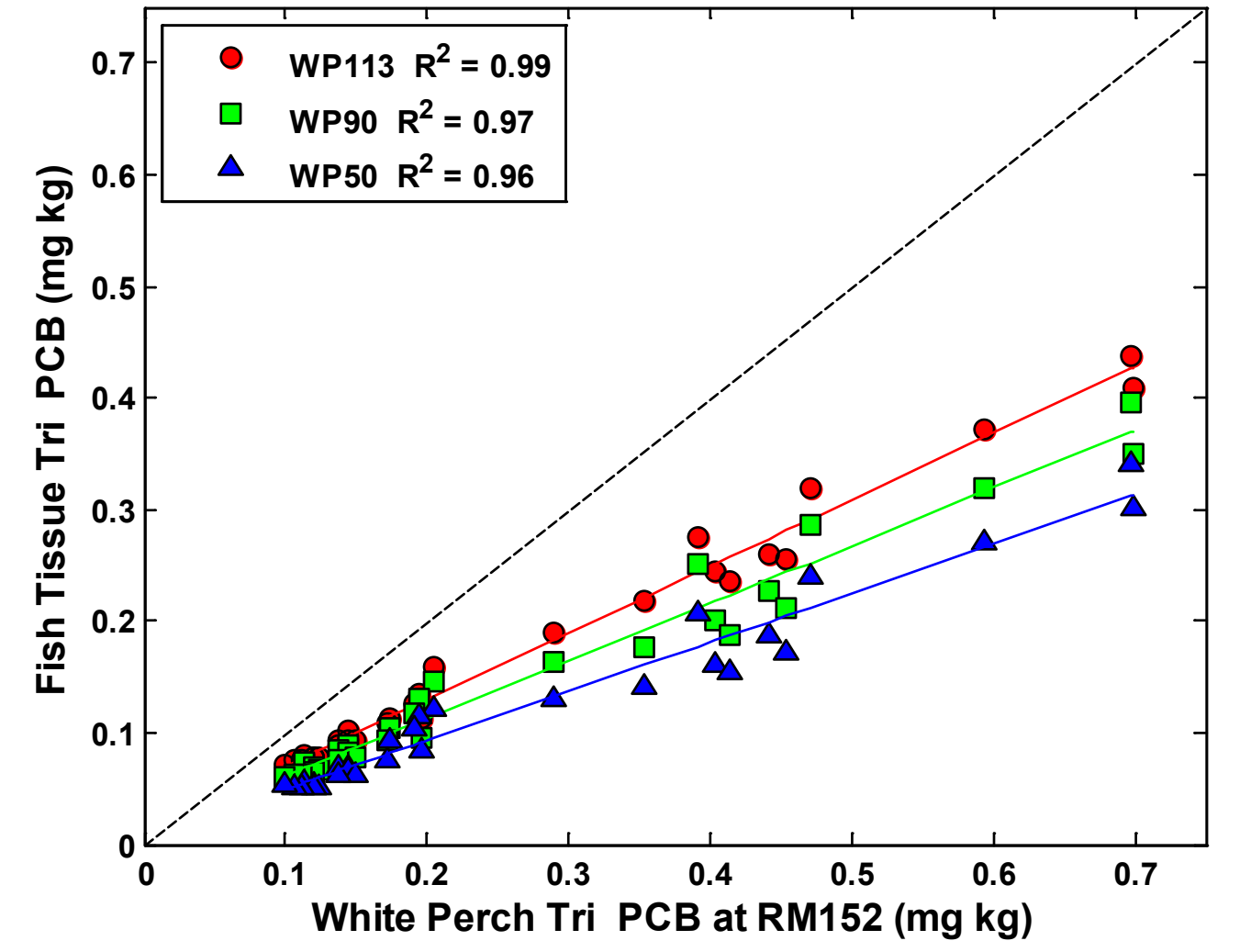
Data Source: USEPA. 2002. Hudson River PCBs Site Record of Decision and Responsiveness Summary.

Mechanistic Model Output
Fish Species Comparison



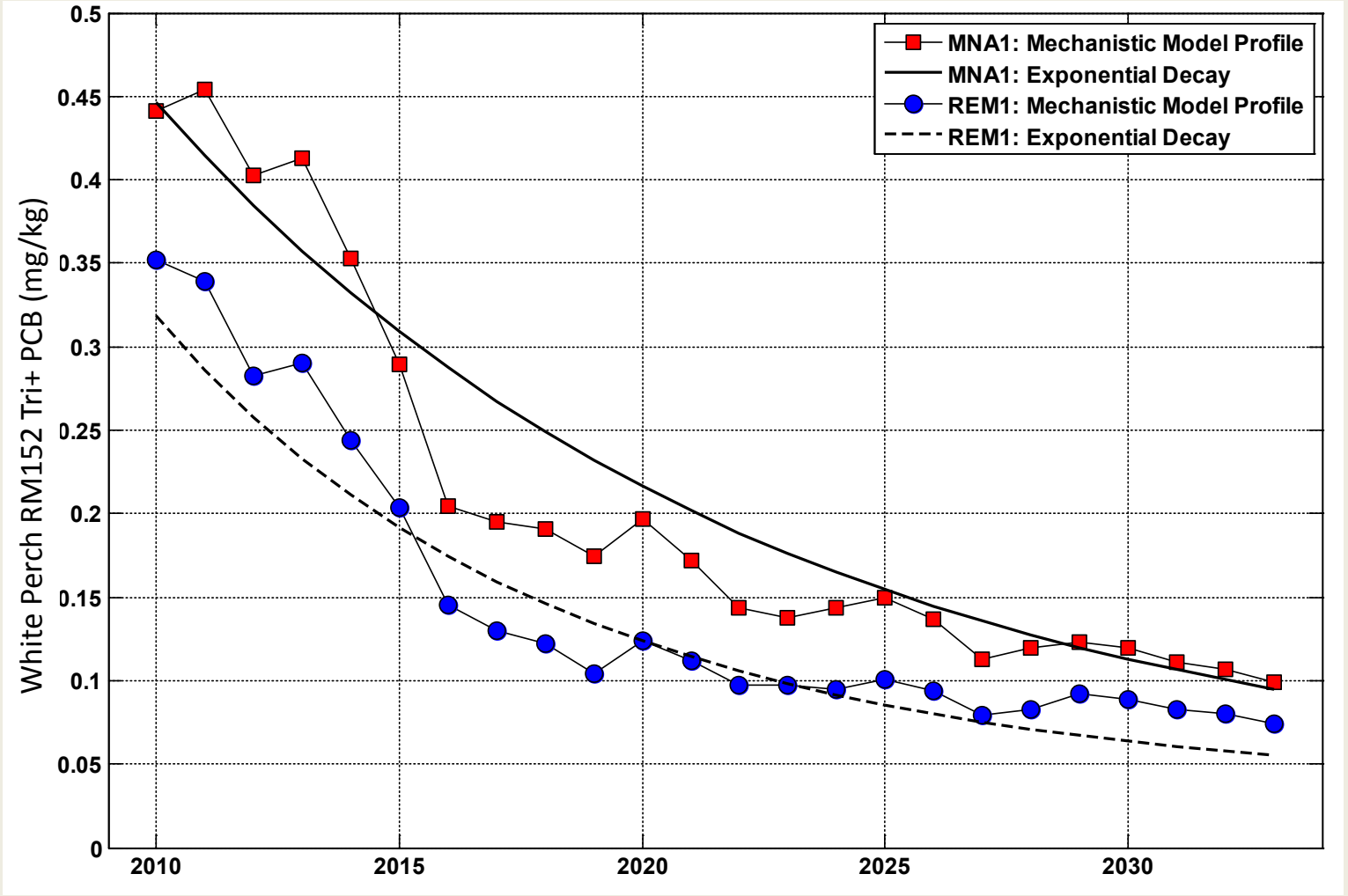
Data Source: USEPA. 2002. Hudson River PCBs Site Record of Decision and Responsiveness Summary.

Mechanistic Model Output
Fish Location Comparison



Data Source: USEPA. 2002. Hudson River PCBs Site Record of Decision and Responsiveness Summary.

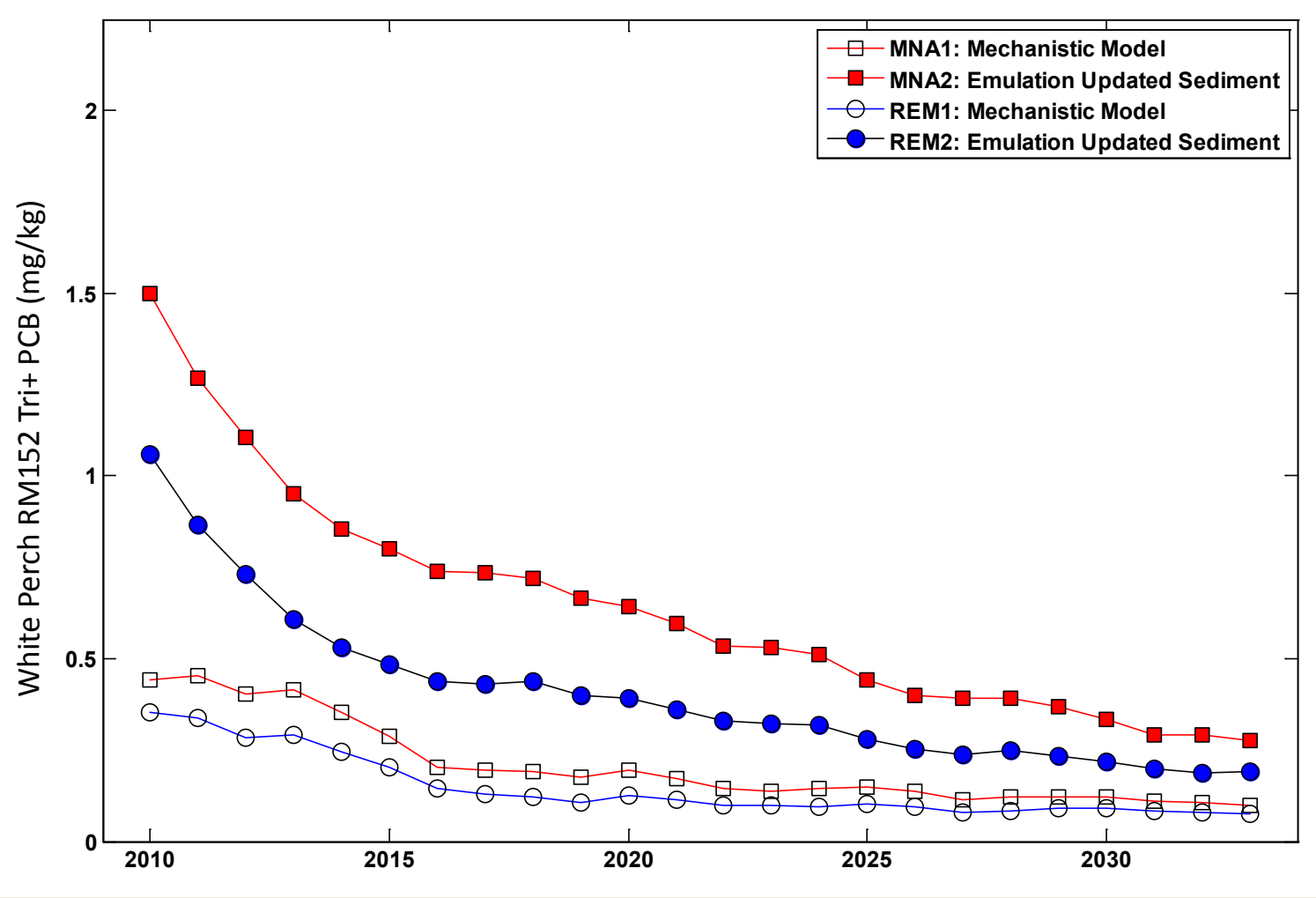
Mechanistic Model Projections vs Exponential Decay (8%) Model



Emulated Model Scenarios

Scenario	Emulated Model Projections
MNA1/REM1	Original model projections for Monitored Natural Attenuation (**MNA1**) and the selected remedy (**REM1**)
MNA2/REM2	MNA (**MNA2**) and the selected remedy (**REM2**) with updated sediment PCBs
REM3	Alternative scenario applying RS1 criteria for MPA and surface PCBs to RS2 and RS3 (**REM3**) with updated sediment PCBs
Exponential decrease	Original (8%) and updated (3%) exponential decrease in sediment PCBs applied to all scenarios

Emulated Model Projections of Fish PCBs with Original (MNA1, REM1) and Updated (MNA2, REM2) Sediment PCBs

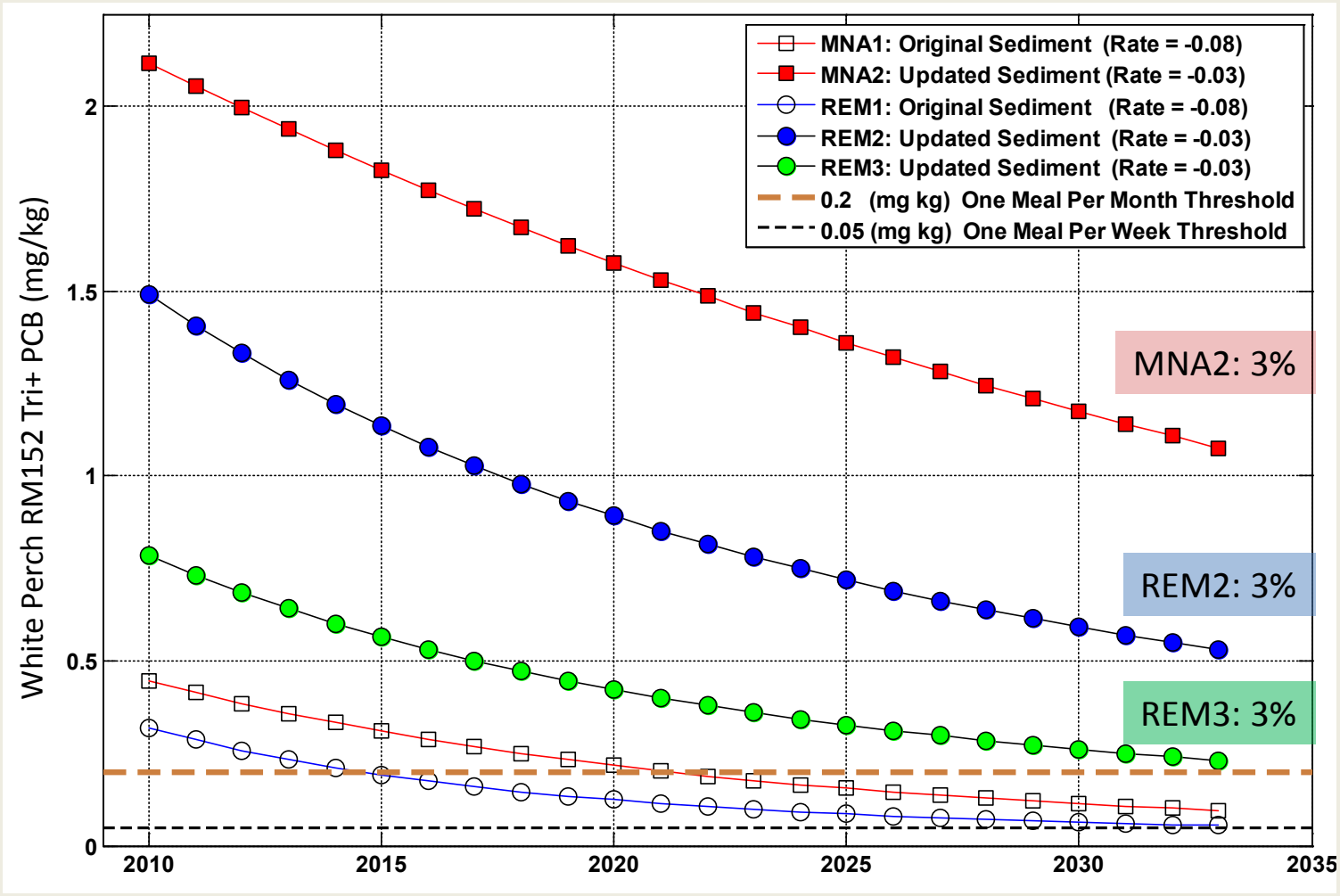


Remedial Action Objectives
Human Health

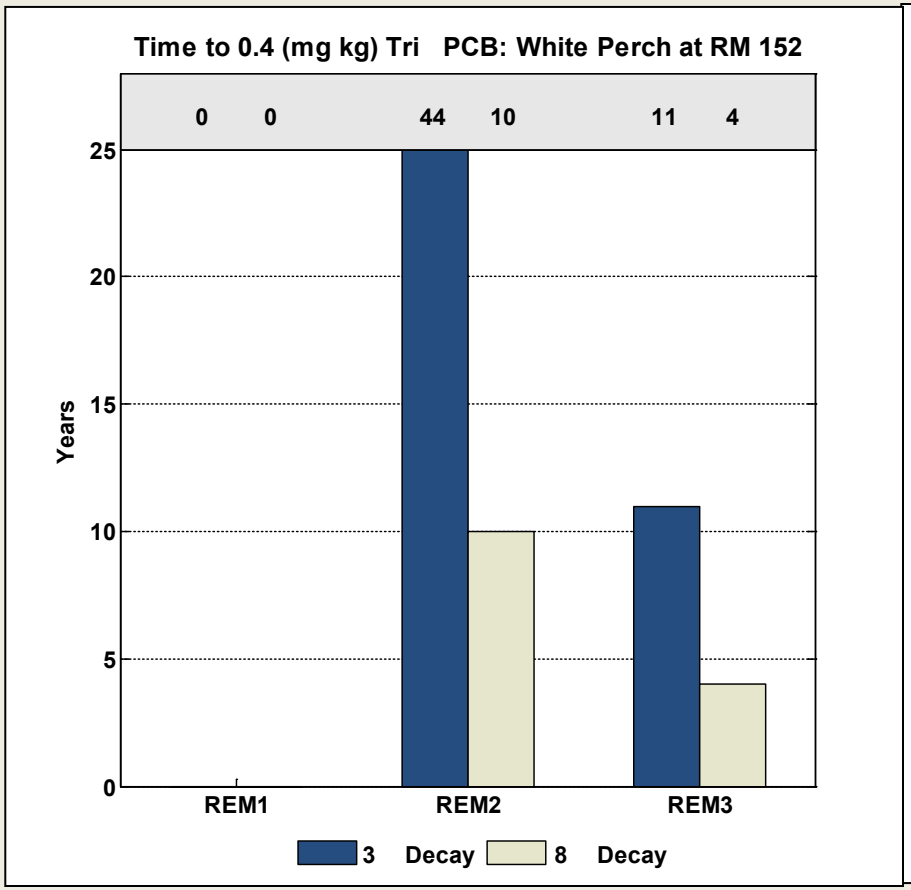
- ***Reduce the cancer risks and non-cancer health hazards for people eating fish from the Hudson River by reducing the concentration of PCBs in fish.***
  - 0.05 mg/kg PCBs in fish fillet, one half-pound meal per week
  - 0.2 mg/kg PCBs in fish fillet, one half-pound meal per month
  - 0.4 mg/kg PCBs in fish fillet, one half-pound meal every 2 months

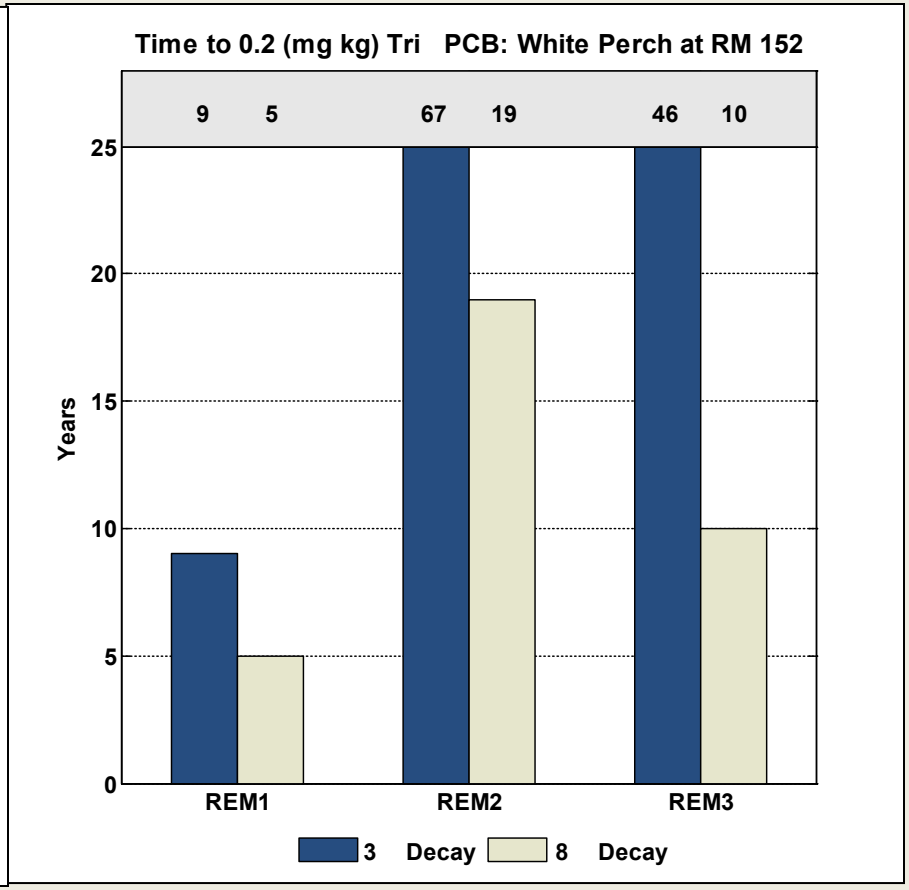
"…the Remediation Goal of 0.05 mg/kg also is expected to be attained in the majority of the Lower Hudson River, due to the lower initial concentration of Site-related PCBs in the Lower Hudson compared to the Upper Hudson." (USEPA 2002)

Emulated Model Projections for MNA and Remedial Scenarios

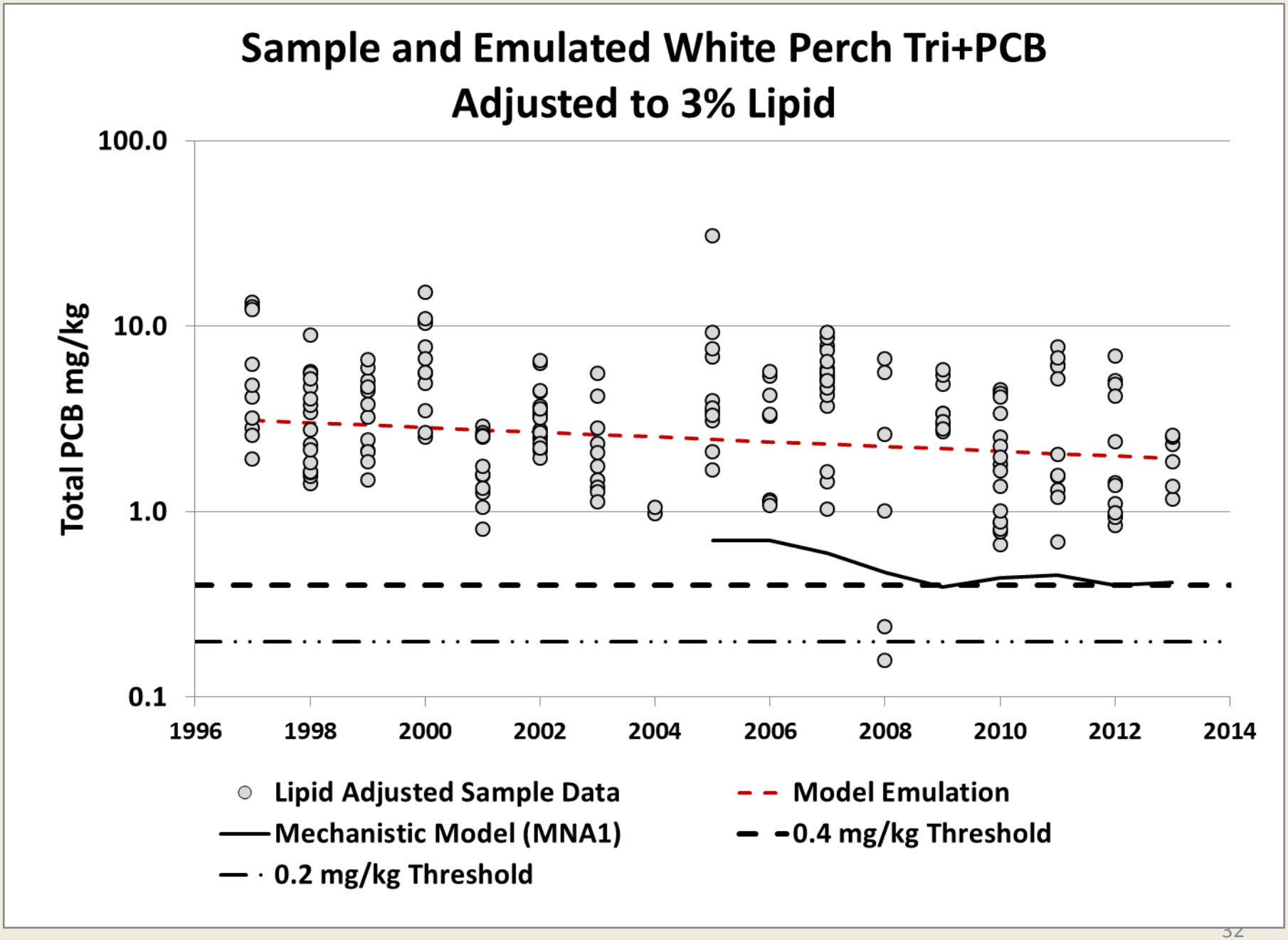


Model Emulation: Post Remediation Years to 0.4 and 0.2 ppm PCB Thresholds





- REM1: Original model initial projected sediment concentrations for selected remedy in 2010
  REM2: Emulated model for selected remedy with updated sediment concentrations
- REM3: Emulated model for revised remedial scenario with updated sediment concentrations


Sample and Emulated White Perch Tri+PCB
Adjusted to 3% Lipid
Total PCB mg/kg
100.0
10.0
1.0
0.1
1996
1998
2000
2002
2004
2006
2008
2010
2012
2014
Lipid Adjusted Sample Data
Model Emulation
Mechanistic Model (MNA1)
0.4 mg/kg Threshold
0.2 mg/kg Threshold

Summary: Model Emulation

- **Application to Hudson River**
  - Reproduced mechanistic model projections of sediment, water and fish PCBs under MNA and the selected remedy
  - Enabled application of updated sediment concentrations and estimated rate of exponential decrease to re-visit temporal projections of LHR fish tissue concentrations
- **Other Advantages**
  - Statistical uncertainty evaluations
  - More accurate model calibration and validation

Summary: Hudson River Sediment and Fish

- Recovery of UHR sediment surface much slower than predicted
- Recovery of LHR fish much slower than original projections
- Applying an enhanced remedy (eg., REM3) would reduce time to achieve PCB thresholds in fish, but still longer than originally predicted for the selected remedy

Use of Models in Decision-Making

- Overestimation of the rate of natural recovery in sediment minimizes difference between remedial alternatives
- Accurate estimation of the rate of natural recovery during RI/FS is essential for comparisons of alternatives
- Without baseline sediment data, relative comparisons of remedial alternatives may be misleading
- Model emulation can be a useful tool in reducing and understanding uncertainty

Conclusions

- Original mechanistic models used were overly optimistic about the rate of recovery of surface sediment under MNA and the selected remedy
- Attainment of Remedial Action Objectives for fish in the LHR will take much longer than predicted
- Additional removal of PCB-contaminated sediment in the UHR needed to achieve reductions in LHR fish PCBs anticipated in the ROD

Estimating Temporal Trends in Sediment

- Why are temporal decay rates for surface sediment overstated?
- What can we do to more accurately estimate rate of recovery in surface sediment?

Why Were Temporal Decay Rates Overstated?

- What factors contribute to the overestimation of rate of recovery?
- Design recommendations for sediment sampling to determine rate of natural recovery in surface sediment concentrations

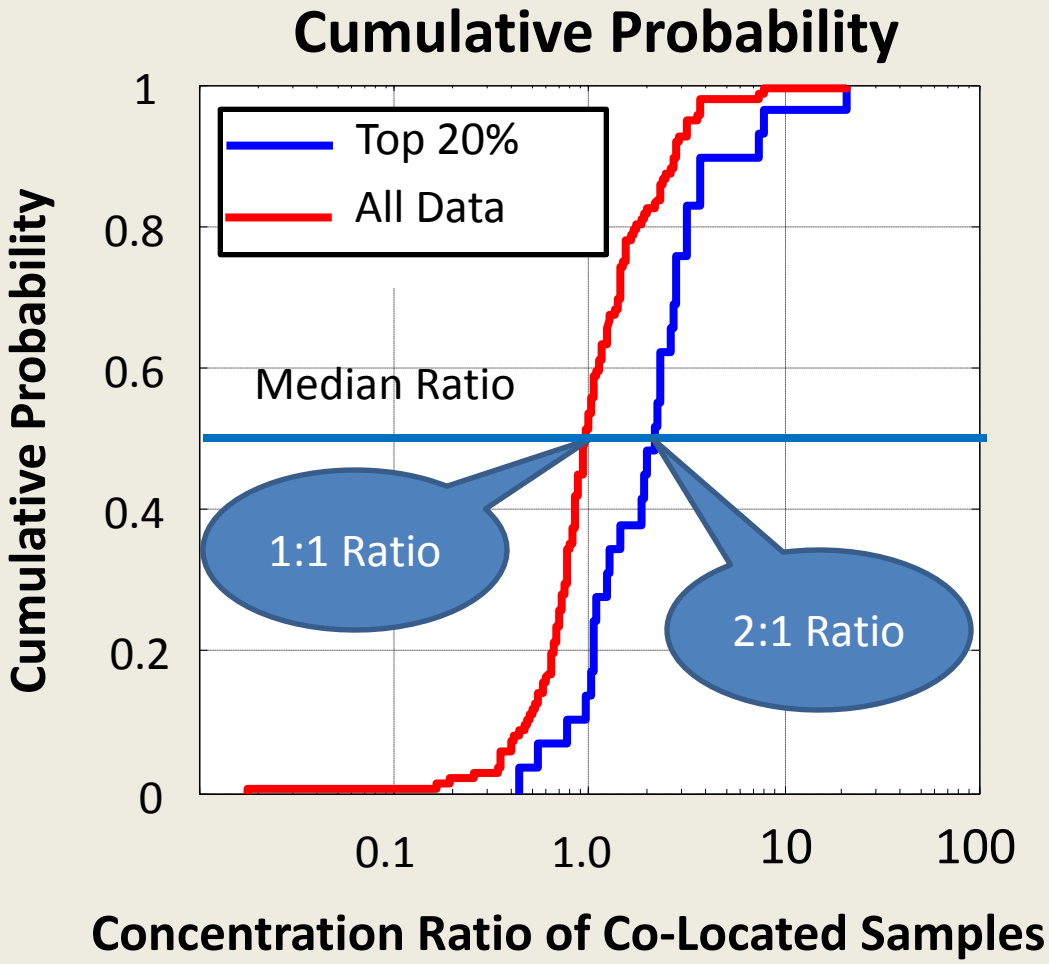
Why Were Temporal Decay Rates Overstated?

- Sedimentation rates in high resolution cores
  - Not all High-Res cores can be dated
  - Those that can be dated are in quiescent areas, not representative of the majority of the study area
  - May bias estimates toward higher sedimentation rates
- Comparison of surface concentrations between time steps
  - RI sampling programs were biased toward higher concentrations
  - Subsequent sampling also biased toward these areas
  - Assumption that trends are easier to detect in high concentration areas

Testing Trend Estimation With Biased Sampling

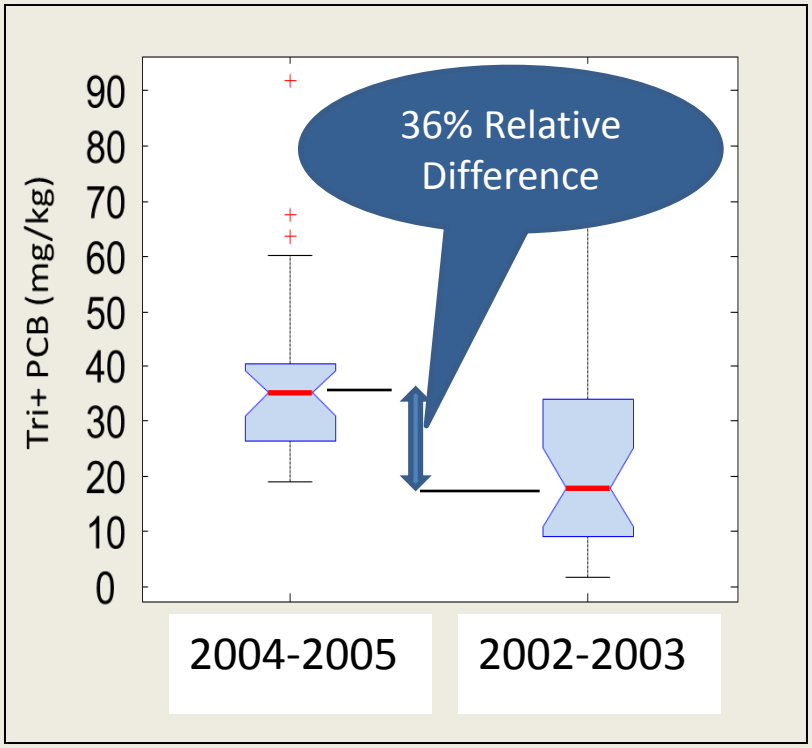
- Used paired co-located surface sediment samples from 136 locations throughout the Upper Hudson collected in 2002-2003 and 2004-2005
- Co-locates within 10 feet of initial sample compared
  - Samples from the upper 20th percentile from 2004-2005
  - Compared with co-located sample from 2002-2003
- Any estimated declines would be artifacts of biased sampling

Distribution of Sample-Pair Ratios



- Median Ratio for all data is 1:1
- Median Ratio for preferentially selected top 20% is ~2:1
- Comparison of secondary sample at locations of top 20% of first sample virtually guarantees apparent decreasing temporal trends

Paired-Sample Comparison Results



- Median concentration for the upper 20th percentile of 2004-2005 sample distribution is 36% higher than the median for paired samples collected 1-3 years earlier.
- Result is an artifact of the biased sampling used to obtain the test set.

"All Models are Wrong, Some are Useful"

- For a decision-maker, useful models provide the ability to discriminate differences in outcome for an array of alternatives
- How do you know if model is useful?
- Need good data, including data for baseline conditions and temporal rate of change in surface sediment concentrations that are representative of the area of concern

Design Recommendations for Sediment Temporal Trend Monitoring Plan

- Incorporate trend monitoring early in site assessment
- Use unbiased sampling procedures
  - Identify important strata boundaries at the outset of the monitoring program
  - Determine sample size using variability of existing data to quantify temporal decay rates with adequate precision for comparisons of remedial alternatives
- Monitor same locations at ~ 5 year intervals
  - Use paired and repeated measures statistical analyses within strata to evaluate local trends
  - Combine results across strata to develop global statements about trend in overall average (SWAC).
  - Interpolation is unnecessary because sampling is unbiased


Questions?

Attachment Q

Recommendations on the Use of Available Data to Evaluate Remedy Effectiveness

Recommendations on the Use of Available Data to Evaluate Remedy Effectiveness

Jay Field & Lisa Rosman

EPA Five Year Review Team

September 15, 2016

Recommendations for Using Available Data to Evaluate Expected Recovery Prior to Dredging

Two Approaches:

- Compare PCB concentrations in sediment, water and fish to expected concentrations prior to dredging
- Compare rates of recovery in sediment, water and fish during Monitored Natural Attenuation (MNA) and source control period (EPA August 17 presentation focused on this approach)

Outline of Presentation

- Available data and issues for consideration
- Compare data collected prior to dredging to model-predicted concentrations
- Evaluate rate of recovery compared to model predictions
- Potential implications and recommendations

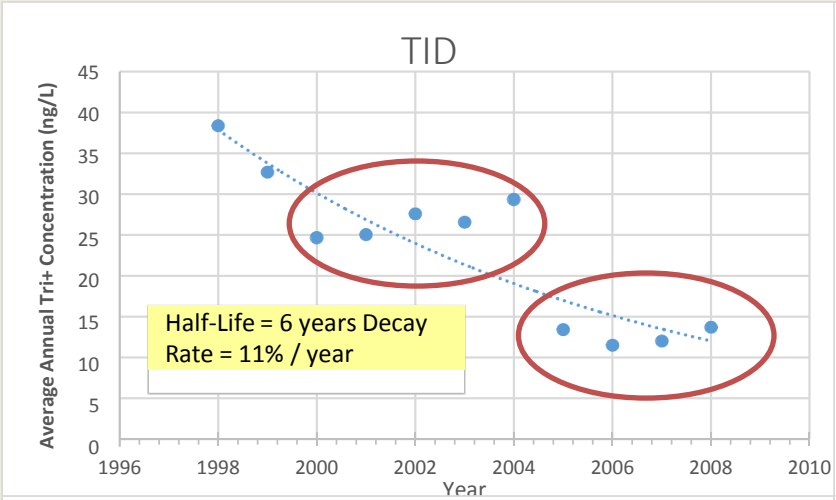
Data Considerations

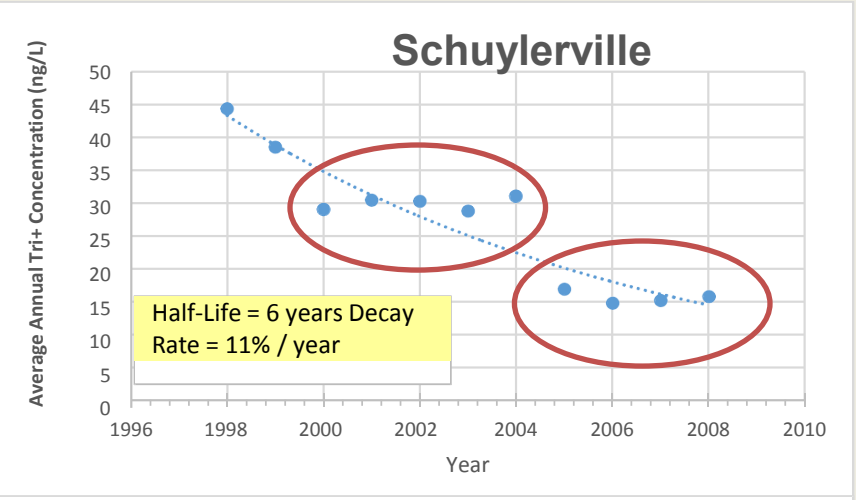
- MNA period includes major source control
  - Are rates of recovery influenced by source control during the pre-dredge MNA period?
- PCBs in fish fillets biased low to unquantified degree
  - Some uncertainty in year when fillet protocol changed
  - What is the impact of the change in fillet protocol after 2006 on fish fillet PCB concentrations and estimated rate of recovery?
- All analyses shown in this presentation use data at reported value (no homologue adjustment)

"MNA" Period Includes Source Control

- Models incorporated >6-fold reduction in PCB load into Thompson Island Pool between 1998 and 2005
  - EPA Responsiveness Summary: "The upstream source control is characterized in the HUDTOX model by assuming an upstream boundary water column Tri+ PCB load of 0.16 kg/day from 1998 through 2004, followed by a step-down reduction to 0.0256 kg/day on January 1, 2005."

Water Column Forecasts (Model Output, yr 2000)





Water model forecast for Thompson Island Dam and Schuylerville during source control upstream of Thompson Island Pool between 1998 and 2005.

Regression line spans major source control period in model forecast.

Modified from EPA 2016-08-17 Hudson River FYR Presentation

Using Fish Monitoring Data to Evaluate Model Predictions

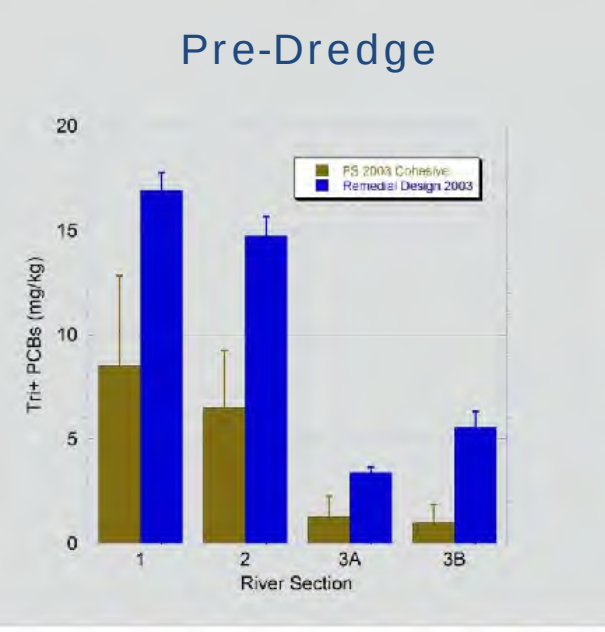
- Post-2006?, fish fillet data collected by GE was biased low due to GE's change in fish processing protocol from "NYS STD (with rib)" to "rib-off"
- Based on a 2014 study of black bass, EPA concluded that lipid-normalized "NYS STD" and "rib-off" for black bass are comparable for evaluating long term trends.
- The degree of low bias for fillets of other fish species (e.g., white perch, yellow perch, brown bullhead, striped bass, channel catfish) is unknown.
- Including post-2006 data can contribute to increased apparent rates of recovery.
- We recommend not using the post-2006 biased low data for trend analysis

PCBs in Post-2006 GE Fish Fillets Biased Low

- EPA preliminary report used 2 approaches to compare rib-in to rib-out fillets in Black Bass
- Regression Approach:
  - "The TPCB regression suggests an approx. 16% bias (16% more TPCB in NYS STD fillets) with a range of 11-21%."
  - "The LPCB regression suggests an approx. 8% bias (8% more LPCB in NYS STD fillets) with a range of 6-10%."
- PCB Ratio Approach:
  - TPCB (wet weight) ~ 75% higher PCB concentration for NYS STD fillets
  - Comparable LPCB ratio is ~22% higher with range of 13-31%
    - ~40% of the rib-on fillets were > 20% higher; ~20% were >=40% higher.

Results are inconsistent with EPA's conclusion "that the lipid normalized data from this period are comparable for evaluation of long term trends."

Surface Sediment PCBs: Model vs Measured in 2003



Section average Tri+ PCBs (ppm) in surface sediments from the SSAP data exceeded the mean by a factor of 2-3 and the upper bound of model predictions

Olive Green Bar: Model Section average and upper bound for cohesive sediments

Blue Bar: SSAP Remedial Design data

Surface sediment represents top 4 cm for model and top 2 inches (5 cm) for remedial design data. River sections 2 & 3 represent cohesive sediments only.

PCB Loads to LHR Higher than Predicted Prior to Dredging

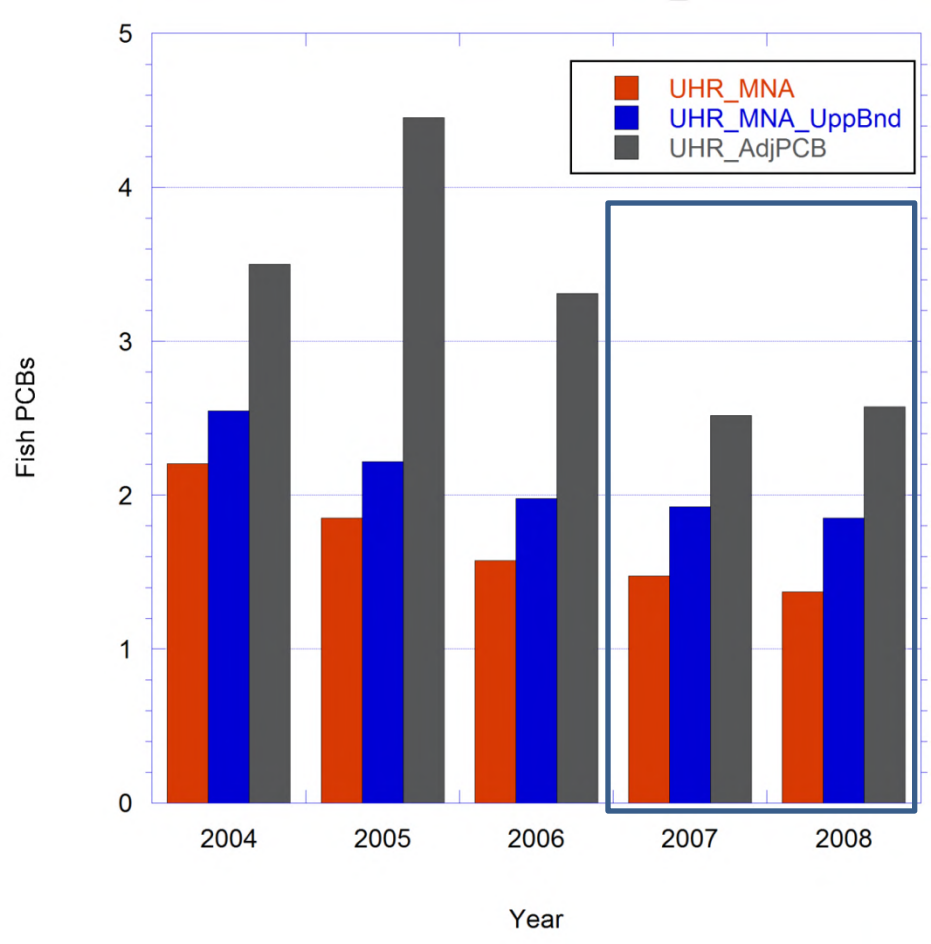
- PCB loads from the upper to the lower river were 3-fold higher than expected prior to the start of dredging and showed little evidence of decline (EPA March 2010; Hydroqual 2010).

USEPA, 2010. Hudson River PCBs Site EPA Phase 1 Evaluation Report, Prepared for: US Environmental Protection Agency, Region 2 and US Army Corps of Engineers, Kansas City District, Prepared by:The Louis Berger Group, Inc., March 2010.

Hydroqual 2010. Evaluation of PCB Concentrations Measured in the Hudson River near Waterford, New York. May 31, 2010, U Appendix A-9. Reports from additional Modeling exercises utilizing the CARP models

UHR Fish: Model vs Data

Model projections of Species-weighted and Section-weighted average and upper bound PCBs (ppm) compared to data from 2004-2008



Total PCBs in fish (2004-2006) ~ 2x higher than model predicted

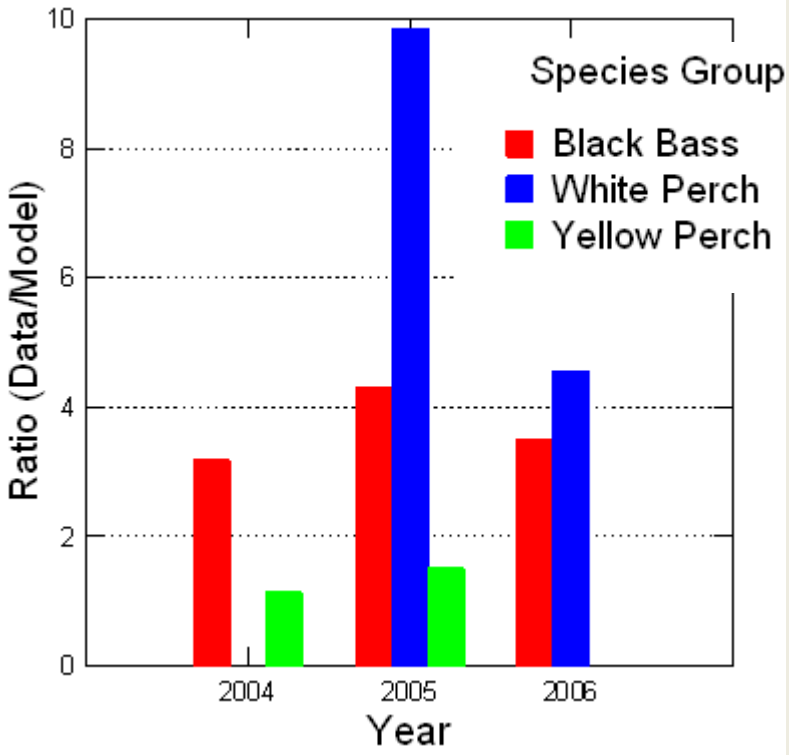
Total PCBs in 2007-2008 fish have unquantified low bias, but also higher than model predicted PCBs

NOTE: PCB concentrations adjusted for lipid content used in EPA FISHRAND model

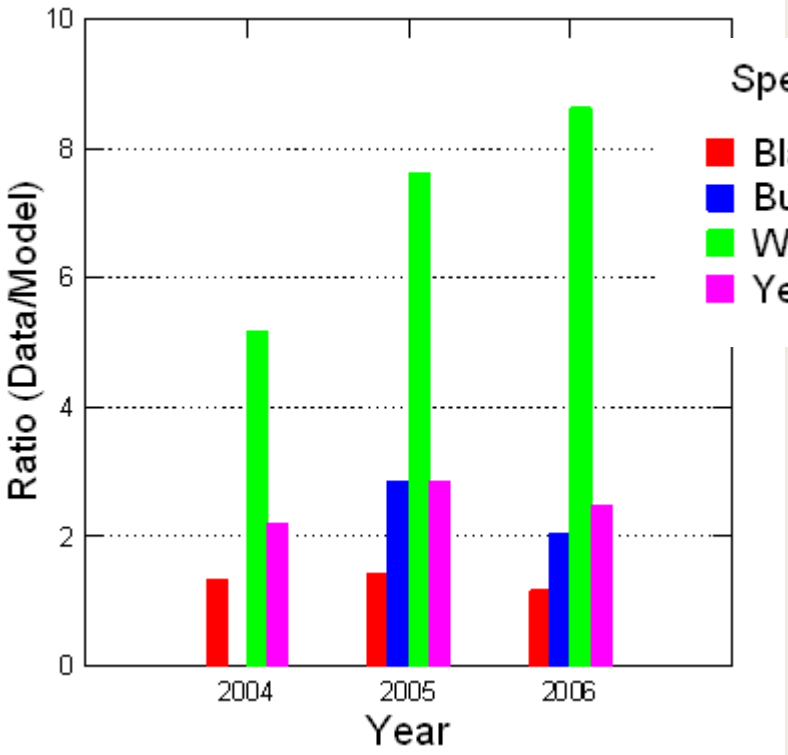
LHR Fish Prior to Dredging

Ratio of Measured PCBs to Model Predictions

Albany/Troy



Catskill



LHR fish PCBs > 2x model predictions

Ratio (Data/Modeled) < 1 Measured PCBs less than model predictions

Ratio (Data/Modeled) > 1 Measured PCBs greater than model predictions

Estimates of Rate of Recovery (Decay Rate)

- Surface sediment concentrations from SSAP (~2003) compared to GE 1991 transect survey (only available pre-dredging surface sediment data for RS2 & RS3)
- PCB load to LHR during MNA and source control period
- Fish PCBs in UHR and LHR for primary species and key long-term monitoring stations (part of baseline monitoring plan) between 1997 or 1998 and 2006

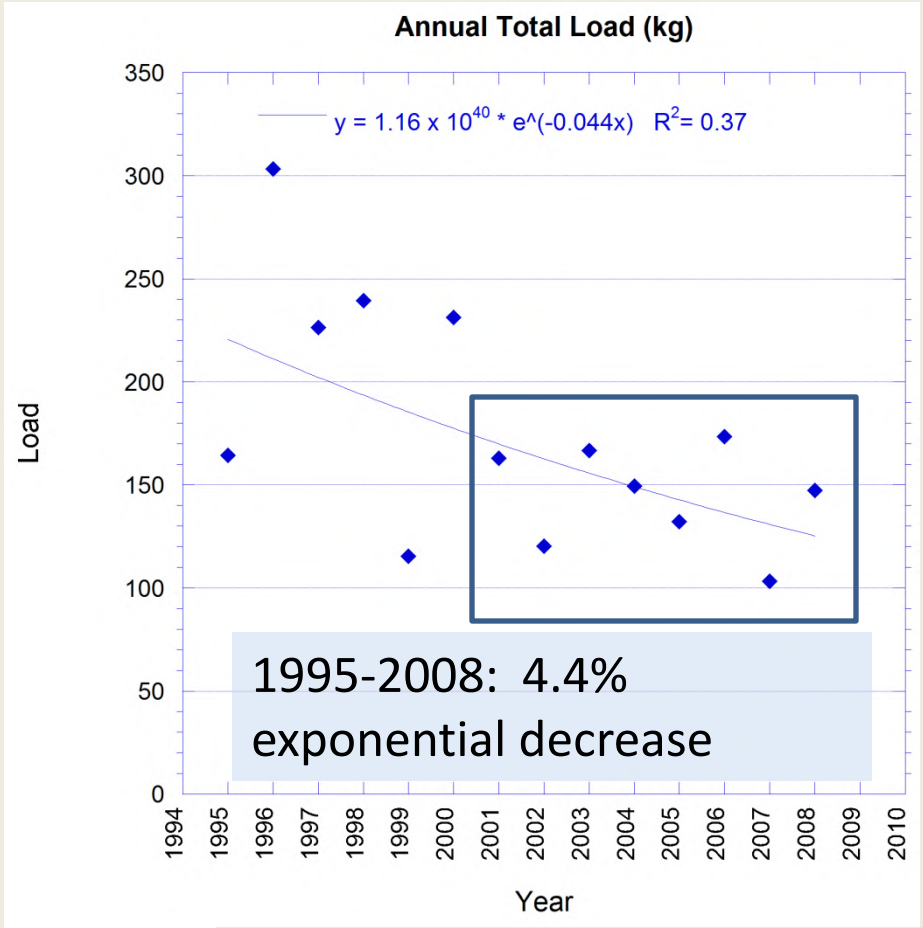
Estimated Pre-Dredge Decay Rate in Surface Sediment

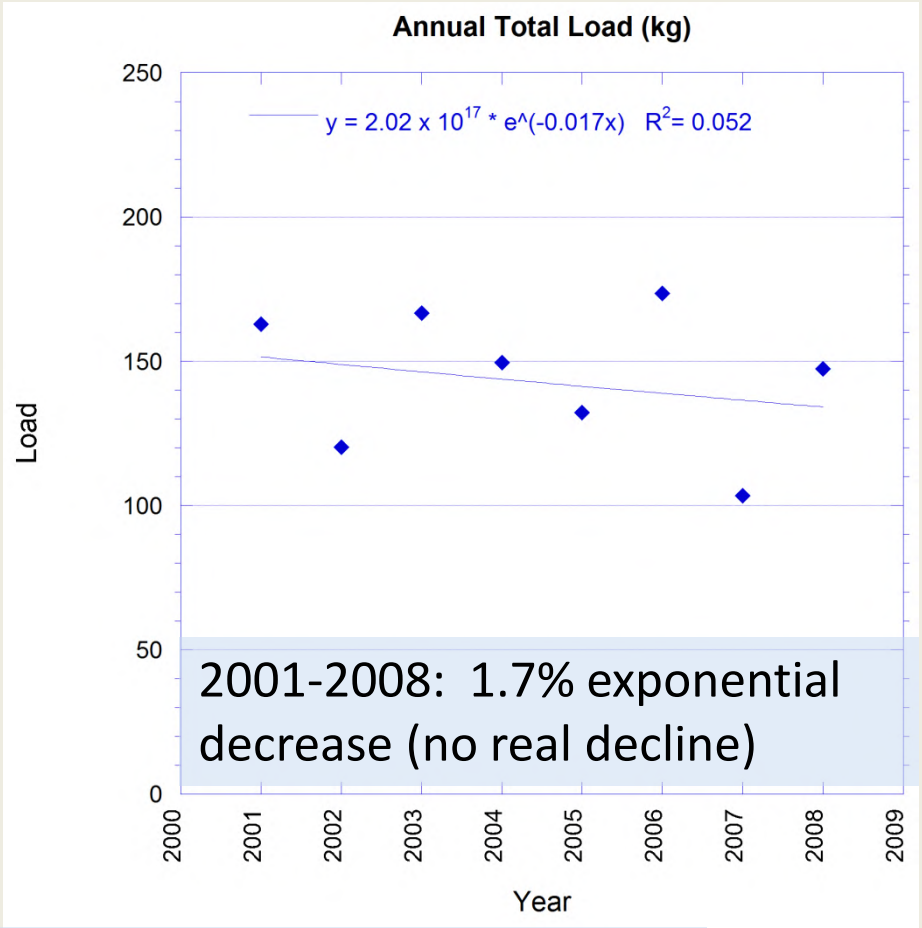
Model Subsection	GE 1991 (ppm)	SSAP 2003 (ppm)	Calculated Exponential Decay Rate
1	20	16.9	1.4%
2	18	15.7	1.7%
3A	4.3	3.4	2.0%
3B	5.7	5.6	0.1%
Average			1.3%
95% CI			-0.1% – 2.6%

Rate of sediment recovery much slower than 7-9% in modeling projections

Measured PCB Load to LHR (MNA) 1995-2008 and 2001-2008

(NOAA Analysis of Load data provided by EPA)





Note: MNA predicted load in 2008 was ~50 kg

Pre-Dredge Fish Recovery Rate (1997/8-2006)

Station	Species Group	1997_2006	1998_2006	Average Decay Rate	Station Average
Thompson Island Pool	Black Bass	-0.101	-0.120	11.0%	
	Bullhead	-0.045	-0.058	5.1%	7%
	Yellow Perch	-0.133	-0.150	14.1%	
	Pumpkinseed (age 1+)	0.048	0.034	-4.1%	
Stillwater	Black Bass	-0.044	-0.075	6.0%	
	Bullhead	-0.056	-0.114	8.5%	6%
	Yellow Perch	-0.019	-0.044	3.2%	
	Pumpkinseed (age 1+)	-0.059	-0.083	7.1%	
Albany/Troy	Black Bass	-0.047	-0.046	4.6%	4%
	White Perch	-0.038	-0.018	2.8%	
	Yellow Perch	-0.041	-0.040	4.0%	
Catskill	Black Bass	-0.081	-0.109	9.5%	
	Bullhead	-0.043	-0.043	4.3%	
	White Perch	0.056	0.041	-4.9%	3%
	Yellow Perch	-0.013	-0.013	1.3%	
	Pumpkinseed (age 1+)	-0.037	-0.037	3.7%	
Poughkeepsie	Black Bass	-0.067	-0.067	6.7%	
	Bullhead	-0.015	-0.015	1.5%	
	White Perch	-0.007	-0.007	0.7%	3%
	Yellow Perch	-0.012	-0.012	1.2%	
	Pumpkinseed (age 1+)	-0.056	-0.056	5.6%	

Decay rate may differ with interval selected

UHR data show highly variable decay rates

LHR decay rates mostly <5%

Decay Rate	Color Coding
>8%	
4-8%	
<4%	

Negative decay rate indicates no change or increase in PCBs

Summary: Pre-Dredging PCB Concentrations and Rate of Recovery

- Model-Data Comparisons
  - Surface Sediment PCBs ~2-3x higher than predicted
  - PCB Load to LHR in 2008 ~3x higher than predicted
  - UHR Fish PCBs ~2x higher than predicted
  - LHR Fish PCBs >2x higher than predicted
- Rate of Recovery
  - Estimated pre-dredge sediment rate of recovery < 3%
  - Pre-dredging PCB load to LHR shows little evidence of decline between 2001 and 2008 (post major source control)
  - UHR fish highly variable with many species/locations < 8% (6-7% station averages)
  - LHR fish mostly < 5% (3-4% station averages)

Why PCB Concentration in Sediment & Fish are the Most Relevant Metrics

- ROD used the number of years to reach human health and ecological risk concentration based thresholds in comparison of remedial alternatives as a basis for selection of the remedy.
- Higher than expected post-dredging surface sediment concentrations over model predictions likely extends time to reach risk thresholds in fish PCBs.
- Time to recovery is determined by both the magnitude of the post-dredging sediment concentration and the rate of recovery.
- Given higher-than-expected pre- and post-dredging concentrations, a higher percent reduction or longer time is required to achieve the expected concentrations in fish

Additional Information

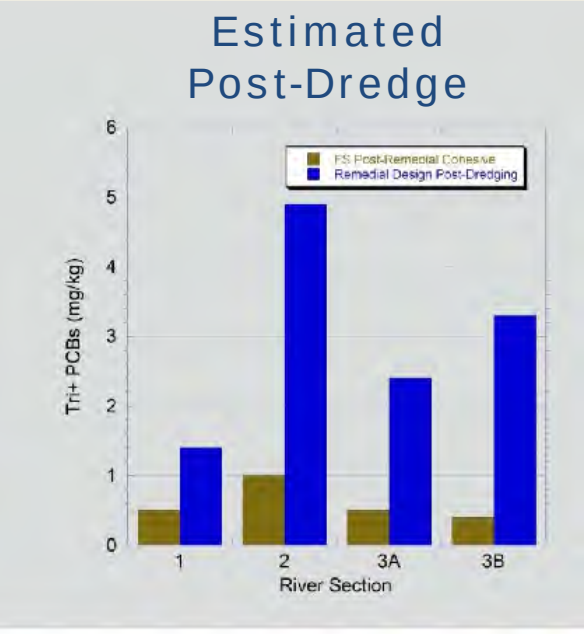
- More PCBs in UHR than the ROD anticipated
  - Mass removed greater than expected
  - Surface sediment post-dredging estimated to be much higher than the models used in the ROD predicted

Mass of PCBs Removed

- Mass of PCBs removed (~65%) was more than 2X the original estimate (150,000 lbs) within the same dredge footprint, which implies that a greater mass of PCBs remain in the river post-dredging than EPA originally expected would be removed by the remedy.
- Some of the underestimate was due to the amount of PCBs found at depth, but PCBs in the surface sediment were also higher, more widespread, and shallower than expected.
- Not reasonable to assume that the increase in mass was confined to within the dredge footprint
- More mass and higher PCBs remaining than expected post-remedy contributes to on-going risk

Surface Sediment PCBs: Model Predicted vs Estimated From SSAP Data

- Estimated post-remediation PCBs for the selected remedy were 3-5X higher than model predictions.
- Differences are greater for River Sections 2 & 3



Olive Green Bar: Model Section average and upper bound for cohesive sediments

Blue Bar: SSAP Remedial Design data

Surface sediment represents top 4 cm for model and top 2 inches (5 cm) for remedial design data

Implications

- ROD expected that the target cleanup levels for RS2 and RS3 would result in post-dredging surface sediment PCBs comparable to RS1
- Estimated post-dredging surface PCBs are ~5X higher than expected in RS2 and RS3 and ~3X higher than expected in RS1
- Using the EPA model projected 8% decay rate (equivalent to a 10 year half-life), achieving expected initial post-dredging sediment concentrations would be delayed by 25 years due to higher post-dredging surface concentrations. A slower rate of recovery would extend considerably the time to recovery.

Recommendations

- Source control is mostly complete and PCB load into the Thompson Island Pool should no longer influence the observed rate of MNA recovery. Going forward, use more realistic rates of recovery for MNA
- Change in fish processing protocol results in unquantified low bias in adult fish PCBs, which makes apparent recovery rates faster: recommend evaluating magnitude of effect in other species (see federal trustee 7/21/16 recommendations)
- Post-remedy concentrations are driven by both recovery rate and initial concentrations: should consider impact of both
- Develop and implement a robust sediment sampling plan to characterize the surface sediment concentrations to provide a strong basis for evaluation and prediction (see federal trustee 2/26/16 recommendations)

Attachment R

USEPA, Powerpoint: PCBs in Fish Tissues at the Hudson River PCBs Superfund Site

Community Advisory Group (CAG) Meeting
Hudson River PCBs Superfund Site
Schuylerville, NY, 30 October 2014

PCBs in Fish Tissues at the Hudson River PCBs Superfund Site:
Update on Results of Baseline and Remedial Action Monitoring (2004-2013)

Marc S. Greenberg, Ph.D.

U.S. EPA OSWER-OSRTI
Environmental Response Team
Edison, NJ

greenberg.marc@epa.gov

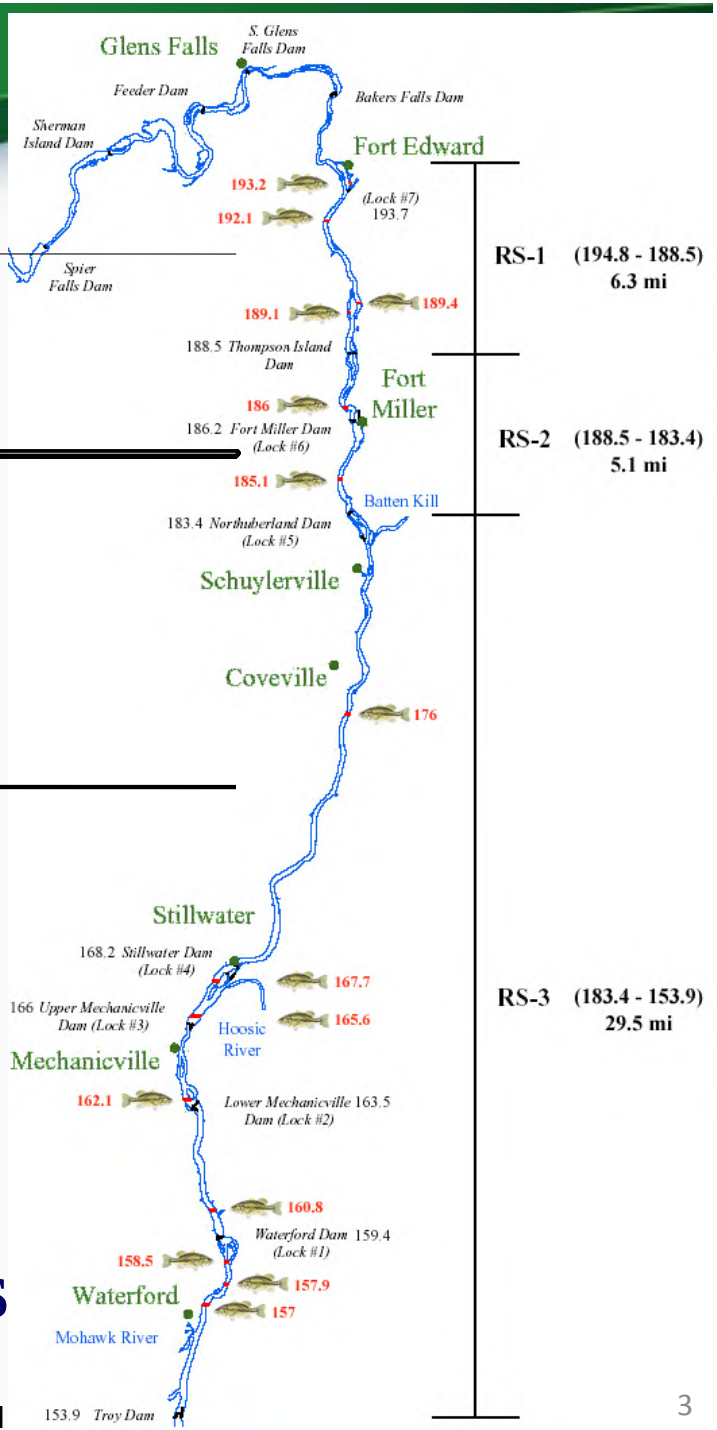
Hudson River PCBs SUPERFUND SITE

Background and Objectives

- Risk from fish consumption by humans and wildlife was the key driver for remediation
- Fish monitoring in the river since 1970s and will continue
- Since 2003: Baseline, remedial action, and post-remedy monitoring that was designed to provide statistical power to address both short- and long-term needs
  - Allows evaluation of annual (short term) changes *and* establishment of long-term trends
  - Allows documentation of interim risk reduction following the remedial action
  - We need to demonstrate that the remedy is moving toward, or achieving RAOs (remedy effectiveness)

Baseline, Remedial Action & Long Term* Fish Monitoring Plans for UHR

River Area	No. Spp. Groups	No. Indiv/Spp Groups	Total Samples
Feeder Dam	**4**	**20**	**80**
RS-1	**4**	**30**	**120**
RS-2	**4**	**25**	**100**
RS-3	**4**	**30**	**120**
Albany/Troy	**4**	**20**	**80**



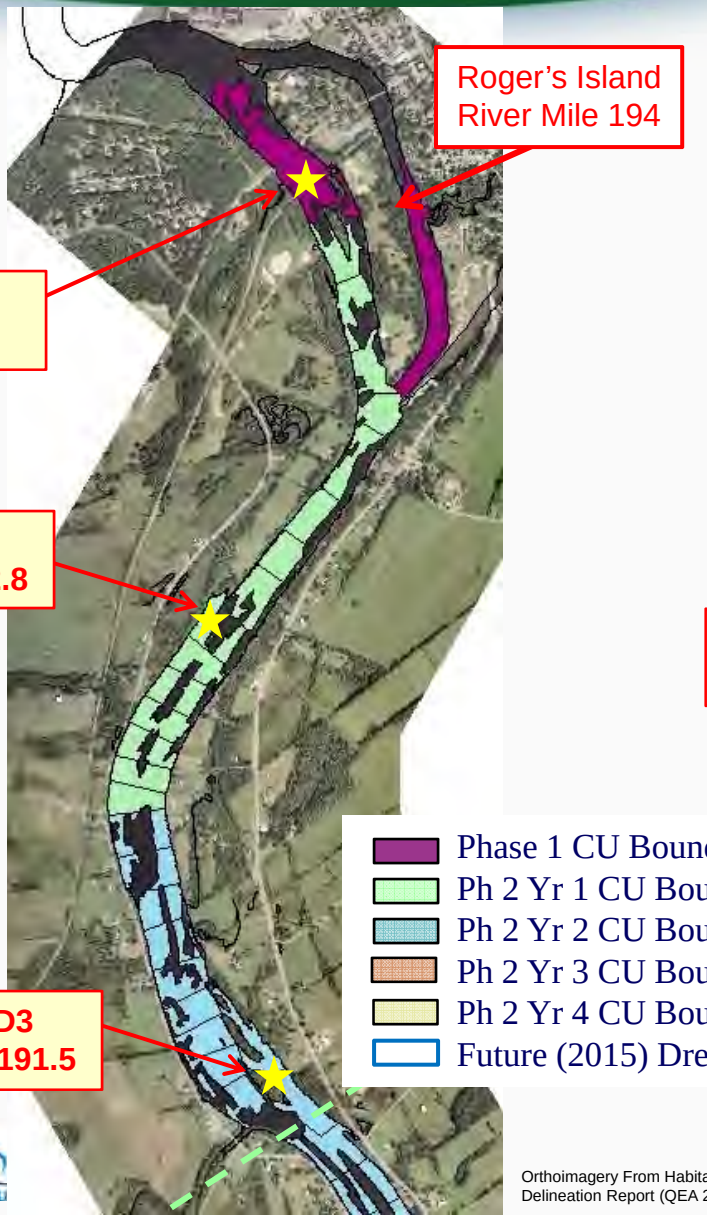
Four species/groups sampled ANNUALLY:

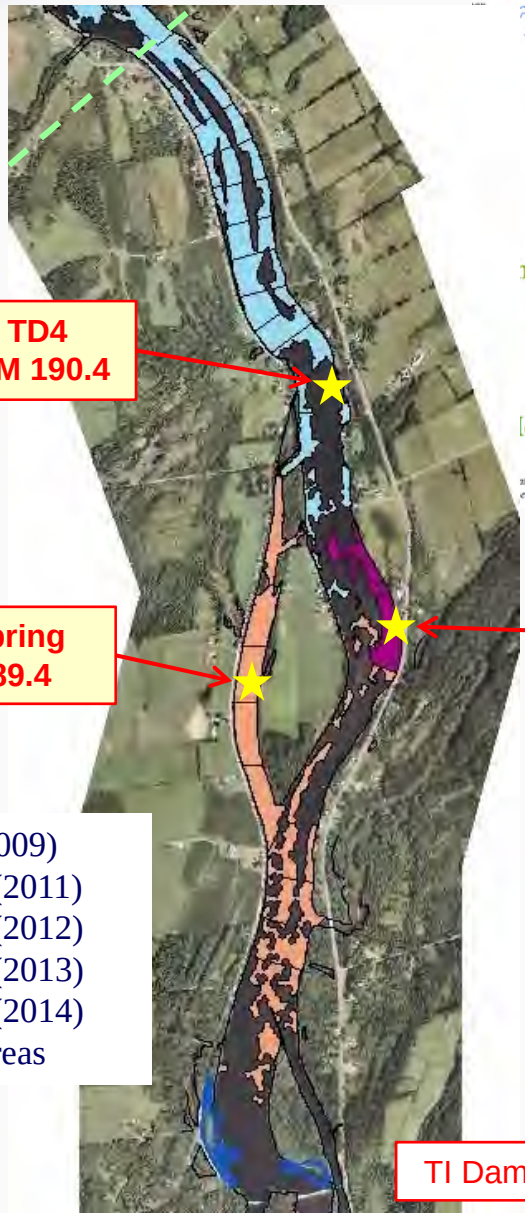
- Top-level pred: Blk Bass (LMB, SMB) SF
- Water col feeder: Perch (YP) SF
- Bottom-feeder: Bullhead (YB, BB) SF
- Yearling: Pumpkinseed WH

Annual composites of Forage Fish; n=10 per RS

* The LTMP may be modified after 3 years of OM&M

River Section 1 Fish Monitoring Stations and Dredging by Year



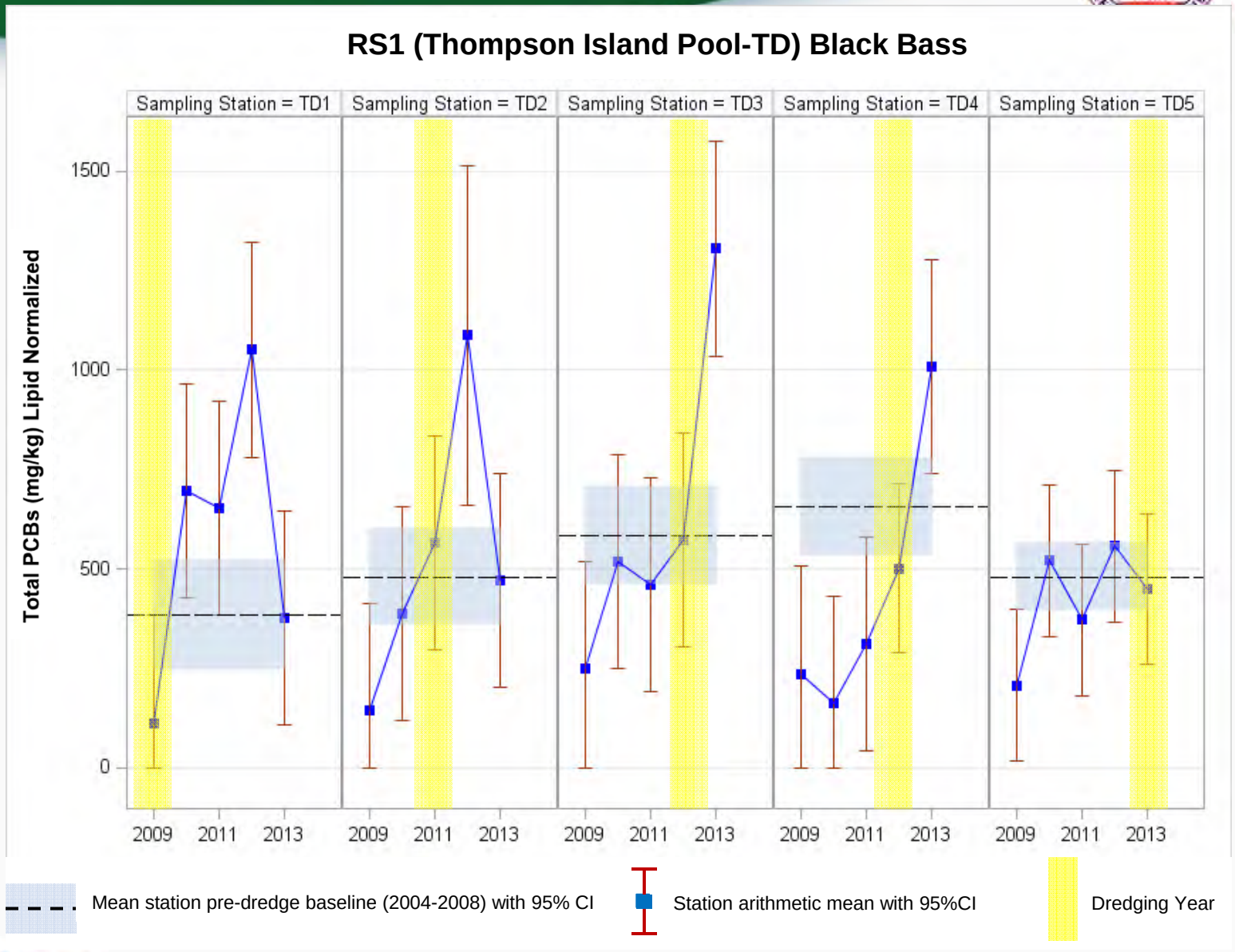


Inset Map, RAMP QAPP Anchor/QEA 2009

- Phase 1 CU Boundaries (2009)
- Ph 2 Yr 1 CU Boundaries (2011)
- Ph 2 Yr 2 CU Boundaries (2012)
- Ph 2 Yr 3 CU Boundaries (2013)
- Ph 2 Yr 4 CU Boundaries (2014)
- Future (2015) Dredging Areas

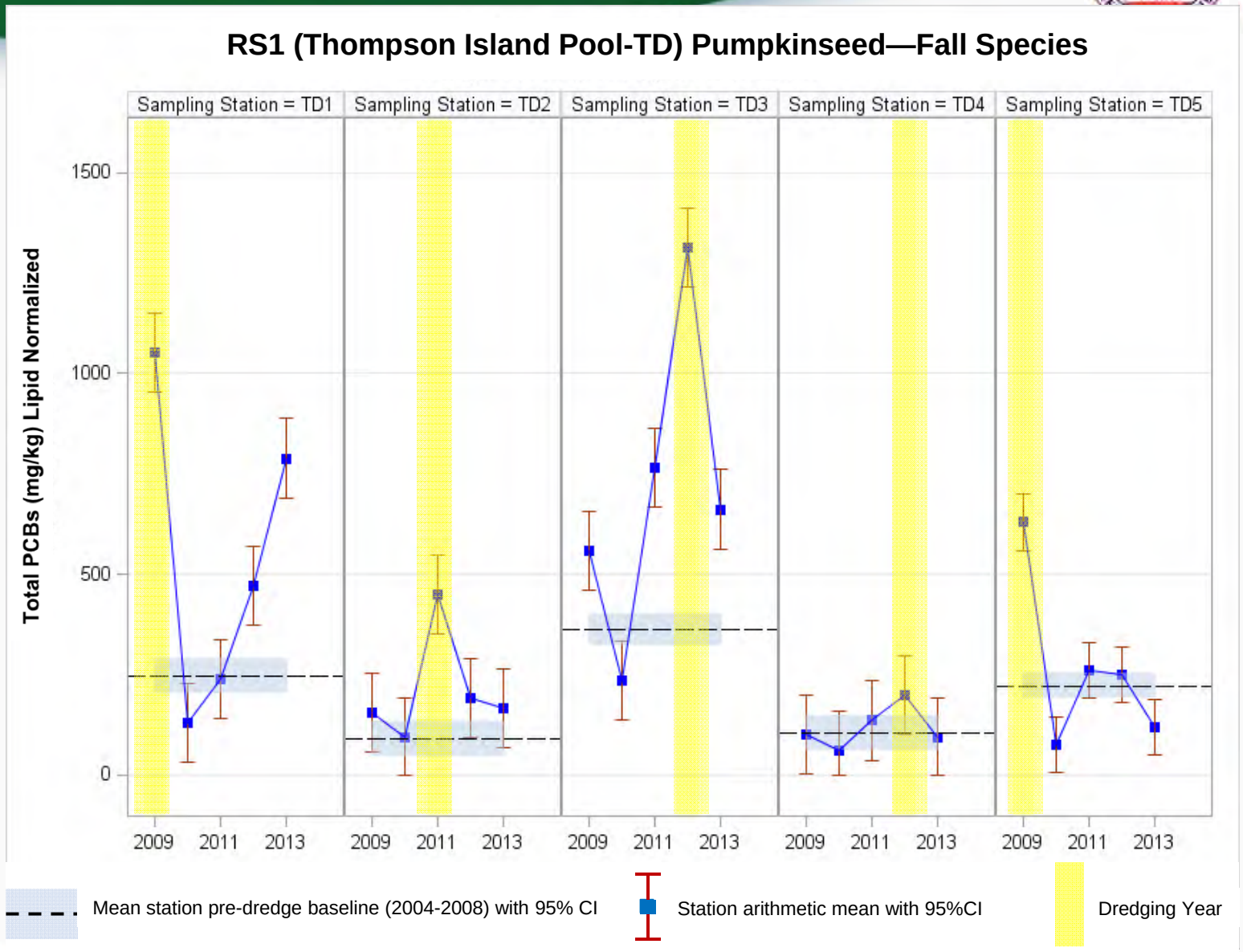
Orthoimagery From Habitat Delineation Report (QEA 2008)

Comparison of Baseline to 2009-2013



Hudson River PCBs SUPERFUND SITE

Comparison of Baseline to 2009-2013



Hudson River PCBs SUPERFUND SITE

River Section 2 Fish Monitoring Stations and Dredging by Year

TI Dam RM 188.5

ND1
RM 187

ND2
RM 186.4

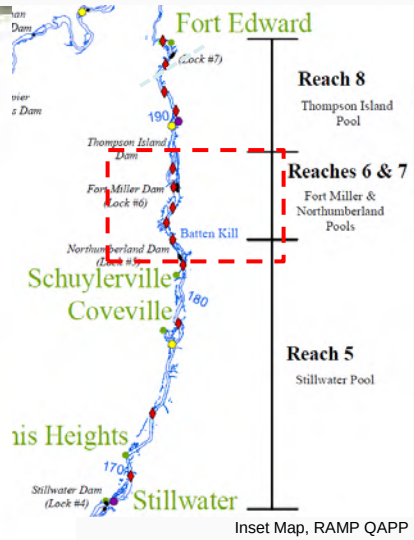
ND3
RM 185.4

ND5
RM 183.5

Northumberland Dam
RM 183.2

SW1
RM 181.8

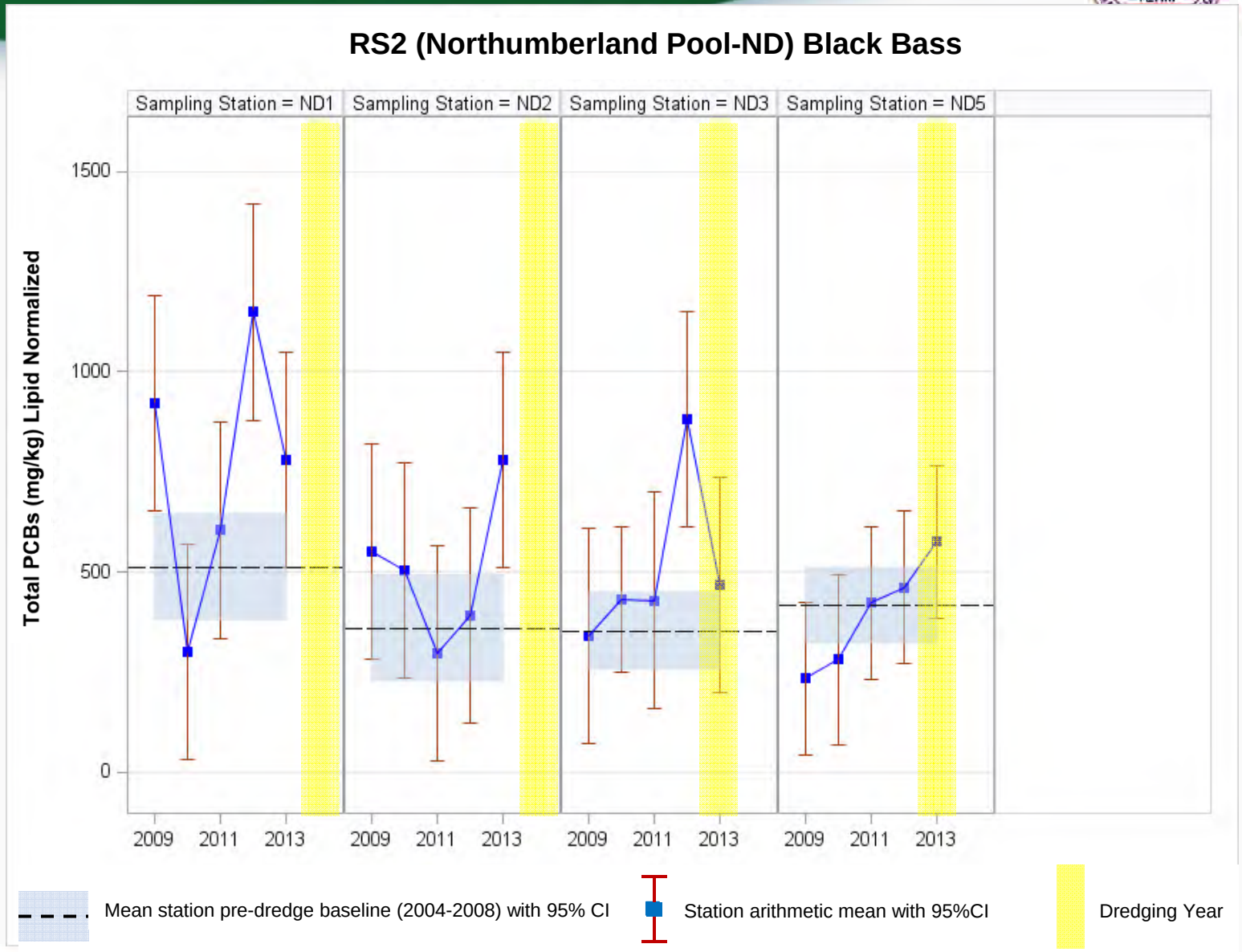
- Phase 1 CU Boundaries (2009)
- Ph 2 Yr 1 CU Boundaries (2011)
- Ph 2 Yr 2 CU Boundaries (2012)
- Ph 2 Yr 3 CU Boundaries (2013)
- Ph 2 Yr 4 CU Boundaries (2014)
- Future (2015) Dredging Areas



Inset Map, RAMP QAPP Anchor/QEA 2009

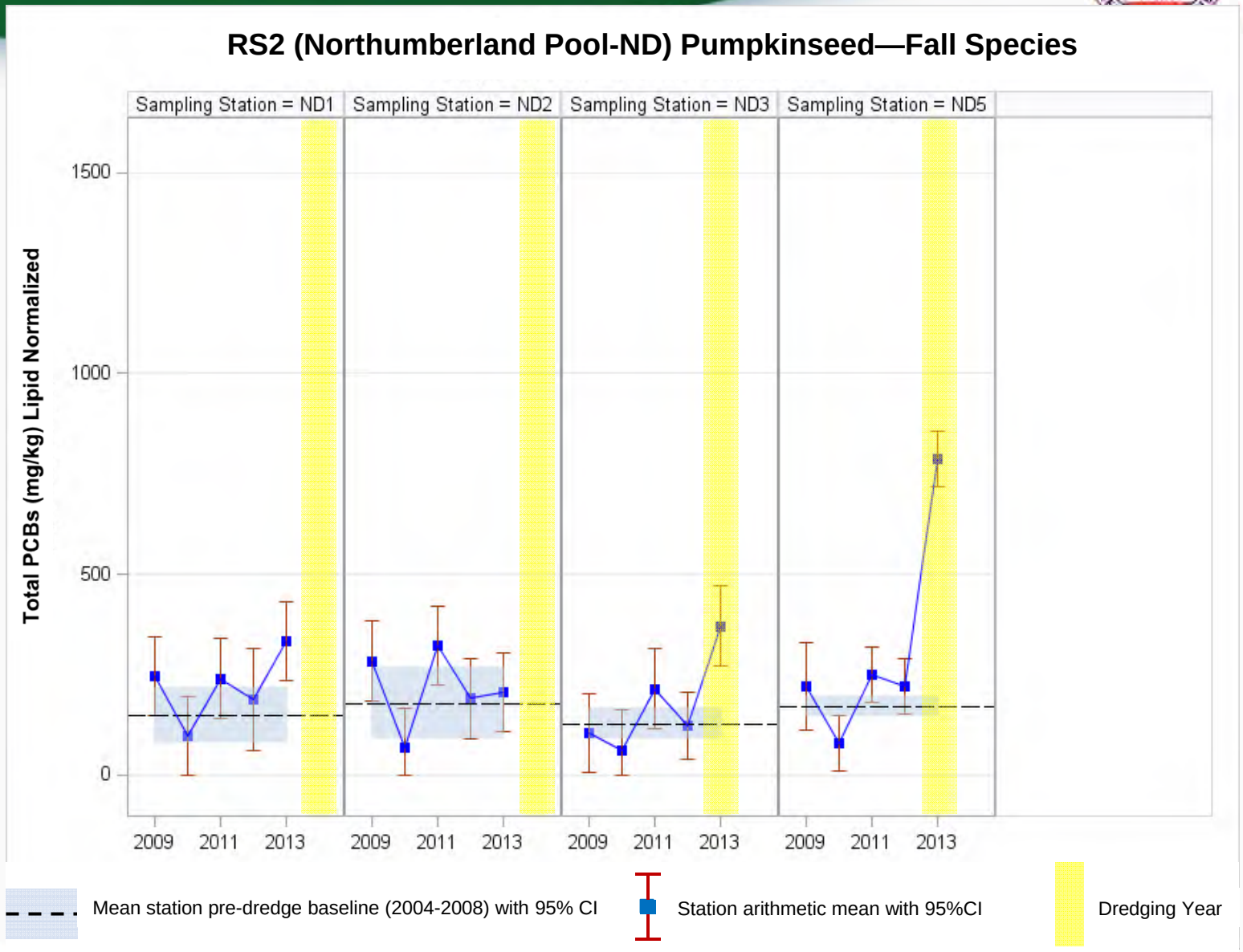
Orthoimagery From Habitat Delineation Report (QEA 2008)

Comparison of Baseline to 2009-2013



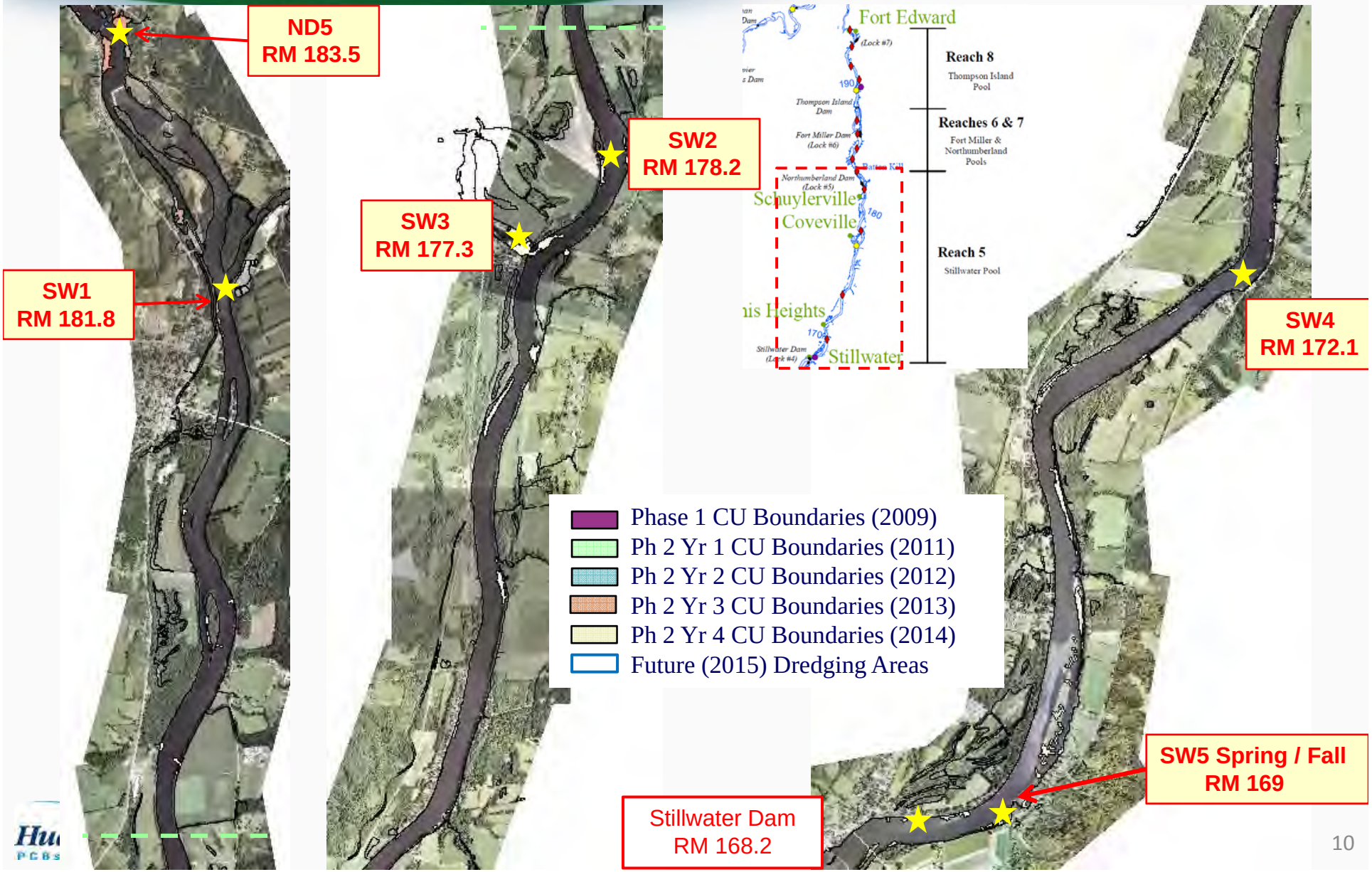
Hudson River PCBs SUPERFUND SITE

Comparison of Baseline to 2009-2013

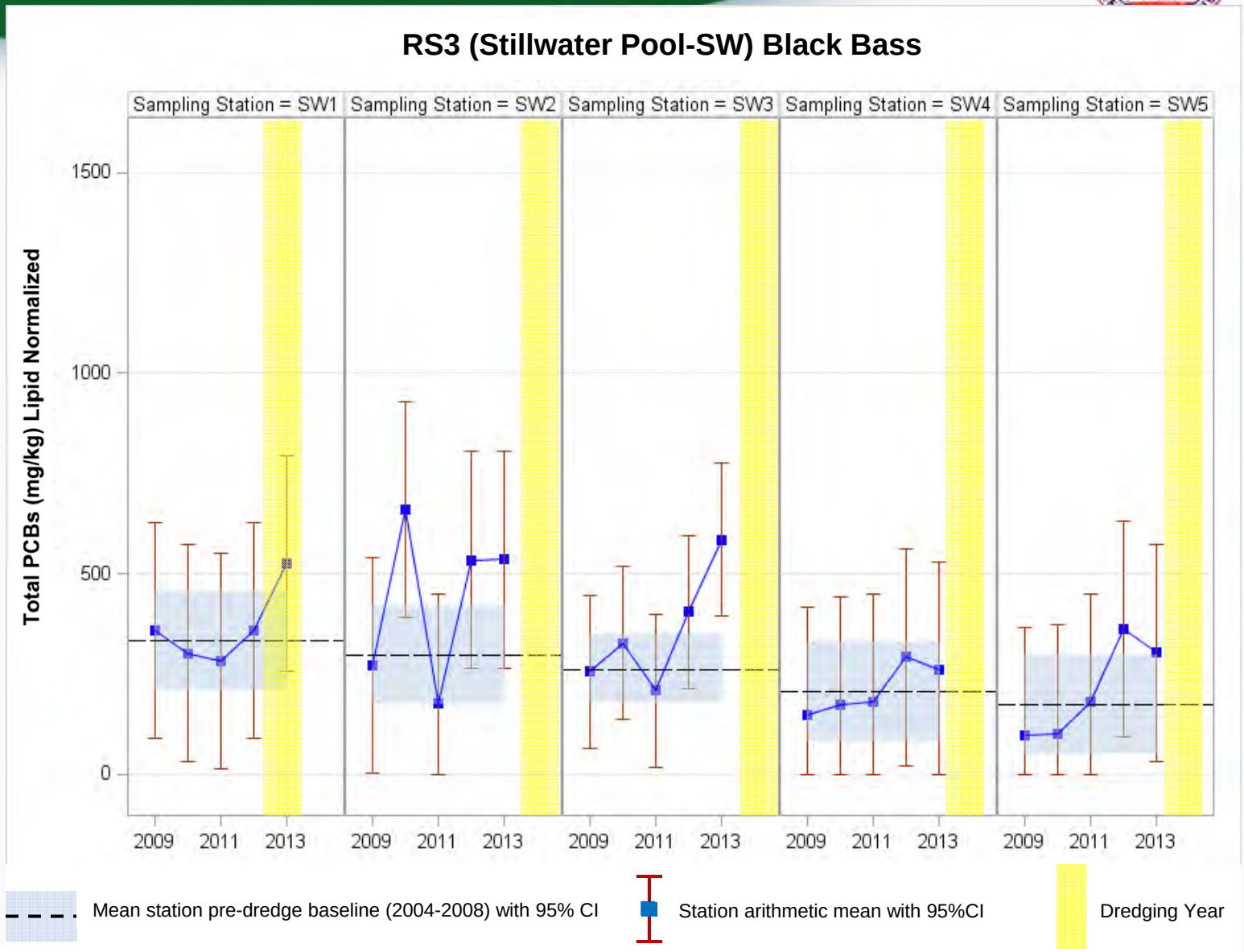


Hudson River PCBs SUPERFUND SITE

River Section 3 Fish Monitoring Stations and Dredging by Year

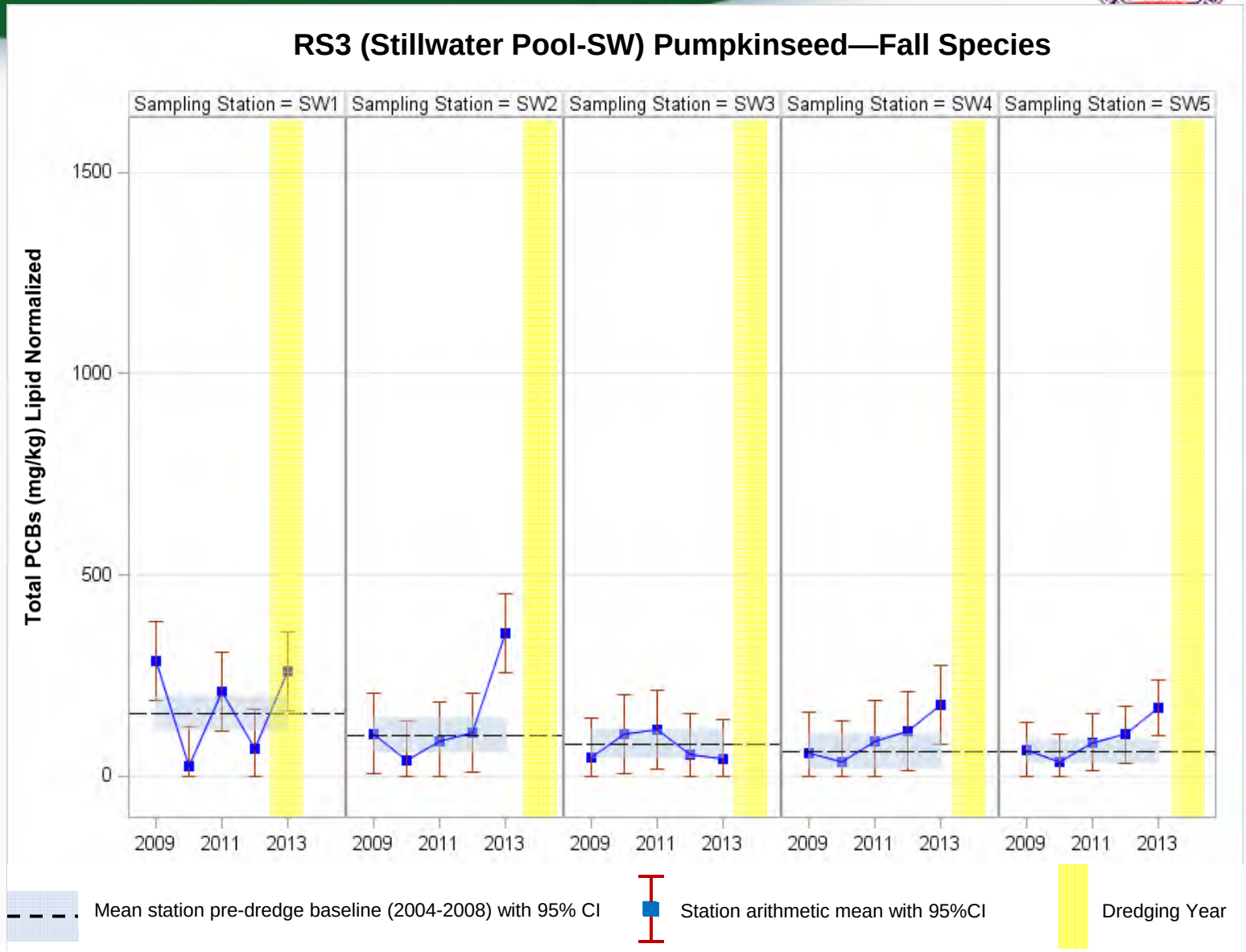


Comparison of Baseline to 2009-2013



Hudson River PCBs SUPERFUND SITE

Comparison of Baseline to 2009-2013



Hudson River PCBs SUPERFUND SITE

Total PCBs in Fish Tissues: Means Comparisons

Adjusted Geometric Mean TPCB in Fish Tissue Pre-Dredge (2004-2008 baseline) vs Post-Dredge (2009-2013)

River Section 1						
Species Group	2009: baseline	2010: 2009	2010: baseline	2011: baseline	2012: baseline	2013: baseline
Black Bass	-	+				
Bullhead					+	
Yellow Perch	-	+		+	+	+
Pumpkinseed	+	-	-	+	+	

Dredging Year

	Neutral p>0.10	+	Increase Post Dredging; p < 0.05
-	Decrease Post Dredging; p < 0.05	()	0.05< p < 0.10

Hudson River PCBs SUPERFUND SITE

Total PCBs in Fish Tissues: Means Comparisons

Adjusted Geometric Mean TPCB in Fish Tissue Pre-Dredge (2004-2008 baseline) vs Post-Dredge (2009-2013)

River Section 2						
Species Group	2009: baseline	2010: 2009	2010: baseline	2011: baseline	2012: baseline	2013: baseline
Black Bass	(-)	(+)				+
Bullhead			-		+	
Yellow Perch	-	(+)		+	+	+
Pumpkinseed	+	-	-	+	(+)	+

Dredging Year

	Neutral p>0.10	+	Increase Post Dredging; p < 0.05
-	Decrease Post Dredging; p < 0.05	()	0.05< p < 0.10

Hudson River PCBs SUPERFUND SITE

Total PCBs in Fish Tissues: Means Comparisons

Adjusted Geometric Mean TPCB in Fish Tissue Pre-Dredge (2004-2008 baseline) vs Post-Dredge (2009-2013)

River Section 3						
Species Group	2009: baseline	2010: 2009	2010: baseline	2011: baseline	2012: baseline	2013: baseline
Black Bass		(+)			+	+
Bullhead	-	(-)	-			
Yellow Perch	-					+
Pumpkinseed		-	-	+		+

Dredging Year

	Neutral p>0.10	+	Increase Post Dredging; p < 0.05
-	Decrease Post Dredging; p < 0.05	()	0.05< p < 0.10

Hudson River PCBs SUPERFUND SITE

Perspectives on Black Bass and Perch

- We have expected that short-term increases in fish PCB levels would occur during dredging
  - Since 2009 we have observed apparent dredging impacts within or immediately below dredge areas.
  - Black bass and perch have shown decreases in PCB levels at stations dredged before or during 2012.
  - We have also observed some increased PCB tissue levels in advance of Phase 2 dredging at some downstream stations.

Perspectives on Pumpkinseed

- We have expected that short-term increases in fish PCB levels would occur during dredging
  - For pumpkinseed (rapid integrators) PCB levels increased in the year of dredging at all stations in Phase 1 (2009), and Phase 2 (2011, 2012, and 2013)
  - Pumpkinseed also indicate some decreases of PCB levels in tissues after dredging:
    - PCB tissue decreases were observed in 2010 (no dredging) and again in 2011 and 2012 (after dredging at or near the station)

Perspective

- We anticipated that short-term, dredging related increases of PCBs in fish would rapidly return to baseline levels, and continue to decline thereafter following remediation
  - Exposures related to dredging were expected to be brief.
    - Dredging only occurs in a given area for single dredging season, or a portion thereof (weeks to months)
    - Tissue concentrations of PCBs in fish have been shown to decrease rapidly following spikes related to exposure events and environmental dredging at other sites

Spikes in tissue concentrations linked to dredging events have been observed to recover

Cumberland Bay Site, Plattsburgh, NY – Yellow Perch, Wilcox Dock

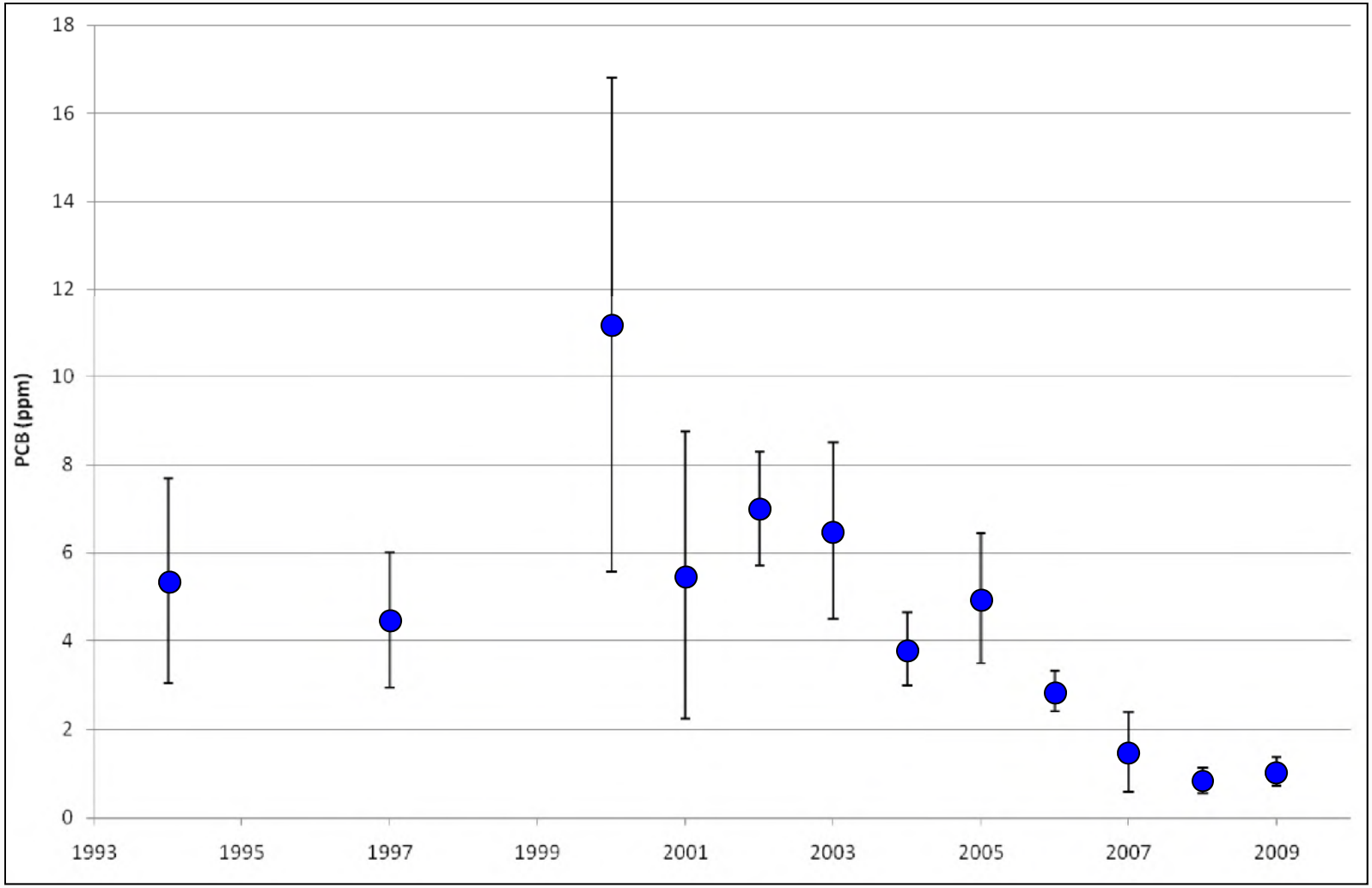


Figure courtesy of NYSDEC (2009)

Parting Thoughts

- Dredging program is not the only factor in this system influencing PCB concentrations in fish
  - Natural variability
  - Flooding, storms, flow conditions
- We have not observed changes in fish tissue concentrations that are outside of expectations.
- Special Study underway in 2014 regarding processing and filleting approaches comparison.
- Annual Monitoring will continue.

Attachment S

Hudson River PCBs Site
Peer Review of Phase 1 Dredging

HUDSON RIVER PCBs SITE
Peer Review of Phase 1 Dredging
Final Report

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With Contributions from:

SRA International, Inc.

September 10, 2010

Note

This report was prepared under contract to the U.S. Environmental Protection Agency (Contract No. EP-W-09-011, Task Order No. 031). The peer review process was conducted in accordance with Paragraph 14 of the Consent Decree (Civil Action no. 1:05 CV-01270, U.S. District Court for the Northern District of New York) and EPA's Peer Review Handbook (EPA Science Policy Council Handbook: Peer Review, December 2000). The peer review was conducted as a "contractor-run peer review" as defined in the Peer Review Handbook.

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EXECUTIVE SUMMARY

The Peer Review Panel (Panel) reviewed the considerable volume of data and reporting from the Phase 1 sediment remediation at the Hudson River PCBs Site to address 4 charge questions about the project. It was clear to the Panel that both the U.S. Environmental Protection Agency (EPA) and General Electric Company (GE) are committed to the success of the project and expended considerable effort to comply with the 2004 Engineering Performance Standards (EPS) during Phase 1. The Panel commends both parties for their extensive efforts to evaluate and report on the information generated during Phase 1 and the effort they expended in responding to the Panel's many requests for additional information and analyses.

Phase 1 showed that the 2004 EPS for Resuspension, Residuals, and Productivity were not met individually or simultaneously during Phase 1 and cannot be met under Phase 2 without substantive changes. EPA and GE proposed changes to the EPS but the Panel finds that the new proposed standards from either party would not contribute to the successful execution of Phase 2. However, Phase 2 can remove the bulk of the polychlorinated biphenyl (PCB) inventory if coring data and the resulting depth of contamination (DoC) model results are improved and focus is placed on quick closure of certification units (CU). The Panel developed an approach along with modified EPS to maximize removal of the PCB inventory in a careful balance with resuspension and residuals goals, while achieving an acceptable level of productivity.

The Panel also recommends building upon the adaptive practices and approaches that have been employed to date by developing a more comprehensive and formalized adaptive management approach to all EPS that includes the annual reassessment of the EPS based on each prior year's data. The challenges encountered during Phase 1, and the adaptations employed by EPA and GE to address those challenges, demonstrate the need for flexibility during Phase 2. This was evidenced in the records of the management meetings to achieve CU closure during Phase 1, and especially by the commitment to this Peer Review process, seeking to refine and improve the EPS and in-field practices. During Year 1 of Phase 2, the Panel recommends collecting additional data to support the further refinement of relevant performance standards to be applied for the remainder of the project's duration. Additional review between Years 1 and 2 of Phase 2, and each subsequent year of the project, should allow for ongoing modification of the EPS to optimize remedial operations while limiting unintended consequences and adverse environmental impacts from these operations.

Phase 1 demonstrated that the Residuals EPS had a substantial impact on the operational success of the project as well as the tangible interaction that exists between Productivity, Resuspension, and Residuals processes and their respective EPS. A key obstacle to simultaneously achieving the performance standards involved incomplete, inaccurate, and imprecise DoC characterization combined with disagreement on how to interpret and attain target levels. This directly affected both the Resuspension and Productivity EPS. The repeated dredge passes and prolonged exposure of sediments in the CUs resulted in increased PCB resuspension and release. The unexpected increase in inventory due to incomplete DoC characterization had the greatest effect on the Productivity EPS in terms of numbers of CUs remediated. The Panel presents revised EPS that accelerate CU closure by establishing an elevation-focused dredge design paradigm, thereby effectively managing residuals, reducing resuspension, and

accelerating productivity without compromising the goals of the Record of Decision (ROD) with respect to overall recovery of the river.

The Panel proposes an elevation-focused dredge prism design that builds on accurate, high-precision characterization of the DoC elevation, a 4-inch overdredge based on vertical tolerance of the dredge and precision of the DoC that ensures rapid achievement of the target elevation (i.e., the elevation of the DoC not including the overdredge) across at least 95 percent of the CU area or subunit area, verification of the target elevation based on high-precision bathymetry, and rapid closure of CU or subunit areas following EPA validation of confirmed elevations.

This approach does not involve redredging to remove dredge-generated residuals or address redefined inventory based on post-dredge confirmation sampling. The CU would be closed based on the results of the residuals sampling results. The CU (or sub-CU) should be backfilled if the average residuals concentration is less than or equal to 3 mg/kg Tri+PCBs and capped if the average residuals concentration is greater than 3 mg/kg Tri+PCBs.

This revised removal and closure approach is the first step toward integrating the Residuals, Resuspension, and Productivity EPS. Through better characterization of the DoC and establishing an elevation-based dredging prism design, Resuspension and Productivity EPS also can be revised to be consistent with the updated dredge depths and volumes. For Year 1 of Phase 2, the Panel proposes Resuspension and Productivity EPS based on metrics consistent with Phase 1: for resuspension, target levels are 2 percent and 1 percent of the dredged PCB mass, measured at Thompson Island Pool (TIP) and Waterford, respectively; for productivity, target volumes are 350,000 cubic yard (CY) per year. Both of these targets (i.e., for resuspension and productivity) should help guide Best Management Practices (BMP), but should not lead to shutting down operations. In other words, the Panel does not recommend interrupting dredging activities if the targets are not achieved during Year 1 of Phase 2; the goal of the interim standards is to establish baseline targets during Year 1 of Phase 2 and to allow dredging to recommence in 2011, while near-field and far-field data are collected.

Based on the results of Year 1 of Phase 2, combined with the Phase 1 results, EPA and GE should refine the performance criteria to establish practicable targets that can be achieved for all 3 EPS. In addition to evaluating the performance of the modified Residuals EPS, the focus between Years 1 and 2 of Phase 2 should be the Resuspension EPS to manage near-field and far-field resuspension, release, and deposition processes, based on an understanding of whether there are increased risks associated with surface sediment deposits containing PCBs released during dredging. The Productivity EPS should also be updated based on a revised volume estimate derived from the elevation-based dredging paradigm. In addition to an annual volume productivity standard, the Panel advances an additional EPS metric: annual areas to be remediated. Area remediated reflects a substantial measure of environmental benefit and could be expressed as a specified number of CUs to close each year. Tracking of total volume and mass of PCBs removed should continue, but the environmental benefit accrued should be based both on mass removal and area remediated. Eventually, an area-based standard could supplant the volume-based productivity standard, if appropriately tied to the elevation-based design.

The Panel found that the models used to develop the 2004 Resuspension EPS cannot be used to adapt revised standards for moving forward. The Panel believes that to do so requires a new model that must be developed collectively by EPA and GE. The GE model may be a useful foundation for this model, and

both model structure and parameters must be agreed upon by EPA and GE. The model must be peer reviewed by an expert panel once EPA and GE complete its development. Similar arrangements have been established at other Superfund Sites, including the Passaic River, the Lower Duwamish Waterway (WA), and the Lower Willamette River (OR). The fate, transport, and risk model must enable EPA and GE to understand the implications of operational changes on long-term recovery rates to support EPA and GE in making appropriate and meaningful risk management decisions about dredging productivity, BMPs, and the long-term fate and transport of PCB residuals and resuspension and release.

The Panel evaluated the results from Phase 1 in order to assess a practicable annual production rate. The evaluation included a detailed review of peak monthly output for each component of the remedial action (i.e., dredging, processing, transportation), dredging and removal output (i.e., numbers and cycle times for dredges and barges), and shipping output to the landfill. The Panel did not discover any single factor that could be adjusted to significantly increase overall productivity. For example, neither increasing the number of barges in service nor increasing the offload rate at the processing facility provided a substantive increase in productivity. Rather, the Panel found multiple lines of evidence supporting 350,000 cy/yr as a reasonable annual productivity estimate for the start of Phase 2. The Panel also found that the productivity schedule should be subordinated to the Resuspension EPS and Residuals EPS. Consequently the 5-year productivity criterion should be dropped to provide more flexibility to complete the work in a manner that protects the integrity of the project and its risk reduction objectives.

Charge Question 1

The experience in Phase 1 does not show that each of the Phase 1 EPS can be consistently met individually and simultaneously. None of the Phase 1 EPS were consistently met during Phase 1. EPA and GE evaluations of the Phase 1 experience do not provide evidence that the EPS could be met consistently and simultaneously if applied without modification during Phase 2.

The Resuspension EPS was not achieved in Phase 1. Resuspension criteria were exceeded, including total PCB concentrations and total and Tri+PCB loads; suspended solids concentration requirements were not exceeded, but alone provide an insufficient basis for understanding PCB resuspension and release. PCB release is the result of a complex set of processes, and, based on Phase 1 results, Total Suspended Solids (TSS) could not be used to predict PCB resuspension and release at this site. Resuspension was due in part to the dredging activities themselves, but was magnified by CUs being left open for extended periods.

The Residuals EPS was not achieved in Phase 1. Residuals management required multiple production passes (not anticipated in the EPS) and the CUs were open longer than intended. The Residuals EPS was not truly tested as envisioned in Phase 1, mainly because inventory was improperly characterized and the EPS assumed that all inventory would be removed with a maximum of 2 passes, followed by additional passes to remove dredge-generated residuals. The incomplete characterization of inventory was attributed primarily to problems with the delineation of the DoC in much of the river, which was rooted in problems with sediment core data, including lack of absolute vertical control on the DoC, poor core recoveries, and inability to characterize the entire soft sediment column by coring to till. Consequently, core sample results fed into the Terrain Model provided inadequate representation of the DoC, and dredging to the Terrain Model DoC fell short in all CUs.

The Productivity EPS was not achieved in Phase 1. None of the 4 numerical productivity criteria (i.e., minimum removal, target removal, maximum monthly rate, and transportation of all material off site by the end of the year) was achieved. The goal of transportation and disposal of all Phase 1 dewatered sediment by the end of 2009 was not accomplished. Ramping up unit processes is possible, but the project cannot be scaled up to meet the anticipated inventory using the current design data.

Charge Question 2

Both EPA and GE proposed changes to the EPS. The Panel finds neither proposal to be adequate, because neither adequately integrates the EPS so that all three EPS can be met individually and simultaneously.

EPA's proposal attempts to simplify the process, but it still relies too heavily on redredging and a complex decision process for closing CUs. Furthermore, EPA's recommended modifications to the Resuspension EPS do not support determination of whether released PCBs increase downstream risk to fish by creating unacceptable levels of surface sediment contamination outside of the remedial footprint. EPA's recommended annual productivity rates are much higher than can practicably be achieved.

GE's recommendations are tied to limiting downstream loading. Their assertion is that loading is tied directly to removal. The Panel finds that delayed closure of CUs is a major contributor to downstream loading. GE strongly recommends closing CUs with single-pass dredging in high-confidence areas and 2-pass dredging in low-confidence areas, while limiting the mass of PCBs removed. The Panel supports an approach that minimizes dredge passes and provides for quick CU closure. However, the Panel does not support placing an absolute limit on the mass of PCBs to be removed, because the mass of PCBs to be removed is unknown and such a limit appears contrary to the ROD.

Charge Question 3

The EPS can be modified for successful completion of the project. However, in addition to revising the performance criteria, changes are needed in the overall management of the project and its objectives. Namely, focus needs to be placed on achieving rapid CU closure to limit resuspension and release, while productivity needs to be measured with regard to the remediated footprint (i.e., equal focus on the area remediated as well as inventory removed), and there should be a more immediate application of backfill or cap based on the residual concentration of PCBs. This can be achieved by proactively determining the DoC, using updated DoC information to establish Design Dredge Elevations that more accurately capture the target inventory, and dredging the inventory based on updated Design Dredge Elevations for each CU and not based on residuals chemistry.

The following steps should be taken to establish an accurate and useful picture of DoC that can drive dredging plans and residuals management:

- Coring Program. Perform recoring of all low-confidence samples. Samples now designated as high-confidence should be verified as high-confidence with respect to the DoC elevation or re-sampled. All sampling must be performed to attain at least 80 percent recoveries of all soft sediments either to bedrock or Glacial Lake Albany Clay (GLAC). Further sediment layers must be reported as actual elevations rather than depth below the mudline, including the existing and future high-confidence core areas. All cores should be analyzed until 2 6-inch layers have Total PCBs below 1 ppm.

- DoC Elevation. Remodel the DoC based on the 1 ppm Total PCBs cleanup level using all high-confidence elevation-based cores to establish the topography of the DoC throughout each CU, referred to as the DoC Elevation. Thus, the DoC Elevation is a modeled elevation based on the sediment core DoC values to ensure that the inventory is captured by the Design Dredge Elevation with an acceptable level of certainty.
- Design Dredge Elevation. Establish the Design Dredge Elevation based on the remodeled DoC Elevation. Set the Design Dredge Elevation initially to 4 inches below the modeled DoC Elevation to account for the vertical accuracy of the dredge, referred to as dredge tolerance. The goal is for dredging to achieve the DoC Elevation in 95 percent or more of the dredged area after a single pass (i.e., at least 95 percent of the dredged area should be at or below the DoC Elevation). Incorporating a factor for dredge tolerance in the Design Dredge Elevation ensures that the dredger attains the DoC Elevations as quickly as practicable (i.e., in a single pass). If the dredger can easily achieve the DoC Elevation quickly and efficiently, the dredge tolerance can be relaxed. If the dredger has trouble achieving this in a single pass, the dredge tolerance should be increased.
- Confirmation Sampling. Perform confirmation composite sampling of surface sediments in each 1-acre CU subunit as soon as possible after attainment of the DoC Elevation in 95 percent or more of the area is confirmed by EPA.
- Sand Cover. Place a 3 to 6-inch sand cover over the CU subunit as soon as possible after confirmation samples are collected (before PCB analytical results are obtained). No verification of placement thickness is required at this time.
- Backfill or Cap. Use PCB analytical results from the composite samples to determine whether area will be backfilled or capped. Then install appropriate final layers. Do not redredge to capture residuals.

Charge Question 4

Both EPA and GE proposed changes to the EPS with concurrent changes to the monitoring and sampling program for Phase 2. However, the Panel finds that it will not be practicable to consistently and simultaneously meet the EPS being proposed by either party and, thus, cannot make a cogent finding regarding the monitoring and sampling programs relative to these proposed standards. Rather, the Panel has addressed this question relative to the modified EPS and processes recommended by the Panel in response to Charge Question 3.

Achieving all 3 EPS in Phase 2 requires an accurate determination of the DoC for all CUs, single-pass dredging to the DoC with a dredge tolerance, post-removal composite sampling to determine whether the CU requires backfilling or a cap, immediate placement of 3 to 6 inches of cover material, and placement of backfilling or cap after the composite sediment sample analysis.

The potential for recontamination of off site areas is not sufficiently addressed in the current monitoring program. While to date there is insufficient information to demonstrate that transported PCB load outside the currently planned CUs in the Upper and Lower Hudson is causing increased PCB concentrations in bedded-sediment concentrations, the Panel believes that expected benefits of the removal action must be demonstrated in the off site areas. If significant increases are occurring that compromise the expected risk reductions, further changes to the removal program would be warranted.

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1 INTRODUCTION

This report summarizes the independent peer review of the U.S. Environmental Protection Agency (EPA) Phase 1 Evaluation Report, the General Electric Company (GE) Phase 1 Evaluation Report, and supporting information. The Phase 1 Evaluation Reports presented EPA and GE's evaluation of the experience of the Phase 1 removal actions with respect to the Phase 1 Engineering Performance Standards (EPS) and set forth EPA and GE's proposed changes to the Phase 1 EPS, respectively. The reports and supporting information were reviewed by a panel of seven independent experts (the Peer Review Panel) in accordance with the terms of the Consent Decree under which Phase 1 of the cleanup was performed.

The Engineering Performance Standards address resuspension, polychlorinated biphenyls (PCB) residuals, and productivity associated with the removal of sediments contaminated with PCBs in the Upper Hudson River, New York. The purpose of the peer review was to consider the implications of the experience gained during the Phase 1 removal actions, as described in the EPA and GE Evaluation Reports and other evidence before the Panel, regarding EPS for subsequent planned removal of PCBs in the Upper Hudson River.

The peer review process included independent review by the individual Panel members, discussions and deliberations among the Panel members, public Peer Review Meetings that took place from May 4, 2010 to May 6, 2010, in Glens Falls, New York, and preparation of this Peer Review Report. SRA International, Inc. (SRA), under contract to EPA, organized and implemented the peer review according to procedures for a "contractor-run peer review," as outlined in EPA's "Peer Review Handbook" (EPA 2000).

This report summarizes the findings of the Peer Review Panel. The findings and discussions presented in Sections 2 through 7 of this report were written by the members of the Peer Review Panel and have been edited only for readability. The remainder of this introductory section provides background information regarding the Hudson River PCBs site (Section 1.1), reference to the EPA and GE Phase 1 evaluation reports (Section 1.2), a description of the peer review process (Section 1.3), and a roadmap to the remainder of the report (Section 1.4).

1.1 Background

1.1.1 Site and Regulatory/Enforcement History

In 1984, EPA classified approximately 200 miles of the Hudson River in the state of New York—from Hudson Falls to New York City—as a Superfund Site, based on PCB contamination of river sediments. This site traditionally has been divided into the "Upper Hudson River," which flows from Hudson Falls downstream to the Federal Dam at Troy, and the "Lower Hudson River," which flows from the Federal Dam downstream to New York City. The sediments were contaminated with PCBs predominantly by discharges from 2 capacitor manufacturing facilities owned by GE. In 1984, EPA issued a Record of Decision (ROD) for the Hudson River PCBs Site, which included, among other things, an interim No Action decision regarding the contaminated sediments.

Between 1990 and 2000, EPA reassessed its earlier decision with respect to the contaminated sediments of the Upper Hudson River to determine whether a different course of action was needed. The reassessment involved compiling and analyzing existing data, collecting additional data, using models to

evaluate human health and ecological risk, and studying the feasibility of various remedial alternatives. In 2002, after completing the reassessment, EPA issued a ROD that calls for, among other actions, targeted removal of approximately 2.65 million cubic yards of contaminated sediments from the Hudson River PCBs site (EPA 2002). Readers should refer to the ROD for further details on the site history, the remedial action objectives, and other aspects of the selected remedy.

EPA and GE entered into Administrative Orders on Consent (AOC) for sampling, analysis, and geophysical characterization of sediments (July 2002) and for remedy design (August 2003). In October 2005, the Justice Department and EPA reached an agreement with GE for GE to construct sediment transfer/ processing facilities and conduct dredging according to the ROD and design plans developed under the 2003 AOC. The U.S. District Court approved the Consent Decree documenting this agreement in November 2006.

1.1.2 Phase 1 Engineering Performance Standards

In addition to specifying the selected remedy, the ROD requires EPA to develop engineering performance standards that "promote accountability and ensure that the cleanup meets the human health and environmental protection objectives of the ROD" (EPA 2002). The ROD specifies the requirement for independent external peer review of reports prepared at the end of the first phase of the remediation to evaluate the removal action with respect to the engineering performance standards. The Consent Decree approved in November 2006 specifies process requirements for the peer review of the engineering performance standards.

EPA published an initial draft of the engineering performance standards in 2003, addressing resuspension, residuals, productivity, and quality of life standards associated with the planned removal of PCBs from the Upper Hudson River. A panel of 9 independent experts reviewed the EPS in accordance with the requirements of the Consent Decree. A public peer review meeting was held on January 27–29, 2004, in Saratoga Springs, New York. Based on input from the Peer Review Panel, EPA modified the EPS and published final EPS in the 5-volume document, "Engineering Performance Standards, Hudson River PCBs Superfund Site" (Malcolm Pirnie and Earth Tech, 2004).

1.2 EPA and GE Findings from Phase 1

Both EPA and GE prepared Hudson River Dredging Phase 1 Evaluation Reports that were completed and submitted to the Peer Review Panel on March 8, 2010. Both EPA and GE evaluated information gathered from Phase 1 and the outcomes of the removal work, and both EPA and GE proposed modifications to the EPS. Findings and proposed modifications to the EPS are documented in the EPA and GE Phase 1 evaluation reports and associated addenda (EPA 2010a, EPA 2010b, GE 2010).

1.3 Peer Review process

1.3.1 Peer Review Charge

The November 2006 Consent Decree specified the process for the peer review of the EPS. The Consent Decree presented 4 charge questions as well as general direction for conduct of the peer review process. The language from the Consent Decree, including the 4 charge questions, is presented in Figure 1.

"14. Peer Review

a. The Peer Review will evaluate the Phase 1 Evaluation Reports. The Peer Review will be conducted in accordance with EPA's Science Policy Council Handbook: Peer Review (December 2000), or any applicable updates thereto; the Office of Management and Budget's *Final Information Quality Bulletin for Peer Review* (December 16, 2004), or any applicable updates thereto; and the provisions of this Paragraph.

b. The Peer Review panel shall, at a minimum, address the issues raised by the following questions:

(1) Does the experience in Phase 1 show that each of the Phase 1 Engineering Performance Standards can consistently be met individually and simultaneously?

(2) If not, and if EPA and/or Settling Defendant has proposed modified Engineering Performance Standards, does the experience in Phase 1 and any other evidence before the panel show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2?

(3) If the experience in Phase 1 and other evidence before the panel does not show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2, can the Phase 1 Engineering Performance Standards be modified so that they could consistently be met in Phase 2, and, if so, how?

(4) If EPA and/or Settling Defendant has proposed modifications to the monitoring and sampling program for Phase 2, are the proposed modifications adequate and practicable for determining whether the Phase 2 Engineering Performance Standards will be met?

d. The Peer Review panel will not evaluate whether the Remedial Action will, or may, achieve the human health and/or environmental objectives of the ROD, nor will the Peer Review panel evaluate whether Phase 2 should be implemented."

Figure 1: Excerpts from the 2006 Consent Decree Specifying the Peer Review Charge

1.3.2 Peer Review Panel Selection Process

Paragraph 14c of the Consent Decree specified the process for selecting a Peer Review Panel to evaluate the Phase 1 evaluation reports and address the charge. Within this framework, EPA and GE established an agreed-upon process for selecting the Peer Review Panel. The process called for SRA to select a neutral Peer Review Panel selector, jointly approved by EPA and GE, who would have the authority to identify and select Panel members, provided that the candidates recommended by the Peer Review Panel selector had no personal or organizational conflicts of interest with respect to the Panel's charge. SRA identified Gregory Hartman of Dalton, Olmsted & Fuglevand as a candidate for Peer Review Selector, and in June 2009, EPA and GE agreed to name Mr. Hartman Peer Review Selector.

Per the Consent Decree, both EPA and GE identified collaboratively the appropriate areas of expertise to be included on the Peer Review Panel as follows:

- Monitoring: Panel members who are selected as monitoring experts will be knowledgeable in PCBs in aquatic media (water and sediments)
- Dredging production, operations, and equipment (including accuracy in dredge cuts and bathymetry)
- Residuals
- Sediment resuspension including knowledge of fate and transport
- Capping including accurate placement of backfill

EPA and GE were afforded the opportunity to recommend to the Peer Review Selector potential members for the Panel. EPA sent SRA a candidate list jointly developed by EPA and GE in August 2009.

SRA developed a conflict of interest (COI) analysis of all candidates and sent it to the EPA project team who then shared the list with GE for review. No COI concerns were raised and the final composition of the Peer Review Panel was determined in September 2009. The following experts were selected as the Peer Review Panel:

- Todd Bridges, U.S. Army Corps of Engineers, Engineer Research and Development Center
- Richard Fox, Natural Resource Technology, Inc.
- Paul Fuglevand, Dalton, Olmsted & Fuglevand, Inc.
- Gregory Hartman, Dalton, Olmsted & Fuglevand, Inc.
- Victor Magar, ENVIRON International Corporation
- Paul Schroeder, U.S. Army Corps of Engineers, Engineer Research and Development Center
- Timothy Thompson, Science and Engineering for the Environment, LLC.

1.3.3 Information Provided to the Panel

Both EPA and GE sent all necessary peer review documentation to SRA who distributed the information to the Peer Review Panel members. The Peer Review Panel was provided documentation to be reviewed both electronically on CD-ROM and through a secure online SharePoint site. Information included the EPA and GE Phase 1 Evaluation Reports and all supplemental information, the EPA Phase 1 Evaluation Report Addendum, and all public comments. SRA forwarded hard copies as appropriate and when requested by Panel members. Documents provided included:

- EPA and GE Background documents (January 2010)
- EPA and GE Items Provided Independent of Panel Requests from the February 17-18 Introductory Session (includes Addendum to the Phase 1 Evaluation Report)
- EPA and GE Items Provided in Response to Panel Supplemental Information Requests following February 17-18 Introductory Session (submitted to EPA March 2, 2010 and forwarded by EPA to GE March 10, 2010)
- EPA and GE Information Provide to the Panel in Response to Information Requests following the May 4-6 Peer Review Panel Meeting

The Peer Review Panel did not review subsequent modeling runs completed by GE that occurred after the May 4-6, 2010 public meeting. A comprehensive list of documents provided to the Peer Review Panel is attached to this report as an Appendix.

1.3.4 Peer Review process

On October 1, 2009, the Panel was requested to visit the Hudson River PCBs Superfund Site to observe high-volume Phase 1 removal actions in progress. Six of the seven members of the Panel traveled to the site to participate in a boat tour, the purpose of which was to provide the Peer Review Panel members with factual information pertaining to the site. Following the tour, the Panel and SRA gathered in a GE

conference room with members of the EPA and GE site teams for an informal question and answer session regarding the general charge of the Panel, the schedule for the peer review, and some of the technical challenges encountered during the Phase 1 activities. The meeting did not include any discussions pertaining to the GE Phase 1 Data Compilation or the EPA or GE Phase 1 Evaluation Reports, nor did it involve any interpretation of data that were collected in connection with the Phase 1 removal actions.

A collaborative approach was implemented throughout the peer review process. SRA organized frequent internal conference calls with the Peer Review Panel members to discuss status of the review and administrative and logistical issues. SRA served as a liaison between the Panel and EPA and GE helping to address Panel member concerns, additional information requests, and technical documentation needs. Any information requests from the Panel were presented to SRA then forwarded to EPA; EPA and GE worked together to provide the appropriate information to the Panel.

The Peer Review Panel members attended 2 meetings held in New York. The first was the Introductory Session held February 17-18, 2010 in Saratoga Springs, New York where EPA and GE presented the data and issues presented in their respective Phase 1 Evaluation Reports. The second was the Peer Review Public Meeting held May 4-6, 2010 in Glens Falls, New York where EPA and GE presented findings and the Peer Review Panel deliberated on issues raised according to the charge questions provided in Section 1.3.1 of this report. There was also a public comment period during each of these meetings providing the public an opportunity to present comments to the Peer Review Panel members. Public comments were provided to the Peer Review Panel electronically and in hard copy as requested.

Subsequent to all public peer review meetings, the Peer Review Panel worked collaboratively to develop this Peer Review Report.

1.4 Organization of Report

The Peer Review Panel findings are presented in the remainder of this report. Section 2 of the report presents an overview of the Panel's findings. Sections 3, 4, and 5 present the Panel's findings for each charge question for each the 3 Engineering Performance Standards, respectively: Resuspension, Residuals, and Productivity. Section 6 provides a summary of these findings organized by charge question, and Section 7 presents concluding remarks of the Panel.

2 OVERVIEW OF THE PANEL FINDINGS

2.1 Overview

The Panel reviewed the considerable volume of data and reporting from the Phase 1 sediment remediation at the Hudson River PCBs Site to address 4 charge questions about the project. It was clear to the Panel that both EPA and GE are committed to the success of the project and expended considerable effort to comply with the 2004 EPS during Phase 1. The Panel recognizes their extensive efforts to evaluate and report on the information generated during Phase 1 and the effort expended in responding to the Panel's requests for additional information and analyses.

The Panel also recognizes that during Phase 1, the project encountered challenges in the implementation of the remedy and the use of the EPS to guide these efforts. In this way, Phase 1 did achieve a critical outcome, in that it elucidated the strengths and weaknesses of the EPS and provided important lessons regarding the design and implementation of the EPS going forward. If these lessons are heeded and incorporated into a modified set of EPS, it is expected that the project will more effectively achieve the desired outcomes.

Phase 1 showed that the 2004 EPS for Resuspension, Residuals, and Productivity were not met individually or simultaneously during Phase 1 and cannot be met under Phase 2 without substantive changes. The Panel recognizes the considerable efforts expended by EPA and GE in developing proposed changes to the EPS based on the lessons learned from Phase 1. However, the Panel finds that neither the EPA proposed modified EPS nor the GE proposed modified EPS would support the successful execution of Phase 2. Consequently, in response to Charge Questions 3 and 4, the Panel has developed and is recommending the implementation of modified EPS and Best Management Practices (BMP).

Phase 1 demonstrated that the Residuals EPS had a substantial impact on project success and on the interaction with the Resuspension EPS and the Productivity EPS. A key obstacle to simultaneously achieving the performance standards involved incomplete depth of contamination (DoC) characterization combined with adherence to the 2004 EPS residual target levels. This directly affected both the Resuspension and Productivity EPS. The repeated dredge passes and prolonged exposure of sediments in the certification units (CU) resulted in increased PCB resuspension and release. The unexpected increase in inventory due to incomplete DoC characterization had the greatest effect on the Productivity EPS in terms of numbers of CUs remediated.

The Panel's proposed modifications are predicated on the Panel's belief—based on our evaluation of the Phase 1 information and our collective experience—that if the DoC is better characterized and a focus is placed on quick closure of CUs, the bulk of PCB inventory can be removed during Phase 2. The Panel proposes revising the Residuals EPS to accelerate CU closure by establishing an elevation-focused dredge design paradigm, thereby reducing resuspension, effectively managing residuals, and improving productivity without sacrificing goals of the ROD with respect to overall recovery of the river.

More importantly, the revised EPS must be designed with the recognition that the tensions created by trying to achieve all 3 standards simultaneously can lead to unanticipated and unacceptable environmental consequences, such as increased resuspension and residuals due to prolonged CU dredging, or reduced productivity due to resuspension and residuals management to meet the EPS.

These tensions should be recognized before entering Phase 2, while seeking to resolve them through adaptive management that involves routine reassessment of dredging operations, BMPs, and dredging performance with regard to the EPS.

Toward this end, the Panel has developed an approach along with proposed modified EPS to maximize removal of PCB inventory in a careful balance with resuspension and residuals goals, while achieving an acceptable level of productivity. Further, the proposed approach and EPS incorporate adaptive management principles and build upon the commitment to these principles demonstrated by EPA and GE during Phase 1.

The Phase 1 Hudson River EPS Peer Review represents the intensely collaborative product of a group of 7 senior sediment remediation experts with diverse and complementary expertise across all of issues involved in remediation of the Hudson River. The Panel's findings reflect an integrated understanding of the contemporary challenges, limitations, and opportunities associated with environmental dredging and sediment remediation and provide a solid foundation to improve the outcome of Phase 2. The Panel has concluded that its findings will not be effective if taken piecemeal, but require an integrated application to provide benefit to Phase 2.

2.2 Structure of Response to Charge Questions

Sections 3 through 5 of this document present the Panel's detailed review of the charge questions. Each section is devoted to a different EPS – Section 3 addresses the Resuspension standard, Section 4 addresses the Residuals standard, and Section 5 addresses the Productivity standard. Each section addresses the four charge questions as they relate to their respective EPS. Section 6 reorganizes the presentation by charge question, presenting a synopsis of the detailed findings presented in Sections 3 through 5 for each of the charge questions.

The charge questions follow a logical line of inquiry. Question 1 lays the foundation for the review, addressing the question of whether the 2004 EPS were met in Phase 1. The response to Question 2 is predicated on the response to Question 1, and the responses to Questions 3 and 4 are predicated on the response to Question 2. During deliberations, the Panel decided that the clearest approach for communicating findings would be to address this logical series of questions for each EPS, rather than proceed question-by-question.

However, the Panel recognizes that the EPS should work together and cannot be addressed independently. Where these inter-connections are particularly relevant to a finding, the detailed responses presented in Sections 3 through 5 address them. This interconnectivity is further addressed in Section 6.

3 RESUSPENSION

CHARGE QUESTION 1. Does the experience in Phase 1 show that each of the Phase 1 Engineering Performance Standards can consistently be met individually and simultaneously?

Finding Rsp.1: The Phase 1 Resuspension Engineering Performance Standard (EPS) could not be consistently met individually during Phase 1, nor could the Resuspension EPS be met simultaneously with the other EPS, and the Resuspension EPS must be revised for Phase 2.

Phase 1 experience clearly indicates that the 2004 Resuspension EPS was not consistently met (Table 1). All criteria set for PCBs were exceeded in Phase 1. The resuspension criteria include total PCB concentration, total and Tri+PCB load, and suspended solids concentration thresholds. The Resuspension EPS requires that the criteria be met at all far-field stations; defined as at least 1 mile downstream of dredging operations.

Both EPA and GE reported that the PCB-related criteria within the Resuspension EPS were not met during Phase 1. On the other hand, the Total Suspended Solids (TSS) near-field and far-field criteria were not exceeded during Phase 1; however, the relation of these measurements to release of PCBs is not evident in the Phase 1 monitoring data.

The failure to meet the Resuspension EPS for PCBs during Phase 1 was caused by multiple factors, including:

- The conceptual model did not account for all potential release mechanisms associated with dredging-related activities (i.e., not just dredge-induced sediment resuspension), therefore data were insufficient to support analysis of activities not directly related to dredging.
- Lack of recognition that suspended solids alone provide an insufficient basis for predicting PCB release rates.
- Underestimates of the total volume and PCB mass dredged during Phase 1.
- Underestimate of the PCB release rate (i.e., the release rate as a percentage of PCB mass dredged).
- Underestimate the downstream cumulative PCB loading rate and its contribution to monitored natural recovery (MNR).
- The rate and magnitude of PCB deposition in the upper and lower river was unaccounted for and not monitored.

The 2004 Resuspension EPS could not have been met because it is based upon the unsubstantiated premise that PCB release and transport are closely and simply related to the rate of sediment particulate resuspension and that a reliable relationship existed between total PCBs and sediment particulates as measured by TSS and/or turbidity. As indicated by the 2004 EPS Peer Review Report, the accumulated body of evidence in dredging studies demonstrates that the resuspension and release of PCBs during dredging cannot be predicted simply by measuring suspended solids and without accounting for dissolved PCB release and transport (Bridges et al. 2008). There are a number of release mechanisms/pathways for PCBs in addition to the release of suspended solids during dredging, including dredging induced release of porewater, dredging induced release of PCB oils, flux from exposed sediment surfaces, resuspension of sediments from exposed surfaces, as well as partitioning from

resuspended particles. Phase 1 demonstrate that there is no reliable relationship between total PCBs and sediment particulates as measured by TSS and/or turbidity; that is, measured TSS and PCB transport from removal operations were not statistically correlated during Phase 1.

The EPS for resuspension was set too low to be met in Phase 1 or Phase 2 without flow and traffic control in the Hudson River. The EPS was based on the premise that resuspension of solids and release of PCBs during dredging could be held to less than 1 percent of the total dredged mass. The experience in the dredging literature shows that resuspension by the dredge can generally be limited to a 1 percent loss (Hayes and Wu 2001, Pennekamp et al. 1996, Palermo et al. 2009); however, this resuspension does not represent the total loss of solids and PCBs. Additional losses occur from debris removal, and erosion of generated/disturbed residuals by high flow events and prop wash. Generated/disturbed residuals typically represents 2 to 9 percent of the total dredge mass of the final pass (Patmont and Palermo 2007). In a riverine system with currents as high as present in the Hudson River during higher flow periods, much of the generated residuals will be lost if the residuals are not covered. Therefore, typical PCB losses in riverine systems such as the Fox River (Steuer 2000) and Grasse River (Connolly et al. 2006) are reported to be in 2 to 3 percent of the mass dredged. These typical results encompassing all sources of losses are consistent with the losses observed in Phase 1. Therefore, setting the EPS for resuspension to achieve losses less than 2 percent without flow and traffic control are unrealistic and not practicable.

Table 1: Comparison of resuspension results to the EPS

Parameter*		Evaluation Level		Control Level		Standard Level		Finding	Was the 2004 EPS met?
		Limit	Duration	Limit	Duration	Limit	Duration		
Far-Field PCB Concentration	Total PCBs	---		350 ng/L	7-day running average	500 ng/L	Confirmed Occurrence	Total PCB Standard of 500 ng/L at Thompson Island Station was exceeded on 3 to 10 occasions. The Control Level was exceeded 4 times (as a 7-day average) at the TIP, but not at Lock 5 (Schuylerville).	No
Far-Field Net PCB Load	Total PCBs	---		117 kg/yr	Dredging Season	---		Total PCBs transport was 437 kg (500 kg GE estimate) past the TIP, and 151 kg (200 kg GE estimated) to Waterford. Tri+ levels were 123 kg past Lock 5, and 61 kg past Waterford.	No
	Tri+PCBs			39 kg/yr					
	Total PCBs	541 g/day	7-day running average	1080 g/day	7-day running average			At Thompson Island, Lock 5, and Waterford, the 7-day running average net loadings for Total PCBs and Tri+PCBs were exceeded. The total Phase 1 PCB load control levels were also exceeded.	
	Tri+PCBs	180 g/day		361 g/day					
Far-Field Net Suspended Solids Concentration	TSS	12 mg/L	24 hrs.-average	24 mg/L	24 hrs.-average	---		Average TSS concentrations at near-field monitoring stations were well below the evaluation criteria of 700 mg/L at 100 m and 100 mg/L at 300 m downstream of the dredging operations.	Yes
Near-Field (300 m) Net Suspended Solids Concentration	TSS	100 mg/L	6-hr average net increase over ambient	100 mg/L	6-hr average net increase over ambient	---			Yes
Near-Field (100 m and Channel-Side) Net Suspended Solids Concentration	TSS	700 mg/L	Calculated from discrete turbidity measurements made in 2 sampling events per day	---	---	---			Yes

* Sources for this data include Table I-1-1 from EPA Phase 1 Evaluation Report 1, and Phase 1 Performance Standards Compliance Plan, May 2009, Table 2-1. See also Tables I-1-2 through I-1-6 and I-2-1 through I-2-3

Finding Rsp.1-1: The far-field PCB concentration limit was exceeded repeatedly.

The PCB concentration standard and control level was exceeded during Phase 1 at the Thompson Island Pool (TIP) and Lock 5 far-field monitoring stations. The Standard Level of 500 ng/L total PCBs, set to protect drinking water supplies at Waterford Station, was exceeded on 3 or 10 occasions (EPA reported 3, whereas GE reported 10 exceedances at Thompson Island; the difference depends on the interpretation of analytical results for certain co-eluting congeners). GE reported that the Control Level of the 7-day running average of 350 ng/L was exceeded during 4 periods at the Thompson Island monitoring station; July 18-22, 2009; July 31-August 10, 2009; September 15-16, 2009; and October 12-21, 2009. EPA acknowledges the Control Level was exceeded at the Thompson Island station; from EPA's Figure I-3-4b this appears to have occurred between July 28 and August 9. Notably, by EPA's measures, the 7-day average total PCB concentrations did not exceed the Control Level of 350 ng/L at the Lock 5 (Schuylerville) monitoring station, but there was 1 exceedance of the 500 ng/L level Resuspension Standard.

Finding Rsp.1-2: The net PCB load criterion was exceeded at far-field stations.

All PCB loading criteria were exceeded at the far-field stations in Phase 1 (Table 1). This includes both the evaluation and control levels as either 7-day running average or as the total mass for the Phase 1 dredging season. The Panel understands that while the loadings were adjusted in the Phase 1 Performance Standards Compliance Plan (GE 2009) based upon the estimated Phase 1 mass, even adjusting again for the actual mass removed, the far-field net PCB load criteria were exceeded (Table 2). Both the EPA and GE reports acknowledge this fact.

Table 2. Estimated and actual PCB transport losses from Phase 1

	Document					
	ROD [1]	EPS Phase 1 [2]	Phase 1 Design [3]	PSCP [4]	EPA Phase Report [5]	GE Phase 1 Report [6]
Total Mass TPCB (kg)	69,800	6,980	10,000	12,564	20,000	16,320
Estimated TPCB Loss (kg)	90.74	65	30 - 59	117	437 past TI 151 to Waterford	500 kg past TI 200 kg to Waterford
Estimated Tri+PCB Loss (kg)	---	---	---	39	123 past Lock 5 61 past Waterford	---
Percent Loss	0.13%	1%	0.3%	0.93%	2.2%	3.1%
	---	---	0.6%	---	0.8%	1.2%

Notes:

1. Record of Decision, Table 3-1 and Page 69 which stated a 0.13% loss due to resuspension
2. EPS (2004) Pages 16 and Pages 95 - 97.
3. Phase 1 Design Report, F.6, Page 6-1. Modeled loss of 0.35% (30 kg) and 0.65% (59 kg) of mass removed.
4. Phase 1 Performance Standard Compliance Plan, pages 13 and 21. Control level criteria adjusted to 117 kg/yr total PCBs; 39 kg Tri+PCB
5. EPA Phase 1 Report, Page I-3.
6. GE Phase 1 Report, page 77 and Table 4.2-3

--- Indicates no value was reported

For the annual load limit, EPA reports 437 kg transported past Thompson Island, while GE reports about 500 kg. The 2 parties report total PCBs of 151 kg and 200 kg, respectively, past Waterford. EPA reports Tri+ levels were 123 kg past Lock 5, and 61 kg past Waterford; GE does not report the Tri+ loads past Lock 5 or Waterford.

Using EPA's 437 kg value, the average daily release rate is 2,497 g/day for the 175-day production period from May 14 through November 4, 2009. Thus, PCB loading exceeded all of the criteria, including the 1,600 g/day criterion deemed unacceptable by EPA in the 2004 Resuspension EPS.

While the Total PCB (TPCB) and Tri+PCB 7-day running average net load at Thompson Island exceeded the Phase 1 Control Levels (1,080 g/day and 361 g/day, respectively) throughout most of the project, the TPCB loads at Lock 5 and Waterford were significantly less than those observed passing the Thompson Island Dam. At Waterford, the 7-day average load was less than the Evaluation Level about 50 percent of the time and only exceeded the Control Level 20 percent of the time.

EPA states in its Phase 1 Report that "EPA's goal of a maximum 1 percent loss rate to the Lower Hudson River was achieved." While this is true, the 2004 Resuspension EPS is very clear in stating that the standard is applicable to all far-field stations, which are defined in the 2004 EPS as stations that are 1 mile or more below the dredging area. A revised Far-Field Net PCB Load standard should be applied to all far-field stations in Phase 2 in order to ensure that the objectives motivating the use of the load standard are met for the upper and lower portions of the river.

That the net PCB loads exceeded the 2004 Resuspension EPS is singularly troubling. The Panel disagrees with EPA's contention that the "Data do not support the notion that settling of PCB-contaminated sediment was a significant contributor to resuspension and recontamination of non-dredged areas." In fact, limited data collected on the net load deposition downstream of the TIP dam make it impossible to substantiate this observation. The incompleteness of the conceptual model in regard to PCB release and deposition mechanisms, and the incompleteness of the monitoring data relative to those release mechanisms, prevents the Panel from reaching credible conclusions about the nature and fate of the PCB releases—the origin of the releases, the long-term consequences of those releases, and what management actions should be taken to reduce or control those releases.

Finding Rsp.1-3: The Total Suspended Solids (TSS) standard was met, but the basis for the standard is invalid.

As pointed out by the 2004 Peer Review, the scientific literature demonstrates the TSS cannot be used as a predictor of PCB release during dredging operations. The Phase 1 data also show that TSS is not a sufficient predictor of PCB release.

Finding Rsp.1-4: Modeling and data collection gaps limit the usefulness of MNR comparisons.

The 2004 Resuspension EPS used HUDTOX and FISHRAND models to simulate water column, sediment, and fish Tri+PCB concentrations as a result of dredging operations. Modeled export loads and potential impacts to the public water supply were written into specific criteria; however, this effort also examined the potential effects of changes to fish tissue concentrations.

A conclusion in the 2004 EPS is that resuspension of PCBs in compliance with the standard would have a negligible adverse effect on Tri+PCB concentrations in Hudson River fish, as compared to a scenario with non-dredging-related PCB releases. The EPS defined a negligible effect as a predicted Tri+PCB

concentration in Upper Hudson fish of 0.5 mg/kg or less, and in Lower Hudson River fish of 0.05 mg/kg or less, within 5 years after the completion of dredging in the Upper Hudson. These results could not be substantiated based on the Phase 1 data.

The 2004 EPS clearly, and repeatedly, references monitored natural attenuation (MNA) as a basis for establishing the upper bound of an acceptable level of sediment release (emphasis added).

> "The cumulative Tri+PCB load at Waterford as forecasted by HUDTOX was used to determine what would be considered a significant release (i.e., resuspension export rate) from the dredging operation…The lower bound will be the ideal conditions of dredging, where there are no sediments being spilled (no resuspension) and ***the upper bound will be the MNA scenario***."

While a comparison to MNA or MNR conditions is worthwhile, the usefulness of the modeling effort has been limited by the following:

- Fish tissue monitoring results have been insufficient to determine the net effect on short- and long-term PCB bioaccumulation downstream of the dredging footprint (see following discussion).
- HUDTOX/FISHRAND models are outdated and inadequate to accurately project MNR and post-dredge fish recovery rates.
- Neither EPA nor GE has sufficient data or a credible tool to project recovery.
- The MNR analysis is incomplete, insofar as it relies on a single line of evidence (namely, comparison of MNR vs dredge-related far-field PCB sediment loads) to evaluate and compare dredge-related releases to MNR releases.

The incomplete analysis done for the 2004 EPS does not consider near-field and far-field PCB deposition rates on the sediment bed surface; accelerated recovery potential in the areas targeted for dredging, primarily due to post-removal backfill and capping, natural sedimentation, and surface sediment mixing; and volatilization that can influence human exposures. An analysis based solely on cumulative loads to compare MNR and dredging is incomplete. A more relevant analysis would measure and predict changes in surface sediment chemical concentrations due to dredging and long-term changes in fish recovery rates to compare the time required for long-term recovery after dredging with the time required for long-term recovery under MNR.

Results of fish tissue monitoring offered limited projections on long-term fish concentrations relative to the no dredging-related PCB releases (i.e., MNR alternative). The fish concentration data collected during and after 2009 dredging operations are a measure of short-term, transient exposures due to water column PCB concentrations produced as a result of dredging. More substantial impact is likely to occur via long-term exposures due to increased surface sediment PCB concentrations resulting from the release, deposition and flux of PCBs into the sediment bed in the upper and lower river. Factors that must be considered in evaluating existing and future fish tissue data include:

- Black bass, bullhead, and yellow perch were sampled in June 2009, before most of the removal activity occurred. If these species were sampled again in June 2010, the results would be relevant to developing projections regarding long-term recovery.

- The forage fish sampling protocol results in limited statistical power due to limited replication.
- The most useful of the existing fish tissue monitoring data sets is the pumpkinseed data. However, pumpkinseed are not a “worst case” species with respect to bioaccumulation potential, as they are neither top predators, directly associated with bedded sediments, nor particularly high in lipid content.
- PCB concentrations in fish take time to equilibrate and may have continued to increase after water and sediment concentrations began to stabilize.

CHARGE QUESTION 2. If not, and if EPA and/or GE has proposed modified Engineering Performance Standards, does the experience in Phase 1 and any other evidence before the panel show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2?

Finding Rsp.2: It is not practicable to consistently and simultaneously meet the Resuspension EPS proposed by either GE or EPA for Phase 2.

Both EPA and GE provided tables of their proposed changes for Phase 2 to the Resuspension EPS at the May 2010 Peer Review meeting in Glens Falls, New York. Those are shown in Table 3 and Table 4, respectively, along with the Panel's response to each of those proposed revisions. In general, the Panel found that neither party proposed changes that can be supported at this time for Phase 2. The Panel specifically finds that (1) the far-field net PCB loads cannot be solely applicable to the Lower Hudson; (2) while load criteria are needed for Phase 2, neither the tools nor data necessary for setting them are available from Phase 1; (3) there is no need to revise the TSS standard, but the rationale for setting that standard should be examined as it is no longer valid; and (4) the far-field PCB concentration standard of 500 ng/L should be maintained.

EPA articulated a broad set of goals in the 2004 EPS to protect human health and the environment, and designed the resuspension standard to avoid disturbing near-field and far-field conditions relative to an MNR trajectory modeled for the ROD. It is both reasonable and important to comprehensively evaluate the long-term effects of planned remedial actions, and to adjust plans and operational practices so as to limit the unintended and undesirable environmental consequences associated with remedial activities. However, EPA's proposed revision to the resuspension standard does not adequately address how the removal action in the river could positively or negatively affect environmental conditions between the TIP and Waterford. As GE has indicated, unrestricted release of PCBs from the removal action could exacerbate risks to both near-field and far-field receptors. However, the Panel was not provided with sufficient evidence to evaluate the environmental consequences of increased mass transport observed in Phase 1, and predicted for Phase 2.

Neither EPA nor GE has proposed scientifically supportable standards. EPA's load analysis, presented in the 2004 EPS and Phase 1 reports, is based on HUDTOX/FISHRAND model projections, which are not reliable for evaluating and setting release loads. GE did not provide the Panel with sufficient detail and explanation supporting their proposed standard change based on new modeling results; therefore the Panel was not able to evaluate the calculations, assumptions, or conclusions of their effort. However, regardless of the modeling details, the Panel believes that the data collected during the 2009 dredging season are unlikely to provide a sufficient basis for a definitive modeling effort concerning PCB releases and their consequences. In this regard, defensible data on near-field resuspension release rates are needed. It is also not acceptable, nor is it consistent with the ROD or 2004 EPS, to constrain the consideration of long-term consequences of release to the Lower Hudson. Consistent with the 2004 EPS definition of far-field, an understanding of the loading to the Hudson River between the TIP Dam and Waterford is required.

HUDTOX is not a proper basis upon which to derive dredging criteria for Phase 1, and cannot be relied upon to derive criteria for Phase 2. In addition to HUDTOX not being built to model dissolved PCB losses,

the key assumption in that model is that losses are from dredging resuspension alone. The model was not constructed in a manner that considered other contributing factors such as debris removal, prop wash from scows and tugs associated with the dredging, prop wash from other vessels in the river, and hydrodynamic scour due to (a) the dredging configuration, and (b) the time newly exposed dredge surfaces were left open. Furthermore, HUDTOX is not capable of evaluating dredge-related or localized resuspension scenarios; it requires input of PCBs as a specific load rate (EPS Volume 1, Section 2.6).

Attachment F of the 2006 Phase 1 Final Design Report included dredge resuspension modeling. The dredge resuspension simulated is only that sediment resuspended in the water column from direct dredge operations, and does not include other dredge-related sources of resuspension such as debris removal, installation and removal of sheet piling, silt curtains, and barge movement. High-flow / event resuspension (erosion) was not considered because dredging activities were not expected to take place during such river conditions (ref. p. 1-4 of Attachment F of the 2006 Phase 1 Final Design Report).

While there is a very real need to set far-field PCB load criteria, neither the data nor the tool(s) presented to the Panel are adequate for setting a revised standard. Additional data will be needed on near-field PCB releases, continued near-field and far-field measures of PCBs (total and dissolved), formulation of a conceptual site model that encompasses all the mechanisms for PCB release, and the development of a new or updated model that can be used to project PCB fate and effects with a higher degree of confidence than is currently available.

Table 3. Summary of EPA's proposed modifications to the Resuspension EPS

EPA Proposed Modifications to Resuspension EPS				Panel Finding	
Standard	**Proposed Change**	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
Far-Field Net PCB Load	Adjust the far-field net PCB load standard; adjust the seasonal load and corresponding daily evaluation and control level loads upwards.	The Control Level for cumulative Tri+PCB load due to the project: 1% of the estimated Tri+PCB inventory. Based on the current best estimate of the PCB mass to be removed, 1% is 670 kg Tri+PCB; the corresponding daily load Control Level is 680 g/day based on a 7-day running average. The Evaluation Level will be 500 kg Tri+PCB. The daily load equivalents will be 490 g/day, based on a 7-day running average. The daily load for the Control and Evaluation Levels will also be prorated to reflect the annual Productivity EPS schedule and the estimated mass of PCBs to be removed in the given year.	Based on new model analysis, a total project net PCB load of 670 kg Tri+PCBs +/- 25% was shown to have only a negligible impact on the Lower Hudson. Evaluation of potential effects of various Tri+PCB loads on Lower Hudson River fish tissue concentrations indicates that a 670 kg project load will yield a similar rate of recovery to 2004 model simulations.	No	The Panel agrees in concept with the need to re-evaluate the numerical load criteria for both the Upper and Lower Hudson River. However, because EPA could not adequately define the environmental consequences of near-field and far-field resuspension and release loads, the Panel maintains that there is insufficient information available to establish revised numerical criteria for the Resuspension EPS. Insufficient Phase 1 data specific to near-field PCB releases exist to support the development of a revised Resuspension EPS.

EPA Proposed Modifications to Resuspension EPS				Panel Finding	
Standard	**Proposed Change**	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
	Revise the station of compliance for load to Waterford, exclusively.	N/A	Waterborne PCB concentrations decrease with distance from dredging. The focus of the load analysis in the 2004 Resuspension EPS documents was loads that would be released to the Lower Hudson; such loads are best measured at Waterford. Thus, this change is consistent with the intent of the performance standard. Based on new model analysis, a total project net PCB load of 670 kg Tri+PCBs +/- 25% was shown to have only a negligible impact on the Lower Hudson. Additionally, a model simulation of the Upper Hudson showed that similar loads in the Stillwater/Waterford pool did not substantively impact this reach of the Upper Hudson.	No	The Panel agrees in concept with the need to re-evaluate the numerical load criteria for both the Upper and Lower Hudson River. However, because EPA could not adequately define the environmental consequences of near-field and far-field resuspension and release loads, the Panel maintains that there is insufficient information available to establish revised numerical criteria for the Resuspension EPS. Insufficient Phase 1 data specific to near-field PCB releases exist to support the development of a revised Resuspension EPS.
Near-Field Net Suspended Solids Concentration	Reduce the near- field net suspended solids (TSS) levels for Phase 2.	Net increase of 50 mg/L TSS above ambient (upstream) conditions at a location: ♦ 300 m downstream of the dredging operation, or ♦ 150 m downstream from any TSS control measure.	Conditions during Phase 1 showed that current suspended solids criteria are too high to be useful and lower criteria are achievable and needed to monitor solids transport and releases. Proposed levels are	No	The Panel does not agree with EPA's rationale for a reduced TSS standard. The 2004 EPS standards were achieved in Phase 1, and the data clearly showed that TSS is not a reliable predictor of

EPA Proposed Modifications to Resuspension EPS				Panel Finding	
Standard	**Proposed Change**	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
		Sustained TSS of 100 mg/L above ambient (upstream) conditions at near-field stations located: ♦ to the side of dredging operations, or ♦ 100 m downstream of dredging operations.	consistent with observations of suspended solids during Phase 1 and should not result in the need for more stringent practices than applied in Phase 1 with respect to suspended solids control.		PCB release. The Panel concluded that any further restriction on TSS loading unnecessarily burdens productivity. The 2004 TSS standard should be maintained. The Panel agrees with discontinuing the use of turbidity data for Phase 2. The collection of near-field TSS data should be continued at least through Year 1 of Phase 2 (along with near-field PCBs) to facilitate model calibration. However, the EPS should clarify how the TSS data will be used (to quantify near-field sediment deposition rates and chemical resuspension, near-field deposition, and far-field release rates). This assessment may be particularly relevant to non-PCB chemicals, such as metals.

EPA Proposed Modifications to Resuspension EPS				Panel Finding	
Standard	**Proposed Change**	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
Far-Field PCB Concentration	Use the 500 ng/L threshold at Thompson Island as a trigger to require operational changes, but not necessarily an operational shutdown, at EPA's discretion.	N/A	The towns of Waterford and Halfmoon are not going to use the Hudson River as their source of potable water during the dredging period. For this reason, the drinking water quality basis for the 500 ng/L is alleviated. However, this level is still seen as important to control the mass loading to the Lower Hudson River and will be maintained to help EPA require operational changes when resuspension is elevated.	Yes	Far-Field EPS criteria for PCB concentration should be maintained and measured at all stations. However, because drinking water sources have been relocated to avoid drawing from the Hudson River during dredging, the Panel recommends that the 500 ng/L concentration limit be used operationally, to help manage dredging operations but not necessarily to shut down operations.
	Maintain the water column Control Level of 350 ng/L for discretionary use by EPA to require (as opposed to merely recommend) appropriate operational changes.	N/A	Using 350 ng/L as a Control Level will help ensure that resuspension does not exceed acceptable levels.	No	The 350 ng/L control level should be maintained as an advisory level only. To do otherwise would unnecessarily impact productivity.

Table 4. Summary of GE's proposed modifications to the Resuspension EPS

GE Proposed Modifications to the Resuspension EPS				Panel Finding	
Standard	**Proposed Change**	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
Far-Field Net PCB Load	The load standard must be determined correctly. Correct numerical load criteria should be developed for the Upper and Lower Hudson. The standard for the Upper Hudson should be based on the benefits to Upper River fish. The standard for the Lower Hudson should be based on a comparison to the load that would occur from MNA. (from GE Report Section 9.1.2.1, page 177) The PCB fate and bioaccumulation models should be used to determine allowable loads for the Upper and Lower Hudson considering the full impact of resuspension, including redeposition. The numbers should be based on minimizing impacts to fish in the Upper Hudson and ensuring that dredging accrues a benefit in the Lower Hudson.	A firm, not to exceed 1,200 kg Total PCB limit, subject to downward adjustment based on redeposition. The net load should be assessed for the entire year, not just during the dredging season, to account for redeposition. The load standard must remain true to its original purpose, to ensure that dredging does not release more PCBs to the river than MNA. The load standard was not, and should not be, based on a percentage of the PCB mass encountered during dredging. A standard based on the percentage of PCB mass allows more PCB to be sent downriver than MNA, and thus eliminates the benefits of dredging originally projected by EPA. The load standard must be a hard cap. EPA originally set this standard as a fixed	New projections of PCB load for natural recovery and dredging that are made using an improved model that is not biased relative to the loads measured during the baseline monitoring program. From these new projections, a determination can be made of the maximum resuspension load that would allow dredging to achieve a net reduction in PCB load to the lower river within the next 20 or so years and preserve most of the benefits to Upper Hudson fish.	No	The Panel agrees in concept with the need to re-evaluate the numerical load standards for both the Upper and Lower Hudson River. The Panel was not provided the information necessary to evaluate the model(s) used by GE to propose these specific numbers. In addition, the Panel believes that there are insufficient Phase 1 data specific to near-field PCB releases to support appropriate calibration and validation of any model.

GE Proposed Modifications to the Resuspension EPS				Panel Finding	
Standard	**Proposed Change**	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
		number, and it should remain fixed to ensure the remedy achieves its intended benefits.			
Near-Field Net Suspended Solids Concentration	♦ Discontinue the use of near-real time turbidity data to estimate TSS concentrations using the TSS/turbidity relationship. ♦ Discontinue TSS compliance monitoring in the near-field. ♦ Limit TSS sample collection to 24-hour composite samples that accompany PCB samples.	---	---	Yes	The Panel agrees with discontinuing the use of turbidity data for Phase 2. Near-field TSS should be continued at least through Year 1 of Phase 2 (along with near-field PCBs) in order to provide data for model calibration.
Far-Field PCB Concentration	GE does not propose any change to the 500 ng/L resuspension standard for PCB concentration in the water column. That standard is based on EPA's drinking water standard for PCBs and should remain in place for Phase 2. (Page 178)	---	---	Yes	The Panel agrees that the PCB chemical concentration EPS should be maintained for all far-field stations.

CHARGE QUESTION 3. If the experience in Phase 1 and other evidence before the Panel does not show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2, can the Phase 1 Engineering Performance Standards be modified so that they could consistently be met in Phase 2, and, if so, how?

Phase 1 demonstrates that the resuspension and redeposition mechanisms operating at the site are not well understood. Further investigations are necessary to develop a Resuspension EPS that is protective of the resources identified in the ROD and EPS, and that can be consistently be met in Phase 2. This standard must address the influence of PCB resuspension and release on recovery in both the upper and lower portions of the river.

The Panel recommends an expanded adaptive management approach be applied to all EPS in order to achieve the expected benefits of the project in Year 2 and in subsequent years. The following standards apply to Year 1 of Phase 2. During Year 1, the Panel recommends collecting additional data to support the development of a meaningful and environmentally relevant Resuspension EPS that will be applied for the remainder of the project duration. The Panel also recommends that the expanded adaptive management approach allow for continuous modification of the EPS to optimize remedial operations and to limit unintended consequences and adverse environmental impacts from those operations. To achieve these goals, the Panel recommends the following:

- Establish a common method for analyzing and presenting PCB data. Tri+ is the PCB measurement basis used in the ROD; the Panel proposes that any future standard for resuspension be expressed as Tri+PCB, but that both total and Tri+PCBs be reported routinely. The exception is the far-field concentration of 500 ng/L TPCB.
- Collect additional near-field and far-field data in Year 1 of Phase 2 to relate operational activities to sediment resuspension and PCB release.
- Set an interim resuspension standard for Year 1 of Phase 2 that Tri+PCB release rates measured at the TIP Dam and Waterford to 2 percent and 1 percent, respectively, of the Tri+PCB mass removed.
- Develop, calibrate, and validate a project-specific fate and transport model to set near-field and far-field resuspension criteria.
- Adaptively manage all EPS to achieve the expected benefits of the project in Year 2. With respect to the Resuspension EPS, based on the Phase 2 Year 1 results, EPA should establish appropriate and achievable criteria that balance the benefits of reduced risks within the dredging footprint against the detriments of increased downstream transport and associated risks.
- Use the 500 ng/L total PCB threshold at the far-field monitoring stations as a trigger to consider operational changes, not operational shutdown. Drop the 350 ng/L Control Level.
- Continue use of near-field TSS compliance monitoring and levels at a minimum for completion of the Phase 1 CUs (9 - 16), and then re-evaluate the utility of TSS after Year 1 of Phase 2. Add PCB homolog measures to stations where TSS is being collected.
- Set allowable transport loads for Phase 2 based upon the findings from Year 1 of Phase 2.

Finding Rsp.3: The Phase 1 Resuspension EPS can be modified to be consistently met in Phase 2; however, neither the data nor the tool(s) presented to the Panel are adequate for defining a practicable standard that meets risk reduction goals.

The data presented to the Panel are insufficient for setting appropriate limits for resuspension and release that are protective of near-field and far-field habitat areas. The goal of a resuspension EPS should be to ensure that the dredging operation is performed as well as practicable, recognizing that any release of contaminants has the potential to cause short- and long-term environmental harm. If the environmental harm is unacceptable, then the environmental dredging protocols or remedial design must be changed. In the absence of additional BMPs, changes in dredging operations, and modification of the other EPS, the experience in Phase 1 provides relatively extensive far-field information; however, the data and tools are insufficient to determine the potential for short- and long-term environmental harm, as negligible information is provided regarding near-field and far-field PCB deposition. In addition, the data and tools are insufficient to determine what is practicable with additional BMPs, changes in dredging operations, and modification of the other EPS. To develop a useful resuspension standard, a single, defensible model is required. The Panel strongly recommends that EPA and GE work together to develop such a model to meet project needs.

The Panel does not have enough information to propose specific revisions to the Resuspension EPS, particularly for the portion of the river between the TIP Dam and Waterford. Given (1) the failure to achieve the 2004 EPS resuspension standard, (2) the absence of supportable projections by either party, and (3) the lack of near-field PCB data, the Panel has laid out a process that relies on interim performance standards to allow for additional data collection during Year 1 of Phase 2, followed by use of an adaptive management approach that includes updating the Resuspension EPS at the end of Year 1 of Phase 2 and for subsequent Phase 2 work. The goal is to produce a scientifically sound and environmentally protective Resuspension EPS that can be consistently met, simultaneously with the Residuals EPS and Productivity EPS. Thus, the Panel has defined an interim standard (to be used for 1 season) based upon observed PCB releases in Phase 1, and proposes that the Resuspension EPS be revised for Year 2 of Phase 2 based upon development of model-validated projections using the additional data to be collected in Year 1 of Phase 2. Further, the project should undergo annual review and should make use of an adaptive management approach that draws from the experience and data gained in each year's efforts to update the operational design and practice for the following year.

Finding Rsp.3-1: Inconsistent data produced by EPA and GE creates obstacles to establishing the validity of scientific conclusions and makes it difficult or impossible for community stakeholders and other interested parties to understand the impact of remediation activities. Therefore, EPA and GE should establish a common method for analyzing and presenting PCB data.

In the Phase 1 report, GE updated the correction factor used to adjust the PCB concentrations of some of the peaks measured by the modified Green Bay Method; EPA did not. The Panel had to expend considerable resources during the evaluation process to compare results between the 2 reports. Inconsistent expression of the Resuspension EPS (as Total PCB *and* Tri+PCBs) also made data analysis difficult. The ratio of Tri+PCBs to total PCBs is not consistent throughout the site, ranging from less than 20 percent to 35 percent Tri+PCB. Understanding the fraction of PCBs that are likely to be volatilized during near-field and far-field transport (i.e., primarily mono and di-PCBs) and the fraction that may potentially redeposit further downstream or that can potentially become available for bioaccumulation is critical to revising resuspension criteria to be protective of near-field and far-field receptors.

EPA and GE should come to an agreement as to the appropriate summation method for PCB data prior to undertaking Phase 2. The same method must be used for all data comparisons, whether they precede Phase 1 or are based on Phase 1 and 2 results.

The Panel also recommends that any future standard for resuspension be expressed as Tri+PCB, but that both total and Tri+PCBs also be reported routinely. One exception to this rule may be the reporting of far-field aqueous concentrations for comparison to the 500 ng/L TPCB water quality standard, which may be expressed as TPCB, consistent with the respective "Applicable or Relevant and Appropriate Requirement."

Finding Rsp.3-2: There is insufficient information from Phase 1 upon which to base a revised Resuspension EPS.

Neither the tools nor the data presented to the Panel will support definitive revisions of the Resuspension EPS that will be protective of the near-field and far-field receptors identified in the ROD and the EPS. Additional near-field data are needed to develop tools that can relate the release mechanisms associated with various dredging unit processes to the increased risks of downstream transport. The Panel's finding that there is insufficient information from Phase 1 upon which to base a revised Resuspension EPS is based on the observation that insufficient data exists to correlate dredge-related operations to PCB resuspension release rates. Also, there is insufficient near-field data to quantify near-field deposition rates and corresponding impacts to human health and wildlife risks.

Finding Rsp.3-2.1: There is insufficient data to correlate dredge-related operations to PCB resuspension release rates.

Table 1 shows Phase 1 estimated and actual PCB resuspension release rates. In reviewing the documents leading up to implementation of Phase 1, the estimated transport loads varied with the estimation of the mass to be removed. The loss rate in the ROD was clearly underestimated at 0.13 percent, whereas the 2004 EPS predicted a range of loss rates (0.25 percent to 2 percent) prior to settling on a predicted rate of 1 percent. The estimates changed again during the Phase 1 Design and subsequent Phase 1 Performance Standard Compliance Plan, but all remained optimistic, predicting less than 1 percent release as per the 2004 EPS. Much higher release rates were reported by EPA and GE, based on the Phase 1 results. EPA and GE's Phase 1 reports differed with respect to the actual total mass removed and total PCBs released past the TIP and Waterford. However, the differences predicted by EPA and GE were largely due to different computational approaches for PCBs removed and for suspended PCB concentrations and resuspension loads. At TIP, EPA estimated a 2.2 percent loss compared to GE's 3.1 percent; at Waterford, EPA estimated a loss of 0.8 percent compared to GE's 1.2 percent. Despite the differences in resuspension release rates reported by EPA and GE, the range of losses reported at TIP (i.e., in 2 percent to 3 percent) are consistent with near-field losses reported in the engineering and scientific literature (Steuer 2000, Connolly et al. 2006).

EPA's Phase 1 Report Figure I-3-18a (Figure 2) presents a compelling representation of PCB mass removed against losses measured at TIP, Lock 5, and Waterford. The Panel confirmed EPA's mass removal projection with the data in GE's Appendix Table G-1c (Summary of Daily Bucket Analysis by CU), and the release rates at the TIP with GE's Figure 5.3-8 (Net Total PCB concentrations at Thompson Island) (Figure 3). While dredge releases are a contributor, there does not appear to be a consistent relationship between mass removed and mass lost solely by the physical act of dredging. Based on EPA's

analysis, PCB release is not solely controlled by dredging rates or mass removed. GE presented an empirical model that relies on rates of dredged PCB mass removed plus current velocities to predict water column PCB concentrations and resuspension release rates at Waterford. The model reasonably predicts Waterford PCB concentrations, suggesting a strong relationship between PCB mass removed and river flow velocities. This information, combined with EPA's analysis, suggests that river flow was a significant contributor to PCB resuspension and release. What is unclear is the extent to which the various aspects dredging operations contributed to resuspension and release (e.g., open CUs, scow operations, dredge rates, dredge depths, bucket size, bucket overflow, and other dredge-related unit processes, such as single lane advance of dredging downstream).

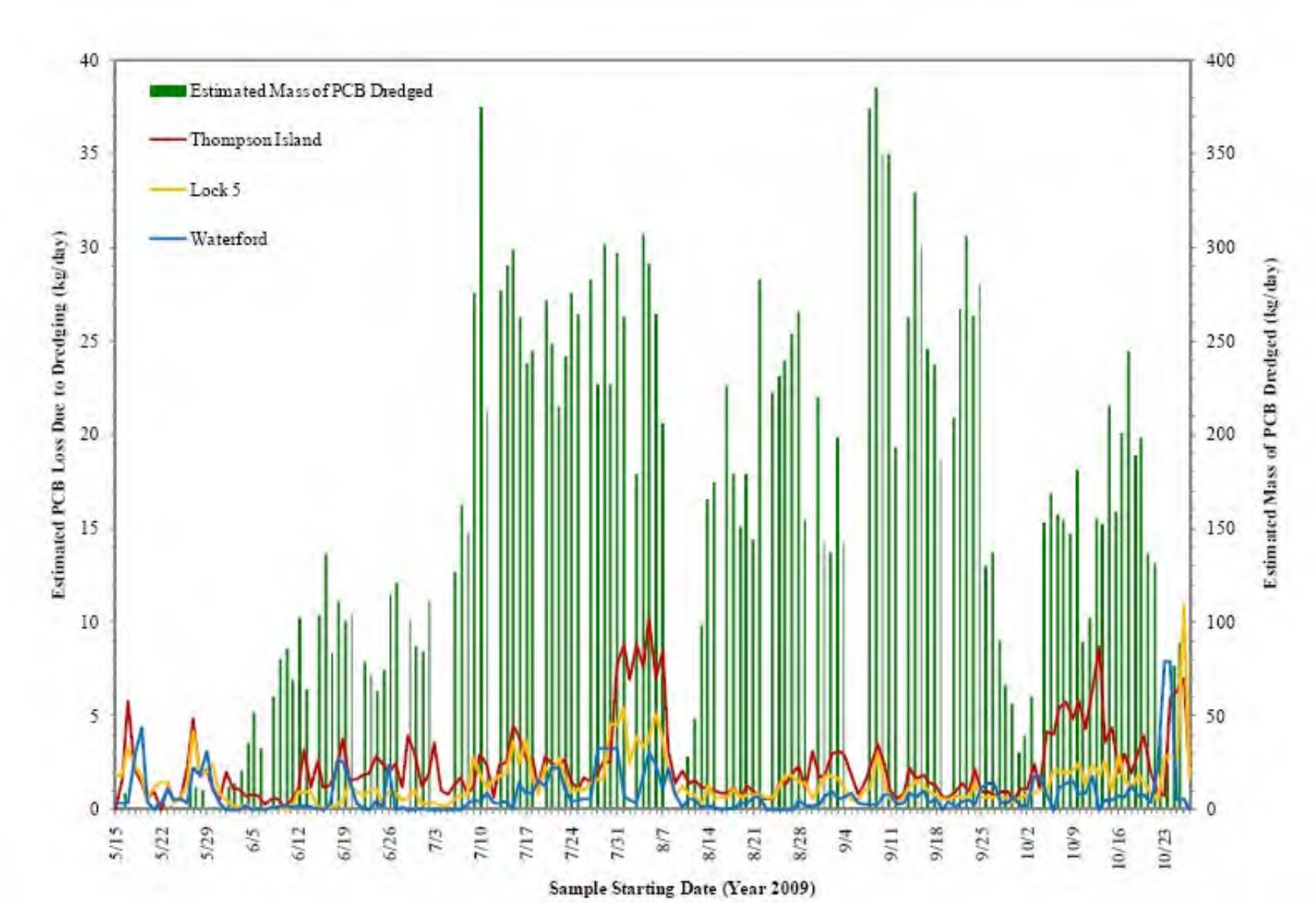

Figure 2: EPA Phase 1 Report March 2010, Figure I-3-18a

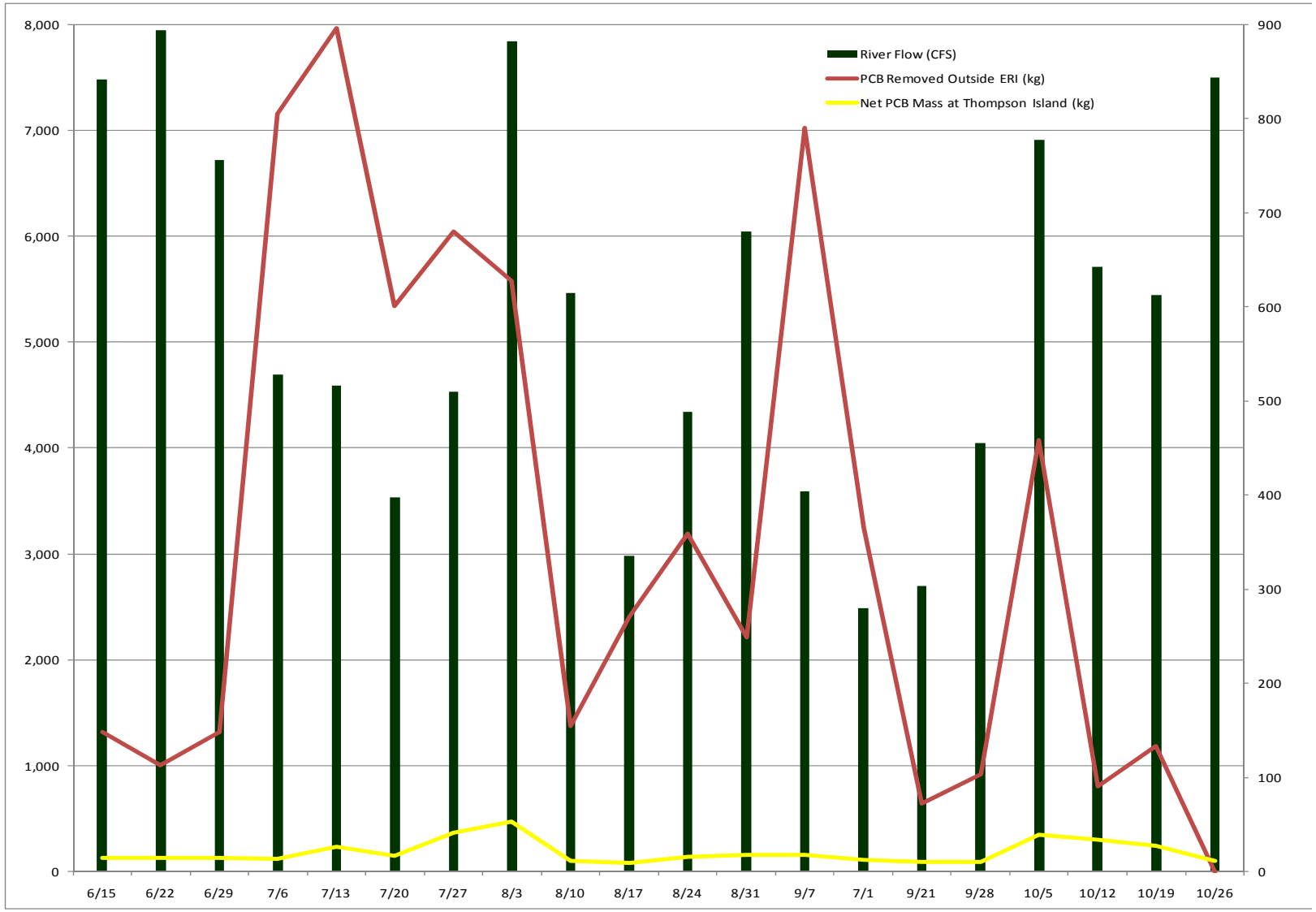

Figure 3: Plot of GE Phase 1 Report Table 5.3-2 Weekly Summary on PCB Removed Outside of East Rogers Island (ERI) and Net PCB Mass at the Thompson Island Station

Finding Rsp.3-2.2: There is insufficient near-field data to quantify near-field deposition rates and corresponding impacts to human health and wildlife risks.

A careful evaluation of the remedy release and redeposition processes is needed; these processes have the potential to undermine the benefits of the remedy that EPA set out in its 2002 ROD. The analyses presented by EPA and GE are neither complete nor compelling. EPA's evaluation compares the downstream transport of PCBs to the MNR trajectories for total PCB loads and fish; EPA does not address redeposition. GE's analysis of potential redeposition (presented in a handout at the meeting in Glens Falls, New York on May 5, 2010) contained a number of simplifying assumptions, and was expressed in terms of change to bed mass. The available data indicate that PCB deposition downstream of dredged areas may be significant.

The Panel evaluated PCB deposition in terms of potential changes in net PCB concentrations downstream of dredged areas using, in part, the assumptions provided by GE during the May 4-6, 2010 Public Meeting. While GE's analysis focused on the potential for newly deposited mass of PCBs, the Panel was interested in the deposited concentrations relative to conditions that currently exist. The following assumptions were employed.

- Area of impact between Thompson Island and Waterford: 2,228 acres (GE handout, May 4-6, 2010 Public Meeting)
- Depositional area between Thompson Island and Waterford: 347 acres (GE handout, May 4-6, 2010 Public Meeting)
- Depositional rate: 0.5 cm/yr (GE estimate, May 4-6, 2010 Public Meeting comment)
- Bulk Density: 1 g/cm^3 (based on Panel-experience with dredge residuals at other sites)
- Phase 1 mass unaccounted for: (GE Phase 1 Report estimate)
  - 506 kg release measured at Thompson Island Dam
  - 199 kg measured at Waterford
  - Delta is 306 mg/kg unaccounted for PCB mass during Phase 1

The unaccounted-for mass of 306 mg/kg would have been lost either through volatilization or deposition. The actual amount of PCB transfer to sediment is less than the amount lost from the water column, because some of the PCB will have volatilized to the air. Volatilization will favor the mono-, di-, and tri-chlorinated biphenyls. However, volatilization is not the primary loss mechanism in this reach of the river, based on the changes in PCB homologue composition with progress downstream. For the initial analysis, the Panel applied the simplifying assumption that all of the Phase 1 mass would be redeposited.

The Panel then predicted conditions after completion of the project, using the 5-year 2004 EPS project timeframe, the proposed new total load limits proposed by EPA (2,800 kgs) and GE (1,200 kg), the projected PCB mass estimates for the entire project, and the observed Phase 1 mass balance loss rates between TIP and Waterford (60 percent).

The results of this relatively simple analysis are presented in Table 5. Assuming deposition across all the available acres between the TIP and Waterford, the surface TPCB and Tri+PCB concentrations from Phase 1 redeposition alone would be 6.8 and 2.3 mg/kg, respectively. Assuming all of the material deposited solely within the known depositional acreage identified by GE, the concentrations would be 44 and 14.5 mg/kg. Notably, these PCB concentration estimates are relatively close to the sediment trap measurements reported by GE.

Table 5. Estimated Phase 1 and Phase 2 near-field PCB depositional potential between TIP and Waterford

Phase 1 or Phase 2 Condition	Depositional Area	Surface TPCB Concentration	Surface Tri^{+}PCB Concentration
Phase 1	2,228 total acres	6.8 mg/kg	2.3 mg/kg
Upper 0.5 cm following Phase 1	347 depositional acres	44 mg/kg	14.5 mg/kg
EPA Target of 2000 kg			
Upper 2.5 cm following Phase 2	2,228 total acres	7.5 mg/kg	2.5 mg/kg
Assumes 60% of 2000 kg release	347 depositional acres	48 mg/kg	16 mg/kg
GE Target of 1200 kg			
Upper 2.5 cm following Phase 2	2,228 total acres	3.2 mg/kg	1.1 mg/kg
Assumes 60% of 1200 kg release	347 depositional acres	20.5 mg/kg	6.8 mg/kg

The Panel then considered how these projected concentrations would compare to those currently downstream of the TIP. Using a table provided in GE's May 5, 2010, handout (Table 6), the projected concentrations are within the range of the surface-weighted average concentrations reported by Reach for both total and Tri+PCBs. Focusing solely on the depositional areas—with the understanding that most of those would be dredged areas—again, the concentrations ranges projected and observed are similar.

Table 6. GE handout to Peer Review Panel May 5, 2010
(Table 3. Average 0 - 2" PCB Concentration in Dredge and Non-Dredge Areas)

Reach	Tri+PCB(mg/kg)		TotalPCB(mg/kg)	
	Dredge	Non-dredge	Dredge	Non-dredge
8	Phase1: 26.7 Phase 2: 22.4	3.6	Phase 1: 83.6 Phase 2: 57.6	7.3
7	24.4	4.1	49.3	7.9
6	21.3	3.9	49.3	7.5
5	5.4	1.7	10.6	2.8
4	12.4	1.8	31.0	3.4
3	6.4	2.2	9.8	3.5
2	8.1	3.2	11.1	4.7
1	2.1	0.5	3.8	0.8

The Panel does not find its own analysis particularly compelling or satisfying. There are too many assumptions, areas over which data were averaged, are too large and too many variables are missing to provide confidence upon which to draw conclusions or to set long-term project resuspension criteria. Within the TIP (Reach 8), some portion of the PCB will have deposited within the Phase 2 dredging footprint and thus will be recaptured. However, some of the PCB will deposit outside the dredging footprint, and the extent of such deposition is unknown. Also, as future dredging progresses downstream, more of the resuspended PCB mass will necessarily deposit outside the dredging footprint. Assuming deposition over a large area ignores what may be very important increases in the near-field PCB concentrations in Phase 2 where the CUs are more spread out. The redeposited material will, at least for a time, remain unconsolidated and available for further migration and may contribute to increased or sustained elevated fish tissues concentrations. The data and evidence needed to credibly and transparently balance the benefits and adverse consequences of different configurations of remedial action and operation simply do not exist. Additional information that would test this condition is discussed in the next finding.

Finding Rsp.3-3: Collect additional near-field and far-field data in Year 1 of Phase 2 to relate operational activities to sediment resuspension and release.

The Panel recommends that EPA and GE develop a comprehensive conceptual model that relates operational activities to resuspension of sediments, chemical release of PCBs, and the production of residual contaminated sediment both within and without the dredging prism. This conceptual model should then be used as a basis for developing a quantitative understanding that facilitates credible predictions about the consequences of operational practices over time. Proposed modifications for the EPS or operational practices made by GE and EPA are largely based on speculations regarding key processes contributing to PCB release. The speculative nature of these proposals is due to the incompleteness of Phase 1 monitoring data and the inability to integrate those data using a comprehensive modeling tool that would provide the technical basis for meaningful adaptive management. The following specific tasks are recommended to further refine the Resuspension EPS.

- Establish TIP and Waterford monitoring programs that are adequately designed to monitor load releases during dredging and that correlate releases to near-field dredging activities and near-field data.
- Collect near-field data surrounding the various dredging related activities (e.g., monitoring releases associated with open CUs, scow operations, dredge rate of advance and other dredge-related unit processes).

Finding Rsp.3-4: Set an interim resuspension standard for Year 1 of Phase 2 that tri+PCB release rates measured at the TIP Dam and Waterford to 2 percent and 1 percent, respectively, of the Tri+PCB mass removed.

A revised resuspension standard should recognize the potential for increased risks associated with downstream transport (including between TIP and Waterford) and should be modified as necessary to address those risks. Based on the Phase 1 results, 2 percent and 1 percent release rates at TIP and Waterford, respectively, are reasonably aggressive target values for mass released during dredging activities. The Panel does not recommend interrupting dredging activities if the targets are not achieved during Year 1 of Phase 2. The goal of the interim standards is to establish baseline targets during Year 1

of Phase 2 and to allow dredging to recommence in 2011, while near-field and far-field data are collected.

Finding Rsp.3-5: A sound remediation process includes a rigorous, formalized process for adaptively managing the project over time to address uncertainties affecting remedial objectives, operations, and performance standards. Therefore, EPA and GE should jointly develop a formal adaptive management plan.

The results of Phase 1 dredging tangibly demonstrate that there are practical limits to our collective ability to predict outcomes for sediment remediation projects. The physical, chemical, and biological processes involved are complex and the uncertainties associated with data and models relevant to those processes are significant. This reality has been amply demonstrated during Phase 1 as well as at contaminated sediment remediation projects across the country.

The pragmatic approach for addressing this reality is to establish an expanded, rigorous, and formalized process for implementing adaptive management. The essential elements for such an adaptive management process are: 1) a comprehensive conceptual model that incorporates remedial activities and regular updating as new information about the system is gained; 2) a formal, mathematical representation of this conceptual model that is used as the basis of remedial design and directing operational practices; 3) operational and performance monitoring that is targeted to address key processes and uncertainties in the conceptual and mathematical models; 4) a commitment to a formal process for capturing information about the remedial system and incorporating that information in the conceptual and mathematical models; and 5) using the integrated understanding provided by the modeling to inform decisions to revise remedial designs and operations as necessary and indicated by the assembled evidence.

A central component of this adaptive management plan will include development of a process for adaptively managing all EPS to achieve the expected benefits of the project. With respect to the Resuspension EPS, based on the Phase 2 Year 1 results, EPA should establish appropriate and achievable criteria that balance the benefits of reducing risks through contaminated sediment removal against the detriments of increased downstream transport of PCBs and the risks produced through that redistribution of PCBs within the river.

Further, the Panel recommends that development of a revised Resuspension EPS include the following.

- The revised Resuspension EPS should be consistent with current dredging practices and practicable limits to reducing resuspension during dredging. This must include an improved sediment characterization for all remaining CUs (i.e., establish high confidence for all CUs) and a more streamlined method for closing CUs in a timely fashion. The Panel recognizes that limited approaches exist for reducing resuspension, including improved BMPs with respect to dredge operations, more rapid closure of CUs, and reduced dredging volumes.
- The Panel does not support the use of silt curtains or other physical barriers to control resuspension release rates given the time requirements and logistical complexities associated with their use and their limited effectiveness in constraining transport of sediment and PCB release.
- The Panel recommends that the project continue to make use of an external panel to help in focusing efforts to establish revised performance standards for the remainder of Phase 2. This effort

should not entail the submittal of new reports by EPA and GE. Instead, the Panel recommends coordinating the analysis and interpretation of the Phase 2 Year 1 data while engaging with the external panel in an iterative manner so as to accelerate the development of a revised Resuspension EPS.

Finding Rsp.3-6: Use the 500 ng/L total PCB threshold at the far-field monitoring stations as a trigger to consider operational changes, not shutdown. Drop the 350 ng/L Control Level.

The goal of the Far-Field PCB Concentration standard established in 2004 was to prevent water supplies for the towns of Waterford, Halfmoon, and Stillwater from exceeding the PCB MCL. However, Waterford and Halfmoon now have an alternate connection to Troy. Stillwater, which draws its water from an aquifer adjacent to the river, has an adequate treatment system for PCBs. Regardless, the Panel agrees it is important to maintain the MCL criterion as part of any revised standard, consistent with the original intent of the EPS that "no public water supplies will be adversely impacted by the remediation, regardless of a given water treatment plant's (WTP's) ability to treat PCB-bearing water."

The Panel recommends maintaining the 500 ng/L total PCB threshold at the far-field monitoring station as a *trigger* to consider operational changes. This standard recognizes that the 500 ng/L remains an "Applicable or Relevant and Appropriate Requirement" and is based on protection of human health in drinking water. However, because the source of drinking water was relocated from the Hudson River, at least for the duration of the dredging work, the standard can be relaxed so as not to require an operational shutdown in the event of a short-term exceedance, and the 350 ng/L control level is unnecessary and should be eliminated.

Finding Rsp.3-7: The basis for the TSS criterion must be reevaluated. Continue near-field TSS compliance monitoring and levels at a minimum for completion of the Phase 1 CUs (9 - 16), and then re-evaluate the utility of TSS after Year 1 of Phase 2. Add PCB homolog measures to stations where TSS is being collected.

The Phase 1 data demonstrate a complete lack of statistical significance between TSS and the transport of PCBs from removal operations during Phase 1. The transport of non-particulate phase PCB clearly indicates that TSS concentration cannot be a reliable indicator of PCB releases as envisioned in the 2004 EPS. Thus, the TSS measurements provided no useful information for managing far-field PCB resuspension and release during dredging operations.

The 2004 EPS numeric TSS standards are adequate for Phase 2, Year 1, and for completing the targeted Phase 1 CUs. The TSS standard should be evaluated in relation to the results of the revised monitoring program, including enhanced near-field monitoring, which the Panel believes will aid in developing a more complete understanding of relevant resuspension processes and what actions should be taken to manage those processes. The reevaluation of the TSS criterion must also consider its relationship to the Residuals EPS and Productivity EPS during the 2011 dredging season.

EPA should discontinue the collection and use of turbidity data.

Finding Rsp.3-8: Transport loads should be based on empirical data as well as risk reduction targets. Set allowable transport loads for Phase 2 based upon the findings from Year 1 of Phase 2.

Insufficient information was provided to the Panel to assess the effects of PCB resuspension and transport on fish tissue concentrations; the only tissue data available were taken during Phase 1, relatively soon after cessation of operations in 2009—the spring 2010 data were not available to the

Panel. Available data indicated short-term, transient exposures due to water column PCB concentrations during dredging. The potential for long-term exposures to increased surface sediment PCB concentrations, if significant, is likely to lead to more substantial and long-lasting impacts.

Because PCB concentrations in fish take time to equilibrate, tissue concentrations may continue to increase after dredging, so monitoring of fish should continue, and should include near-field and far-field locations.

Resuspension criteria for near-field and far-field PCB load targets for Phase 2 based on limiting impacts to fish in the Upper Hudson and ensuring that dredging accrues a benefit to both the Upper and Lower Hudson need to be developed. The sediment and fish tissue data collected in 2010, along with near-field and continued far-field PCB measures, will help determine the relationship between PCB releases and downstream effects and update MNR and remedy forecasts, and to develop a PCB load standard that ensures a net environmental benefit from the remedy.

MNR comparisons also should be based on surface sediment recovery rates inside and outside the CUs, and not on a cumulative mass loading to a downstream location alone. Resuspension criteria should be based on the changes occurring in surface sediment concentrations due to remedial action, both in the CUs and in areas external to and downstream of the CUs; this is as opposed to basing resuspension criteria solely on far-field release load calculations. At a minimum, changes to sediment concentrations in all portions of the river must be calculated and measured. The fate, transport, and risk model must enable EPA and GE to understand the implications of operational changes on long-term recovery rates.

An adequate standard is one which achieves the goal articulated in the 2004 EPS, that is, the maximum allowable load must result in a net reduction of transport to surface sediments in the upper and lower Hudson compared to MNR within a timeframe that corresponds with the ROD (i.e., 20-25 years). Development of the standard requires calculating the following,

- Transported load that would cause a perceptible increase in fish tissue concentrations in the lower Hudson (short-term and long-term), and compare those to MNR
- Transported load that would compromise risk reduction in the upper Hudson, due to contamination of non-footprint areas
- Transported load associated with excessive surface water concentrations

CHARGE QUESTION 4. If EPA and/or GE has proposed modifications to the monitoring and sampling program for Phase 2, are the proposed modifications adequate and practicable for determining whether the Phase 2 Engineering Performance Standards will be met?

Both EPA and GE proposed changes to the EPS with concurrent changes to the monitoring and sampling program for Phase 2. However, the Panel finds that it will not be practicable to consistently and simultaneously meet the EPS being proposed by either party and, thus, cannot make a finding regarding the monitoring and sampling programs relative to these proposed standards except for those items that have been specifically addressed under Charge Question 2, above. Rather, the Panel addressed Question 4 relative to the modified EPS and processes recommended by the Panel in response to Charge Question 3.

Finding Rsp.4-1: Monitor residuals outside the dredge prism.

Phase 1 inadequately evaluated the generation of residuals outside of the dredge prisms and CUs. Near-field and far-field PCB deposition have the potential to adversely increase ecological and human health risks. EPA and GE should evaluate whether off-CU deposits have the potential to increase risks to levels that are unacceptable when compared to the reduced risks associated with dredging and backfilling.

EPA and GE should establish a residuals monitoring program that evaluates the potential for near-field PCB deposition outside of dredged CUs. The program should test the potential for near-field and far-field off site deposition and generation of dredged residuals in low-, moderate-, and high-flow areas downstream of dredged CUs. Continued monitoring requirements after Year 1 of the Phase 2 dredging program should be determined by EPA and GE, based on the results of Phase 2, Year 1, and subsequent years. Based on the off site/off-prism monitoring results, EPA and GE may adjust BMPs and/or dredge volumes if the risks associated with the generation of residuals outweigh the reduced risks associated with dredging.

The near-field and far-field monitoring results should be integrated into updated site-specific sediment transport and risk exposure models to consider the role of generated residuals outside of the prism on ecological and human health risks. Off-CU residual deposits should not adversely increase baseline surface sediment Tri+PCBs concentrations on off-CU areas, such that post-dredge recovery rates are slower than would be achieved via MNR. This evaluation should not be based on a comparison of cumulative loads, but instead should be based on long-term potential fish exposures associated with surface sediment deposits and surface water PCB resuspension and releases, and should include a combined assessment of dredged and undredged areas over a 25-year period.

Finding Rsp.4-2: The revised Resuspension EPS must be based on an updated conceptual model of the fate and transport of PCBs during dredging, and the ecological risks associated with releases during remedial operations. Data collection in Phase 1 was inadequate to calibrate and validate such a model. Therefore, develop a project-specific fate, transport, and bioaccumulation model (to be used in common by EPA and GE) to set near-field and far-field resuspension criteria.

Developing an appropriate and achievable Resuspension EPS requires balancing the benefits of reduced risks within the dredging footprint against the risks associated with increased downstream transport and air releases. Currently, the project lacks a transparent, scientifically sound and state-of-the-art model that adequately addresses dredging-related release mechanisms and contributions to downstream transport from remedial activities, potential for deposition of released PCBs associated with the entire

project area, and impacts to fish tissue during and after completion of the remedy. Resuspension, release, and residual-formation processes must be clearly represented to ensure that operational decisions made over subsequent phases of the project, and the consequences of those decisions, are transparent to the public, stakeholder communities, and EPA and GE. As discussed previously, the HUDTOX FISHRAND models used in the development of the ROD and EPS do not provide a reliable basis for evaluating and setting release loads. The results of the mechanistic modeling presented to the Panel by GE is insufficient to make near-field operational decisions to control resuspension, release, and residuals, and Phase 1 data collection were insufficient to compare MNR and dredge-related impacts on or benefits to the environment.

The Panel found other critical problems with data collection, models, and analysis in Phase 1, such as:

- Insufficient near-field data were collected during Phase 1, making it difficult to understand cause-and-effect relationships between the various dredging-related activities and downstream resuspension and release.
- EPA's "Multiple Regression Model" that attempts to simultaneously consider "over 28 dredging-related variables…for association with water column concentrations"—while potentially helpful to screen important variables for future monitoring that may influence resuspension (e.g., for further monitoring)—provides insufficient causal evidence for explaining relationships between near-field activities and far-field releases.
- The mechanistic modeling results presented by GE that correlated release with PCB mass removed and flow rates present a reasonable correlation between measured and model-predicted releases, but fail to explain the relationship between the multiple dredging-related processes and release rates. Figure I-3-18a (PCB Mass Dredged and PCB Mass Lost to Water Column at Far-field Stations during Phase 1) of the March 2010 EPA report indicates that the PCB mass removed is not well correlated to PCB release rates at TIP, Lock 5, and Waterford. The GE modeling results suggest that river currents contributed significantly to release rates. This important finding suggests that factors such as unclosed CUs may have contributed substantially to resuspension and release rates.

There is a very real need to set an allowable load limit for the Hudson River dredging project, but neither the data nor tool(s) needed to do so currently exist. To that end, the project must develop a set of models that incorporate hydrodynamics, sediment transport, fate and transport of PCBs, and bioaccumulation of PCBs in the Upper Hudson River (from Fort Edward to Troy Dam).

- Use a single model, developed collectively by EPA and GE; the GE model may be a useful foundation for this model. The model structure and parameterization must be agreed to between EPA and GE. The model must be peer reviewed by an expert panel once EPA and GE complete its development. Similar arrangements have been successful at other Superfund Sites, including the Passaic River, the Lower Duwamish Waterway (WA), and the Lower Willamette River (OR).

The model should meet the following requirements:

- For transparency, all code must be made available to the full development team (GE and EPA) and the Peer Review Panel.

- Establish a steering team made up of technical representatives from GE and EPA in order to ensure the best application of scientific and engineering principles.
- Apply the Panel's changes to the Residuals and Productivity EPS in Year 1 of Phase 2.
- Use data from Phase 1 for initial model calibration, but incorporate data from Year 1 of Phase 2 for final model calibration and validation.
- Complete model efforts and projections in a reasonable timeframe, in order to set criteria for Year 2 and beyond.

The Panel further recommends the following be considered in model development.

- The model should be populated with variables that reasonably predict real-world conditions. In other words, the goal of the model should not be to develop overly conservative estimates to overcome uncertainties, but rather to develop predictions based on reasonable and defensible assumptions and variable parameters so that the model can be useful for adaptive management.
- The model should reflect uncertainties associated with the data and the model's ability to predict future conditions. To this end, the model should predict the range of results associated with MNR predictions and with dredging-related releases. (A single number, whether 1200 kg or 2000 kg, inadequately represents the complexity of the system and uncertainties associated with data collection, chemical analysis, modeling, and interpretation of results.)
- Comparing the effectiveness of dredging with MNR is reasonable; dredging should not make conditions worse than MNR. However, comparisons made to date have been inadequate, particularly the cumulative load comparison between MNR and dredging responses. Therefore, the model should be designed to predict surface sediment concentrations, fish PCB uptake, and long-term recovery for the entire river, and should include near-field and far-field reaches of the river, including those areas that undergo dredging. The goal should be to ensure that the long-term trajectory of PCB-related impacts on the river, during and after dredging, does not exceed the impacts associated with baseline conditions.

Finally, the Panel recommends that an independent review of the projections with results from Year 1 of Phase 2 be conducted, similar to that conducted after Phase 1. This Panel is willing to participate in independent review for the model, project, and EPS for the duration of Phase 2; however, the independent review team also should include a modeling expert.

4 RESIDUALS

CHARGE QUESTION 1. Does the experience in Phase 1 show that each of the Phase 1 Engineering Performance Standards can consistently be met individually and simultaneously?

Finding Rdl.1-1: Phase 1 did not achieve the 2004 Residuals EPS.

The 2004 Phase 1 Residuals EPS for the Hudson River PCBs Superfund Site were not consistently met. The Residuals EPS assumed the removal of all inventory with a maximum of 2 passes, plus a maximum of 2 redredging passes to address generated residuals (with a potential additional pass to remove inventory). Poorly defined DoC resulted in more inventory than expected, causing disagreement between EPA and GE regarding the effectiveness of inventory removal. Because inventory was not sufficiently characterized before construction commenced, the Panel believes that the Residuals EPS were not truly tested as envisioned.

Residuals management decisions were confounded because the Residuals EPS were not intended to address excess inventory. Even with inventory left behind, CUs were not closed in a timely manner, only about half of the CUs designated for Phase 1 were finished, and PCB concentrations left behind after dredging were higher than allowed in the Residuals EPS. Thus, residuals management during Phase 1 required multiple production passes and the CUs were open longer than had been planned.

EPA suggests that the additional inventory passes were entirely due to poor characterization of the DoC and that the Residuals EPS were achieved once the inventory was removed. The Panel disagrees with this assessment and believes that inventory and generated residuals are linked and should be managed in concert.

Finding Rdl.1-2: The experience in Phase 1 does not show that the Residuals EPS could be met simultaneously with the Productivity and Resuspension EPS.

During Phase 1, 18 CUs were targeted for dredging. With a 150-180-day dredging season, 1 CU should have been closed every 8-10 days. In actuality, only 10 CUs were dredged; therefore, 1 CU should have been closed every 15 to 18 days. Further, upstream CUs should have been closed prior to downstream ones to avoid recontamination of closed CUs. With 3 or 4 dredges at work, a single CU should not have been open for more than 35 days. In fact, however, Phase 1 CUs were open for an average of 113 days.

Only 1 CU (CU 17) was closed in adherence with the Residuals EPS (i.e., only CU 17 was backfilled after having achieved residuals less than 1 ppm Tri+PCBs, as defined in the upper 6-inch sediment surface). Seven CUs were closed by capping at least a portion of their respective areas because it was not possible to achieve a residual Tri+PCBs concentration of less than 1 ppm in the upper 6 inches within the maximum allowable number of post-inventory dredge passes. The other 2 CUs were forced to be closed via capping as the end of the season approached. About 25 percent of the total area in these CUs were closed out of compliance with the Residuals EPS and would have required further dredging if there had been enough time. While additional redredging could have reduced Tri+PCBs concentrations in residuals, it is unclear that redredging would have achieved levels required by the Resuspension EPS.

In the attempt to meet the Residuals EPS, a disturbed residuals layer was created on the sediment surface, which was subject to erosion by currents and vessel traffic. Erosion of the residuals layer was likely a significant source of resuspension, possibly accounting for as much as 75 percent of PCB losses according to the data shown in Table 7. The losses from CU 18 were estimated to be at least 75 percent less than the losses from the West Rogers Island CUs; even the East Rogers Islands CUs, which had vessel traffic, were estimated to be at least 60 percent less than the losses from the West Rogers Island CUs.

Table 7. Summary of PCB losses

Location*	Conditions	Approximate PCB Loss, %
CU 18	Very restricted flow and traffic by the use of a sheet pile enclosure	0.5 to 1
East Rogers Island CUs	Restricted flow by use of rock dike but unrestricted traffic	1.3 to 1.7
West Rogers Island CUs	Unrestricted flow and traffic Below a weekly average flow at Fort Edward of 5,000 cfs At flows greater than 6,000 cfs	Average: 3.5 to 4.2 Between 0.9 and 4.3, with an average of 2.1 Between 4.6 and 7.4, with an average of 6.1

* Data sources include: GE May Deliberations presentation, GE Chapter 5 key findings, GE 5.3.2.2 Effect of River Velocity on PCB Release Rate, and GE 5.7.1 Overall Extent of PCB Release.

Finding Rdl.1-2.1: Inaccurate DoC makes it unlikely that the Residuals EPS can be met.

Phase 1 demonstrated significant challenges associated with the Residuals EPS:

- Insufficient distinction was made between generated residuals and undisturbed residuals (termed inventory), mainly because the DoC was inadequately delineated in most CUs.
- As applied in the decision flowchart, the Residuals EPS defines residuals as inventory whenever the surface-sediment average concentration measured greater than 6 ppm Tri+PCBs after dredging; however, the reasoning for this distinction was not clearly grounded in science or risk management.
- Coring was conducted using ineffective techniques for the conditions encountered, particularly debris and sediment types.
- Data from cores with poor recoveries (i.e., material recovered was less than the depth that the core was pushed) and incomplete core penetration into soft sediments (i.e., there were soft sediments below the core sampling depth) were fed into the Terrain Model that inaccurately predicted dredge elevations.
- Data input to the Terrain Model were not tied to absolute elevations. Instead, the model output was defined in terms of depth below sediment surface. As a result, the DoC changed with surface-sediment elevation changes, contributing to the inaccuracy of the Terrain Model.

Finding Rdl.1-2.2: The experience in Phase 1 does not show that the Residuals EPS can be met for Phase 2.

For Phase 2, there is low confidence in the DoC for approximately 40 percent of the areas to be dredged. Furthermore, the lack of vertical control on DoC elevations—as discussed previously—increases the uncertainty associated with the high-confidence cores, which may be inadequate to accurately establish the DoC elevation using the Terrain Model.

According to EPA, only in CU4 did the average Tri+PCB concentration consistently decline with each dredge pass. This underscores the difficulty associated with achieving target PCB concentrations via repeated dredge passes, and the importance of establishing an accurate DoC based on core results that confidently determine the absolute elevation of the 1 ppm Total PCB depth. In the absence of better data that more accurately establish DoC elevations with certainty, the experience in Phase 1 demonstrates that the Residuals EPS cannot be met during Phase 2.

Finding Rdl.1-2.3: Excessive complexity makes it unlikely that the Residuals EPS can be met.
The Residuals EPS is overly complex, as reported by the 2004 EPS Peer Review Panel. The EPS includes 8 different cases for determining how to address residuals; only 4 of the cases were actually employed during Phase 1. Determining which of the 8 cases applies to a particular CU entails analysis of the following metrics for each CU or portion of a CU being evaluated:

- Average Tri+PCBs concentration
- Individual sample concentrations
- Median Tri+PCBs concentration
- Area weighted average Tri+PCBs concentration in a moving 20-acre area consisting of the CU under evaluation and the 3 or 4 previously dredged CUs within 2 river miles of the current river unit (measured along the river centerline)

These metrics were compiled into the following tabulation to determine the next step for the CU being evaluated:

Certification Unit Arithmetic Average (mg/kg Tri+PCBs)	No. of Sample Results >15 mg/kg Tri+PCBs AND < 27 mg/kg Tri+PCBs	No. of Sample Results > 27 mg/kg Tri+PCBs	No. of Redredging Attempts Conducted

The 20-acre CU averaging was never implemented in part because the unexpected inventory made it difficult or impossible to analyze generated residuals, and in part because the timing of CU closures with neighboring CUs made 20-acre averaging not practicable. Thus, considering upstream CUs became irrelevant to closing individual CUs.

Depending on confirmation sample results, the Residuals EPS requires redredging all or part of a CU, backfilling, or capping. EPA asserts that 2 redredge passes is sufficient to remove inventory and GE agrees that most of the inventory was removed in 2 dredge passes. Based on EPA and GE's observations, better coring results will provide more confidence in the DoC output of the Terrain Model, and thus will contribute to improved residuals management by reducing the number of passes required.

Finding Rdl.1-2.4: Reliance on individual sample results makes it unlikely that the EPS can be met.
The current Residuals EPS relies on the results of individual samples, in addition to CU averages, to determine the need for redredging, backfilling, or capping. Redredging or capping is required if individual sample Tri+PCB concentrations are greater than 15 ppm or 27 ppm.

Table 8. Residual PCB (Tri+ppm) sampling results for 6-inch confirmation sampling after dredging

CU	Number of Sampling Nodes	Number of Residual Samples with High PCB Concentrations after Dredging Passes*					
		Tri+PCBs Concentration of 15-27 ppm			Tri+PCBs Concentration >27 ppm		
		After 1st Pass	After 2nd Pass	After 3rd Pass	After 1st Pass	After 2nd Pass	After 3rd Pass
1	43	0	-	4	0	-	1
2	40	7	6	5	12	11	9
3	47	3	2	0	10	5	0
4	42	2	5	3	17	7	1
5	28	5	3	3	2	0	0
6	40	3	1	0	1	2	2
7	41	3	4	1	15	8	2
8	52	4	3	3	6	3	5
17	40	3	0	0	8	1	0
18	47	5	0	0	8	1	0
Total	420	35	24	19	79	38	20

* Compiled from EPA's Phase 1 Evaluation Report (March 2010) figures in Appendix IIB Post Dredging Core Maps from Different Dredging Passes.

After 3 dredging passes, 6 out of the 10 CUs contained at least 1 node above 27 ppm Tri+PCBs, and 5 out of the 10 CUs contained more than 1 node between 15 and 27 ppm Tri+PCBs, precluding backfilling or capping. Redredging provided limited and apparently diminishing returns. In 3 of 10 CUs, more than 40 percent of the area contained Tri+PCBs concentrations above 15 ppm after the first pass (based on an average 33 core samples); in 3 of the 10, more than a quarter of the area contained Tri+PCBs concentrations above 15 ppm after the second dredging pass. About 10 percent of the dredged areas contained Tri+PCBs concentrations above 15 ppm after the third dredging pass.

From these data the Panel concluded that:

1. The poorly delimited DoC contributed significantly to the increased number of dredging passes required ***and*** the length of time CUs were left open.
2. The increased number of dredging passes and erosion for exposed contaminated sediments significantly contributed to the downstream PCB loads (i.e., exceedances of the Resuspension EPS).
3. An accurate DoC based on better coring would lead to quicker and more efficient removal of PCB inventory.
4. Backfilling or capping the relatively minor residual mass left in place was the norm for Phase 1, and this norm should be carried forward into Phase 2.

CHARGE QUESTION 2. If not, and if EPA and/or GE has proposed modified Engineering Performance Standards, does the experience in Phase 1 and any other evidence before the panel show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2?

Finding Rdl.2: The experience in Phase 1 and other evidence before the Panel does not show that it will be practicable to consistently and simultaneously meet the Residuals EPS proposed for Phase 2 by EPA and GE.

Both EPA and GE provided tables of their respective proposed changes for Phase 2 to the Residuals EPS at the May 2010 Peer Review meeting in Glens Falls, New York. While some of these changes have merit, as a group they do not result in a consistently achievable EPS that meets the requirements of the ROD in concert with the Resuspension EPS and Productivity EPS. Specifically, the Panel did not find the proposed changes addressed the critical needs for a well-defined DoC with appropriate elevation controls, and a means to decrease the number of dredging passes and quickly close the CUs (Table 9 and Table 10).

The Panel noted that EPA's proposed changes do consider the need to reduce dredging passes to both shorten the time that CUs are open and subject to erosional forces, and also to increase productivity (Table 3). However, the proposed changes seem designed to formalize modifications implemented as part of Phase 1, and practicably do little to change the cycle of dredge-test-dredge-test. For example, EPA's proposal to reduce the number of response categories from 8 to 4 in effect carries over the Phase 1 decision process to Phase 2. This will result in a continuation of the dredge-test cycle, and continue to leave CUs open for many months. To meet the Productivity EPS, the duration that the CUs are open needs to be reduced by at least 70 percent (from 113 days to 35 days as presented in Finding Rdl. 1-2); EPA's proposed changes are likely to reduce the duration by the time for the last redredging pass. Typically, sampling, analysis and dredging of the last redredging pass took about 3 weeks, yielding a reduction in the duration of about 20 percent (21 days out of 113 days). To meet the Resuspension EPS as well as the Productivity EPS, dredging would need to be reduced to 1 or 2 passes; however, EPA's proposed changes would reduce removal to a minimum of 2 passes and a maximum of 4 passes, with 3 to 4 passes most likely.

Navigation channels present a special case for consideration in Phase 2, and the Panel agrees with EPA's proposed change that would avoid capping in navigation channels to the degree practicable. If/where capping occurs within the navigational channel, a minimum of 14 feet of draft must be maintained. The Panel recommends that EPA and GE work with the New York State Canal Corporation to establish an operational elevation that would consistently maintain 14 feet of vessel draft.

The Panel found that GE made some practical recommendations to the Residuals EPS, including cessation of dredging upon contact with either hard (rock) substrate or GLAC, and requiring capping only when the residual surface sediment Tri+PCB concentration is greater than 3 mg/kg. The Panel also supports resampling within low-confidence areas, but finds that GE's proposal does not go far enough to solve the overall problem of identifying the DoC. As discussed in response to Charge Question 3, 100 percent resampling in low-confidence areas is required, and confirmation sampling is needed even in high-confidence areas.

Table 9. Summary of EPA's proposed modifications to the Residuals EPS

EPA-Proposed Change to Residuals EPS			Panel Finding	
Proposed Change to EPS	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
Reduce the number of cases from 8 to 4 primary response categories.	The 4 maintained cases are: 1. The standard is met or almost met 2. Residuals are present 3. Inventory is present 4. Recalcitrant residuals or inventory is present	The intention is to simplify and streamline the standard based on Phase 1 results. Four of the cases included in the Residuals Standard were not encountered during Phase 1 and are not likely to be encountered during Phase 2.	No	While the reduction in cases simplifies the decision flow chart, it does not improve productivity or resuspension. The proposed criteria would still result in multiple redredging and resampling cycles instead of closing CUs quickly.
Remove the 20-acre averaging option and backfill testing requirement.	N/A	The conditions where the 20-acre averaging could be applied did not occur during Phase 1 and are unlikely to occur in Phase 2.	Yes	Panel agrees that this was not applied in Phase 1, and would not be applicable to Phase 2.
Eliminate use of the 99% UCL (6 mg/kg criterion) as a basis to decide CU sampling requirements.	N/A	Rather than use 6 mg/kg criterion to trigger sampling at depth, full penetration and analysis of all 6" core segments in a minimum 24" core (unless bedrock or dense clay is encountered) will be required for all post-dredging cores due to Phase 1 experiences with missed inventory and underestimated DoC.	No	The proposed change as worded by EPA implies that the cycle of dredging followed by testing and then more dredging would continue. This pattern negatively impacted the Resuspension EPS and Productivity EPS, and must be changed. The Panel agrees that additional sampling must occur, but it must occur prior to any additional dredging in Phase 2.
Permit capping without formal petition to EPA only after completion of the first pass and at least 1 additional dredging pass targeting only the top 6" of material. In other words, in order for capping to be permitted, the inventory must have been removed as confirmed by post-	No numerical criteria are changed for this revision. This applies only to Case 4 – Recalcitrant residuals or inventory present.	The Residuals EPS contemplated limited capping as a contingency to address residuals in the presence of difficult bottom conditions. The option for capping is not meant to compensate for any deficiency in dredging design. However, during Phase 1, capping was sometimes employed primarily to isolate inventory and this should be avoided in Phase 2.	No	Productivity could be improved by eliminating the second redredging pass and streamlining the decision process. However, without accurate DoC data prior to dredging, the benefits would be minimal as multiple inventory passes might still be required.

EPA-Proposed Change to Residuals EPS			Panel Finding	
Proposed Change to EPS	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
dredging coring and an additional pass targeting just 6" (residuals) must have been performed.				
Confirm DoC in post-dredging cores.	Two contiguous segments less than 1.0 mg/kg Total PCBs are required to confirm that DoC is known.	During Phase 1, there were situations where sediment cores were observed to reach a value of less than 1.0 mg/kg in a single 0 - 6" segment only to see concentrations rise again deeper in the profile.	No	The proposed change does not provide for reduction of dredging passes or quick closure of CUs, both of which directly affect productivity and resuspension. Unless DoC is determined prior to dredging, the dredging plan will remain suboptimal, entailing extra dredge cuts, passes, surveys, sampling, and testing. In addition, depth of coring must be limited when bedrock or GLAC is encountered.
Simplify identification of noncompliant nodes for reviewing dredging pass results.	Target average value of 1.0 mg/kg Tri+PCB, using only the ranked, measured nodal values in a simple accumulating average.	As implemented in Phase 1, locations that appeared to be compliant with the standard on 1 pass caused the mean to exceed the Residuals EPS threshold after later passes, requiring redredging (or capping) in the previously compliant location. This problem is eliminated by this simplified process.	No	This change, which specifically calls for a second—laterally more extensive—dredging pass to increase captured inventory, addresses a symptom (in Phase 1, locations that appeared to be compliant with the EPS on 1 pass caused the mean to exceed the EPS after later passes, which resulted in redredging or capping), but fails to account for the mechanisms that render formerly compliant nodes noncompliant. Unnecessary dredging is likely to result, reducing productivity and increasing resuspension.
Simplify identification of redredging or capping boundaries.	The area associated with noncompliant nodes extends to the periphery of compliant nodes or to the edge of the CU. Where a compliant node is surrounded by noncompliant nodes, the area associated with the compliant node is dredged to the average depth of the surrounding noncompliant nodes. Generally, 3 compliant nodes are required to define an area that does not require redredging.	In Phase 1, a sophisticated algorithm was a source of much discussion and often resulted in unusual dredging geometries. A more conservative approach is needed in light of poor spatial correlation and DoC uncertainty.	No	The proposed simplified geometry would reduce the potential for compliant nodes to become noncompliant by using a common-sense approach that recognizes the lack of precision in dredging and DoC. However, without a broader framework, built on averaging rather than a patchwork approach, benefits would be minimal.

EPA-Proposed Change to Residuals EPS			Panel Finding	
Proposed Change to EPS	**Proposed Numerical Criteria**	**Rationale**	**Accept Proposed Change?**	**Rationale**
Identify nodes with high probability of exceeding the Residuals EPS threshold early in the CU dredging process to mitigate uncertainty in DoC estimation.	Target concentration of 1.0 mg/kg Tri+PCB, permitting only a mean of 1.49 after the last pass.	As implemented in Phase 1, locations that appeared to be compliant with the Residuals EPS on 1 pass later caused the mean to exceed the EPS threshold after later passes, requiring redredging (or capping) in the previously compliant location. Areas identified in this manner will meet the true threshold of 1 mg/kg, regardless of the outcome of subsequent redredging attempts at the noncompliant locations.	No	This approach would set a target concentration of 1.0 mg/kg Tri+PCB, permitting only a mean of 1.49 after the last pass. The goal is to reduce conversions from compliance to noncompliance after subsequent passes. This approach shows some of the flexibility required for variability in site conditions and dredging performance, but the specified concentration is impracticably low for the PCB concentrations observed in the sediment profile and realistically achievable dredging residuals.
Avoid capping in the navigation channel whenever possible. If it is necessary, however, design and implement such that the top of cap allows for a minimum of 14 feet of draft to allow for future maintenance dredging by the NYS Canal Corporation.	Caps must allow 14 feet of draft in navigation channels.	Capping was not expected in the navigation channel. However, during Phase 1 the installation of a subaqueous cap was required in and around Rogers Island. The caps in the navigation channel were placed such that the navigation depth of 12 feet was met. The 12-foot depth, however, does not account for the need to conduct maintenance dredging of sediments that become naturally deposited on top of the cap. The tops of any caps placed in the navigation channel in Phase 2 must be at least 14 feet deep in order for NYSCC to maintain adequate channel depths.	Yes	This proposed change will reduce potential adverse impacts of prop wash on cap stability while accommodating maintenance dredging.
Eliminate the concepts of ‘inventory pass’ and ‘residuals pass’ from the Residuals Standard. Consider all passes simply as dredging passes.	N/A	Rarely in Phase 1 was subsequent dredging after the first pass exclusively done to remove inventory or residuals. The categorization of particular dredging passes, which has no impact on implementation of the Residuals EPS, became a distraction during project discussions.	Yes	This change may reduce confusion and streamline decision making. However, the categorization of individual dredging passes is not expected to have any impact on productivity or resuspension.

Table 10. Summary of GE's proposed modifications to the Residuals EPS

GE-Proposed Change to Residuals EPS		Panel Finding	
Proposed Change to EPS	**Rationale**	**Accept Proposed Change?**	**Rationale**
In high-confidence areas, dredge to the design prism and sample to determine the appropriate cap or backfill.	In Phase 1, the design dredge prisms in high-confidence areas removed close to 90% of the inventory in these areas. Each subsequent pass removed only a few percent of the mass, but impacted productivity and prevented CU closure.	No	The Panel agrees in principle with the proposed change, but finds that without accurate DoC—including elevation controls—prior to dredging, the approach is inadequate to meet the Productivity EPS and Resuspension EPS. Thus, this approach must be accompanied by more accurate DoC delineation. In Phase 1, design dredge prisms (i.e., first dredge pass) in high-confidence areas removed 80 to 85% of inventory. According to GE, subsequent passes removed only a few percent of PCB mass while reducing productivity and preventing CU closure. Based on the overall PCB removal reported, subsequent dredging passes appear to have removed possibly 20% or more of the PCB mass originally in some high-confidence areas being redredged. This large additional removal in some high-confidence areas results from inadequate elevation controls (the DoC appeared to be off by about 4" in high-confidence areas) in the original sediment coring to determine the DoC and from the 3" tolerance allowed above the dredge prism that was set roughly at the DoC.
Collect data in low-confidence areas—redefine DoC to convert to high-confidence areas. Then, dredge to the design prism and sample to determine the appropriate cap or backfill.	See Above	Yes	Additional core data should be collected in all low-confidence areas, but also in high-confidence areas. Inadequate penetration of the cores and lack of elevation controls for all cores was directly responsible for improper DoC characterization.
When hard bottom is encountered above a dredge prism elevation, do not dredge further in that location, but install the appropriate cap or backfill.	Dredging on bedrock is illogical and difficult to implement.	Yes	Dredging on bedrock is impracticable. No dredging of bedrock or rock outcroppings should be attempted.

GE-Proposed Change to Residuals EPS		Panel Finding	
Proposed Change to EPS	**Rationale**	**Accept Proposed Change?**	**Rationale**
When glacial clay is encountered above a dredge prism elevation, do not dredge further in that location, but install the appropriate cap or backfill.	Dredging GLAC is illogical because it is not contaminated with PCBs and slows productivity by impacting the processing facility.	Yes	GLAC is not contaminated with PCBs and dredging this clay slows productivity. No dredging of GLAC should be attempted.
Modify the existing dredge removal tolerances to allow a certain percentage of the 10 x 10 ft. compliance grid cells to be above the existing tolerance on an acre basis following the dredge pass to minimize the amount of unproductive time spent removing small quantities of sediment above the dredge cutline tolerance limit.	Achieving the Phase 1 removal tolerance on a 10 x 10 ft. grid was very time-consuming, and ultimately residual PCB concentrations determined the next step in any event.	No	This proposal is not compatible with GE's first 2 proposed changes, above. If the Phase 2 Residuals EPS retains multiple dredge passes, then this change would improve productivity somewhat.
Capping should not be required unless the residual surface sediment Tri+PCB concentration is greater than 3 mg/kg.	This would allow the simple application of backfill to residual concentrations that pose no significant threat to the recovery of the river. The existing Residuals EPS allows backfilling in areas containing up to 3 mg/kg in certain circumstances. Experience in the Grasse River indicates that 1 foot of backfill achieves about 95% reduction in surface sediment PCB concentrations (Connolly et al. 2007). This reduction would achieve a Tri+PCB concentration of 0.15 mg/kg when applied to 3 mg/kg sediments. The proposed criterion for capping is similar to the criterion adopted for the Fox River (EPA 2007), which allows a 6" sand cover over Total PCB concentrations as high as 10 mg/kg.	Yes	Surrounding areas not designated for dredging have surficial Tri+PCB concentrations greater than 1 mg/kg (typically 3 to 6 mg/kg). The change would improve productivity and avoid resuspension, achieving significant risk reduction via isolation.
The dredging completion form (Form 1) and the backfill and capping form (Form 2) should be combined into a single review and approval step.	EPA would oversee verification of dredged elevations, determination of residual core sampling locations, residual core sample collection and analysis, redelineation of any redredge surfaces, development of backfill or cap surfaces, and verification of placed backfill or cap surface elevations; no formal approval would be required before proceeding to the next step in the process.	Yes	This would speed the re-dredging and CU-closure process, increasing the area that can be remediated in a season. A single review and approval step is particularly appropriate if the proposed single dredge pass changes are adopted; redredge surfaces will not be required in that case.

CHARGE QUESTION 3. If the experience in Phase 1 and other evidence before the panel does not show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2, can the Phase 1 Engineering Performance Standards be modified so that they could consistently be met in Phase 2, and, if so, how?

Phase 1 demonstrated that the Residuals EPS had a substantial impact on project success and on the interaction with the Resuspension EPS and the Productivity EPS. Incomplete DoC characterization combined with adherence to the 2004 EPS residual target levels directly affected both the Resuspension and Productivity EPS. Repeated dredge passes and prolonged exposure of sediments in the CUs resulted in increased PCB resuspension and release. The unexpected increase in inventory due to incomplete DoC characterization had the greatest effect on the Productivity EPS in terms of numbers of CUs remediated. The Panel proposes revising the Residuals EPS to accelerate CU closure by establishing an elevation-focused dredge design paradigm, thereby reducing resuspension, effectively managing residuals, and accelerating productivity without sacrificing goals of the ROD with respect to overall recovery of the river.

Attempts to meet the Residuals EPS led to the need for repeated dredging and cleanup passes, surveys, sampling, and chemical analyses that delayed closure and reduced overall productivity by as much as 30 percent. Table 11 shows that 50 percent of the dredge time was spent redredging, sometimes in response to individual sample values. Despite repeated dredging and cleanup passes to achieve a residuals concentration of less than 1 mg/kg Tri+PCBs, it was achieved throughout the entire area in only 1 of the ten CUs dredged in Phase 1.

Table 11. Days spent dredging per CU (composed from March 2010 GE Table 6.4-1)

CU	1st Pass (d)	2nd Pass (d)	3rd Pass (d)	4th Pass (d)	5th Pass (d)
1	37	19	28	16	9
2	38	17	9	4	-
3	28	20	12	-	-
4	30	14	2	-	-
5	31	17	8	-	-
6	31	19	6	-	-
7	26	16	16	2	-
8	27	10	22	5	-
17	22	14	5	-	-
18	38	16	1	-	-
Total	308	162	109	27	9

Risk management should be strongly factored into determining how much redredging to undertake. When dredged areas are covered with backfill or an engineered cap (standard practice for this project), residuals become isolated, making them unavailable to biota. Excessive redredging reduces productivity, increases project costs and time, and poses increased environmental risks by increasing the time that contaminated sediment surfaces are exposed to the environment and by increasing resuspension potential during periods of exposure. The Productivity EPS measured project progress on a cubic-yard-dredged basis, whereas progress is more accurately reflected by the size of the area remediated and contained. Thus, focus on achieving target Tri+PCBs concentrations in accordance with the Residuals EPS hindered productivity on an areal basis. This is especially true under conditions where a) risk reduction is affected primarily by the area remediated, as opposed to the volume or mass remediated, and b) the total volume targeted for removal in each area is uncertain—both conditions are true of this project.

Finding Rdl.3: The Phase 1 Residuals EPS can be modified to be consistently met in Phase 2; however, additional steps are necessary to simultaneously meet the Productivity EPS and Resuspension EPS, achieve risk reduction goals, and accomplish the requirements of the ROD.

The Productivity EPS and Resuspension EPS must be integrated with the Residuals EPS so that all 3 standards are achievable. This approach requires an understanding of the limitations of dredging productivity, residuals management, and resuspension/release potential. Using improved DoC elevations, EPA and GE should establish fixed dredge elevations and revise the predicted Phase 2 dredge volume accordingly. This information should be used to establish a realistic productivity goal and dredging timeline, relying on predetermined dredge elevations, rapid CU closure, and more liberal use of backfilling or capping, as appropriate. This approach must also rely on an expanded and formalized adaptive management process to facilitate routine operational modifications based on experience.

Finding Rdl.3-1: The project should focus on single-pass sediment removal (i.e., efficient dredging of DoC output with an acceptable confidence), quickly dealing with residuals through backfilling or capping.

The value of redredging beyond the DoC is questionable, since all dredged areas will ultimately be backfilled or capped. This is especially true for multiple redredging passes. According to GE, greater than 90 percent of the PCB inventory was removed in the first 2 dredge passes, and only approximately 7 percent more inventory was removed via subsequent redredge passes (in general, during dredging of what would be deemed residuals instead of inventory).

According to EPA, except for CU1, 98 percent of the Phase 1 inventory was removed from completed CUs. However, extreme measures were taken to achieve this level of inventory removal; the time and effort dedicated to dredging residuals would have been spent more effectively on activities that would have improved overall productivity, accelerated CU closure, and reduced resuspension and dredge-generated residuals to a considerably greater extent.

Significant changes in approach will be required to attain the desired rate of closure (1 CU per 8-10 day period). For example, compositing confirmation samples would manage the occurrence of outliers that pose limited risk to biota. Compositing also obviates the need for trigger concentrations of 15 and 27 ppm Tri+PCBs, further simplifying the CU closure process.

It should be noted that the ROD, which calls for removal of all inventory in the defined footprint (based on mass-per-unit-area analysis) operationally defined as above 1 ppm Total PCBs, was written prior to

issuance of specific technical guidance by EPA that clearly acknowledges the viability of capping for managing risks (*Contaminant Sediment Remediation Guidance for Hazardous Waste Sites* (EPA 540-R-05-012)). Further, it is common knowledge among dredging practitioners and environmental scientists and engineers that dredging activity always leaves behind some residual material. Though the Phase 1 Residuals EPS allow for capping, the decision process to use capping requires extensive and repeated dredging to demonstrate that dredging alone cannot achieve target residuals levels, violating the spirit of the dredged residuals standard which attempted to limit the number of dredging passes. More efficient and extensive use of capping would improve productivity and reduce resuspension while achieving risk reduction goals. Furthermore, the fact that EPA and GE employed caps fairly extensively during Phase 1, including for areas where PCB concentrations above the Residuals EPS were left in place for near shore areas with steep slopes, establishes precedence and indicates acceptance for the use of capping to manage areas with elevated PCB levels.

Finding Rdl.3-2: Perform investigations to define DoC, confirm DoC, and drive dredging plans and residuals management.

The project's failure to meet the Residuals EPS in Phase 1 can be directly attributed to poor DoC modeling, which was itself due to poor cores. Only about 40 percent of Phase 1 cores characterized DoC with high confidence. In Phase 2 only about 60 percent of cores characterize the DoC with high confidence. In addition, the DoC determined from the coring lacks adequate vertical positioning controls to tie the DoC to a datum for accuracy, even when a reasonable level of precision was achieved.

DoC must be accurately and precisely defined prior to designing dredge cuts to avoid repeated dredging passes and inventory recharacterization, which can adversely impact the river's long-term recovery and impose unacceptable environmental and human health risks. Accurate DoC provides confidence that residual PCB concentrations are generally derived from generated residuals and are much lower than those in the volume targeted to be dredged.

The following steps should be taken to establish an accurate and useful DoC that can drive dredging plans and residuals management.

- Coring Program. Perform recoring of all low-confidence samples. Samples now designated as high-confidence should be verified as high-confidence. All sampling must be performed to attain at least 80 percent recoveries of soft sediments and must be cored either to bedrock or GLAC. Sediment layers must be reported as elevations rather than as depth below mudline, using state-of-the-art positioning for horizontal and vertical control. All cores should be analyzed until 2 6-inch layers have Total PCBs below 1 ppm.
- DoC Elevation. Remodel the DoC using all high-confidence cores to establish the topography (terrain model) of the DoC throughout each CU, referred to as the DoC Elevation. Consideration should be given in the modeling to precision/uncertainty of the DoC measurements in order to ensure that the inventory is captured in the dredge prism. The uncertainty of the DoC is a matter of concern when single pass dredging is being considered, especially in light of reported paired cores having an averaged difference in DoC 11.2 inches in 67 paired high-confidence cores and a median differences of 9 to 12 inches (EPA March 2010, Chapter II, Section 2.5).
- Design Dredge Elevation. Set the Design Dredge Elevation initially to 4 inches below the modeled DoC to account for the vertical accuracy and precision of the dredge, referred to as dredge

tolerance. The goal for dredging is to achieve the DoC elevation in 95 percent or more of the dredged area after a single dredge pass (i.e., at least 95 percent of the area dredged in the 1-acre subunit should be at or below the modeled DoC elevation). Incorporating a factor for dredge tolerance ensures that the dredger attains the modeled DoC Elevations as quickly as practicable (i.e., in a single pass).

- Post-Dredge Elevation. Confirm that the DoC Elevations have been met after dredging, allowing closure of the CU, or subunit. Adaptive management should be used to update the dredge tolerance. If the dredger demonstrates that the DoC is consistently achieved with a single pass (i.e., at least 95 percent of the dredged area at or below the DoC Elevation), then the magnitude of the dredge tolerance included in the Design Dredge Elevation can be reduced for subsequent areas. If the dredger has trouble consistently capturing the DoC in 95 percent or more of the dredged area after a single pass, then the magnitude of the dredge tolerance in the Design Dredge Elevation should be increased for subsequent areas.
- Confirmation Sampling. Collect and composite 6-inch residuals samples as soon as possible after EPA confirms dredging is complete in a CU, or subunit, based solely on the elevation measurements. Recommendations for this sampling are given in Table 12.
- Sand Cover. Place a 3-to-6-inch sand cover over the CU subunit as soon as possible after residuals samples are collected (PCB analytical results are not required for this step). No verification of placement thickness is required at this time.
- Backfill or Cap. Use PCB analytical results for the residuals composite sample to determine whether an area should be backfilled or capped. Then install appropriate final layers on top of the sand cover for closing the subunit after dredging of the CU and all upstream CUs are completed. Perform appropriate confirmation monitoring to verify backfill or cap placement in accordance with design specifications. Do not redredge to capture residuals.

Finding Rdl.3-3: Prior to dredging a CU, update the Design Dredge Elevations and remove inventory with a single dredging pass.

The Phase 1 dredging program required multiple unplanned redredge efforts to remove unanticipated inventory. This resulted in the CUs being opened for extended periods. In open CUs, PCB-contaminated sediment was exposed to ongoing disturbance from river flow and vessel traffic, which continued to erode and transport contaminated sediment down river. CUs should be closed more quickly during Phase 2 to reduce the magnitude of PCB release prior to closure and to simultaneously meet all the engineering performance standards. The only way to reduce the number of passes while satisfying the goals of the Residuals EPS is to more precisely establish Design Dredge Elevations prior to dredging. The dredge prism should be updated as follows.

- Establish DoC using high-confidence cores throughout each CU, and generate an updated high-confidence DoC Terrain Model to establish the topography of the DoC throughout the CU (DoC Elevation) such that the DoC topography contains all of the inventory with acceptable certainty, considering the variability of the DoC in paired high-confidence cores.
- The Design Dredge Elevation should be established based on the updated DoC Terrain Model, limitations of the dredge to cut a slope, river hydrodynamic conditions, and a realistic estimate of

residuals generation and management that are based on an understanding of exposure risks associated with surface sediment PCB deposits.

- The Design Dredge Elevation should initially be set at 4 inches below the DoC Elevation to compensate for tolerances in vertical positioning of the dredge bucket.
- Use an adaptive management approach to adjust the Design Dredge Elevation according to actual dredge performance, integrating knowledge of dredge productivity, CU closures, and resuspension.
- Set the Contractor's Dredge Prism to capture the full extent of the Design Dredge Elevation.

Finding Rdl.3-4: Use an adaptive management approach to adopt dredging BMPs to manage residuals.

Dredging activity disturbs sediment and increases short-term environmental exposures to buried contaminants, resulting in the resuspension of PCBs in the water column and the formation of loose, PCB-containing residuals on the bed surface, both within and outside of dredged areas. Changing the manner in which the dredge removes the material from the river can reduce the amount of resuspension and residuals that are generated. An adaptive management approach should be used to incrementally implement the following dredging BMPs, monitor benefits, and adopt, modify, or eliminate BMPs and performance standards based on monitoring results.

Single-Pass Dredging Program

Preparing the Contractor Dredge Prism based on an updated high-confidence DoC Terrain Model and a Design Dredge Elevation will allow for single-pass dredging (including an allowance to compensate for the vertical tolerance in dredge bucket positioning) with a high degree of confidence that inventory is being removed effectively. With a well-defined dredge prism, dredging can be completed in a single event, accelerating CU closure and minimizing exposed PCBs. Monitoring the post-dredging bed elevation with high-precision bathymetric surveys provides an adequate basis to confirm that the targeted material is removed, and provides feedback to adjust (i.e., to adaptively manage) the Contractor Dredge Prism (i.e., to adjust vertical dredge tolerance requirements) to assure removal in a single pass.

Incorporation of a vertical dredge tolerance in the Design Dredge Elevation is prescribed to assure that the DoC Elevation is achieved in a single pass. The dredge tolerance factor is intended to balance the goals of attaining required elevations in a single pass and limiting the dredging of non-target material.

Each certification subunit should be dredged until completion, and dredging should proceed from upstream to downstream to the extent practicable. As soon as practicable and following completion of each subunit, a bathymetric survey should be conducted to confirm that the sediments were removed in accordance with the criteria established in the revised Residuals EPS (e.g., the elevation of 95 percent or more of the dredged area should be at or below the established DoC Elevation).

Stop Dredging at Rock and Clay

The contractor should stop dredging whenever till is encountered, whether GLAC, bedrock, or other hard bottom/rock. Continuing to dredge into till material provides no environmental benefit, while increasing the downstream release of PCBs by keeping the CU open, and unnecessarily expending energy and time.

Stair Step Cuts

The Phase 1 dredge plan allowed for a vertical cut face for the full depth of the dredge prism. Vertical cuts can result in bank sloughing, which can release contaminants to the water column (resuspension) and increase dredge-generated residuals.

Stair-stepping the cut involves offsetting the bucket placement through the depth of the cut to produce a more stable, sloping cut face that is less likely to slough or fail. This approach will reduce bank failure and associated residuals and resuspension release.

Sequence Dredging Bank to Bank and from Upstream to Downstream

The Phase 1 report shows cases where a single dredge lane was advanced downstream as far as 600 to 800 feet in the direction of flow. This longitudinal approach creates a thalweg effect that can increase local river flow velocities, and can contribute to resuspension and release by eroding the cut's side slopes and bed. Vessels passing through such a cut also have the potential to accelerate resuspension via slipstream and prop wash velocities. Erosion and sloughing in the cut area increase resuspension of PCBs into the water column and downriver. This condition is exacerbated during higher river flow conditions. Modifying the dredging sequence and monitoring the effects will optimize productivity while reducing generated residuals and resuspension by erosion of residuals within the cut area, and encouraging deposition within the CU instead of downstream of the CU.

Instead of dredging long downstream longitudinal lanes, dredging should be short cross-stream lanes dredged from bank-to-bank, then upstream to downstream. Dredging should target 1-acre CU subunits (or another appropriately sized subunit) that are designed to limit creation of a thalweg-like channel. The subunit should be dredged from bank to bank or in predefined areas not necessarily aligned with the direction of water flow. To accelerate closure, and to the extent reasonably practicable, each dredged subunit should be surveyed, sampled, and covered while dredging on adjacent or downstream subunits proceeds.

Final backfilling or capping should occur strictly from upstream to downstream.

The Panel recognizes that dredging will have to occur simultaneously at multiple locations along the river, making strict adherence to an upstream to downstream requirement impossible. For this reason, the BMP may involve dredging in multiple CUs located upstream and downstream of each other, as long as the final backfilling or capping is completed sequentially from upstream to downstream. Because Phase 1 indicates that residuals likely contribute to resuspension and release from upstream to downstream, the immediate placement of a 3-6-inch coarse sand layer will control near-term releases, buying time within a single dredge season to complete the final backfill and capping from upstream to downstream. All backfilling and capping must be completed before dredging terminates at the end of each year.

Initial Sand Cover Immediately After Dredging

Dredging generates a layer of residual sediment with a higher water content and lower shear strength than the native deposit, commonly referred to as dredge-generated residuals. This residual layer is more easily eroded than the native, undisturbed sediment bed, and consequently results in more erosion and resuspension of PCBs than the predredge condition. Leaving the disturbed residual sediment exposed in the river for long periods increases resuspension to the water column.

An Initial Sand Cover consisting of a thin layer (3 to 6 inches) of coarse sand should be placed as soon as possible after dredging of a CU subunit is complete, and following EPA verification of the dredged elevation. The Initial Sand Cover will provide a relatively clean, less erodible surface within the footprint of the dredge cut, limiting the resuspension and release of contaminated residuals and limiting short-term surface sediment exposures during construction. The Initial Sand Cover will also act as the first lift of a sand backfill or cap layer. The thickness of this cover does not require confirmation after placement, beyond the verification that an appropriate volume of sand was placed to achieve the target fill amount; in other words, placement can be controlled by the volume or weight of cover materials delivered to each defined area combined with global positioning system (GPS) information provided by the placement contractor. This cover is critical to controlling resuspension and is applied most effectively as soon as possible to contain the residuals. The cover can be placed before the data from confirmation sampling are collected.

The entire CU can be closed after dredging of all of CU subunits is completed and all upstream dredging is complete. The method of closure, backfilling or capping, is based on analytical results of composite 6-inch residuals cores as described in Table 12.

Table 12. Summary of recommended changes to the Residuals EPS

CHANGE TO RESIDUALS EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER EPS
1. DEPTH OF CONTAMINATION (DoC) a. Collect additional cores in high- and low-confidence areas, as well as areas of missing data, to more accurately define the elevation of the DoC for all Phase 2 CUs with a high degree of confidence. b. Use coring methods and equipment capable of penetrating debris and reaching the rock or clay substrate with good to excellent core recovery (i.e., >80%). The equipment used in the SSAP does not meet this criterion. c. Collect, log, and process intact cores. d. Generate a high-confidence DoC Terrain Model for each CU based on the new coring data.	Cores will be characterized in 6" intervals for TPCB and Tri+PCB concentrations. The DoC Terrain Model will establish a DoC at the level where Total PCBs are < 1 ppm. The data also will be used to establish surface sediment and till elevations at the time of collection. The project also will benefit by the collection of some high-confidence cores to validate the current understanding of DoC elevations in high-confidence areas. At a minimum, collect: ♦ Low Confidence Cores: Repeat 100% of these cores ♦ Missing Data: 100% collection in areas lacking data ♦ High Confidence Cores (recommended): Repeat 20% of high confidence cores to validate elevation DoC elevations. If new cores do not adequately validate the DoC, resample high confidence cores as necessary to establish high-confidence DoCs for input into the Terrain Model. ♦ Vertical Positioning Controls. When coring, measure surface sediment and till elevation to 0.1 ft; include real time water	The Phase 1 closure process for Residuals negatively impacted both the Resuspension and Productivity EPS. Leaving CUs open to scour while going through the validation and redredging process was very likely a significant source of PCBs resuspension and downstream release. Two factors contributed to prolonged open CUs: incomplete DoC determination during the design phase, and preoccupation with sediment volume and PCB mass removals as the primary metrics of success in lieu of a risk-based goal that focuses on remediated areas and CU closure. These two factors contributed to resuspension and release and reduced productivity rates. The focus should be on effective single-pass dredging, rapid CU remediation and closure, improved productivity, and reduced resuspension and release. The intent for resampling is to improve confidence in the DoC and the 1 ppm Tri+PCBs neat line, obviating the need for multiple	Confident characterization of sediments and the DoC in remaining CUs along with single-pass dredging have the greatest potential of any modification to the dredging program to reduce PCB resuspension and release and to increase overall project productivity.

CHANGE TO RESIDUALS EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER EPS
	elevations measurements. Horizontal Positioning Controls. Established x-y coordinates using GPS system capable of sub-foot accuracies.	redredging passes and providing confidence in removing the target inventory. The recoring program is a monumental task and may not be accomplished in a single construction season. The revised and improved DoC delineation should be staged to meet the needs of each subsequent dredging season (i.e., next year's CUs).	
2. DESIGN DREDGE ELEVATIOIN a. Prior to dredging a CU, update the Design Dredge Elevation using high-confidence cores and the updated DoC Terrain Model, combined with an understanding of hydrodynamic conditions and risk reduction goals. b. The Design Dredge Elevation should initially be set to below the level where Total PCBs are < 1 ppm to accommodate the vertical dredge positioning tolerance. c. Adjustments to the Design Dredge Elevation at CUs or subunits could be considered if the following can be demonstrated: a) adequate inventory removal, b) the ability to design and construct a cap that will meet predefined	The Design Dredge Elevation should initially be set to 4" below the DoC Terrain Model to compensate for tolerances in vertical positioning of the dredge bucket. If more than 95% of the dredged area is consistently below the DoC Terrain Model Elevations in the bathymetric survey after the design dredging pass, the 4" vertical dredge tolerance may be relaxed through adaptive management. Likewise, if 95% of the area is not consistently at or below the DoC Terrain Model Elevations in the bathymetric survey after the design dredging pass, the vertical dredge tolerance adjustment to the dredge prism should be maintained or increased through adaptive management.	The updated DoC Terrain Model, using reliable DoCs from the new core sampling, will provide the degree of certainty necessary to allow for single-pass dredging (including an allowance to compensate for the vertical tolerance in dredge bucket positioning) with a sufficient degree of confidence that inventory will be removed effectively and efficiently. Multiple passes to remove generated residuals are inefficient, have limited success in achieving the 1 mg/kg Tri+PCB goal, and leave CUs open unnecessarily.	Efficient and effective removal of inventory will speed up closure of CUs, which in turn will reduce resuspension and release and increase productivity.

CHANGE TO RESIDUALS EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER EPS
performance goals regarding chemical exposure and hydrodynamic conditions, and c) advantages in terms of reduced risks, e.g., where removal of deep sediment deposits may incur greater environmental harm via resuspension and release than benefits gained by additional inventory removal.			
3. DREDGE METHODS AND SEQUENCE a. Eliminate the concepts of 'inventory pass' and 'residuals pass' from the Residuals EPS. Consider all passes simply as dredging passes. b. Dredge to the Design Dredge Elevation within a subunit in a single pass. Once the DoC Elevation is achieved, there normally will be no further dredging; rather, dredging will be followed by expeditious confirmation monitoring and placement of an Initial Sand Cover. c. Within a CU, and to the degree reasonably possible, dredge from upstream to downstream, sequentially completing each subunit (typically on the order of an acre each) before moving to the next downstream subunit. d. Within a dredging season, allow	No numerical criteria.	Modifications to the dredge methods and sequencing are intended to reduce the amount of time that each CU remains open and to reduce the loss of PCBs downstream through resuspension. With an accurately defined DoC, the dredging can be completed in a single pass, and the CU can proceed directly to closure. Expeditious placement of an Initial Sand Cover following dredging provides immediate reduction of resuspension losses and improves long-term effectiveness. Dredging CU subunits from upstream to downstream will eliminate the dredging of a narrow channel running the full length of a CU area, which concentrates river flow and likely increases PCB losses	Efficient and effective removal of inventory will speed up closure of CUs, which in turn will reduce resuspension and release and increase productivity.

CHANGE TO RESIDUALS EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER EPS
for dredging of multiple CUs that are located downstream of one another, provided that the final cap or backfill placement proceeds from upstream to downstream, within a single year's dredging, to complete all annual dredging, backfilling, and capping before the year's end. e. Stop dredging wherever GLAC is encountered in the dredge prism. f. Stop dredging wherever rock or hard bottom conditions are encountered in the dredge prism. g. Complete dredge cuts with stair-stepped side walls, rather than vertical side walls, to reduce bank sloughing and associated generation of residuals and resuspension. h. Avoid longitudinal dredging that creates thalweg-like conditions in the presence of exposed PCBs.		downstream caused by scour of disturbed PCB-containing residuals in the dredge face. Allowing dredging of CUs downstream of other active CUs is based on the understanding that limited upstream to downstream recontamination will occur as soon as upstream areas are covered with an initial backfill layer, provided that the final backfilling and capping is completed from upstream to downstream within a single season. Stopping dredging wherever either GLAC or hard bottom/rock is encountered is based on the understanding that no benefit is achieved by attempting to remove such material, while at the same time increasing the downstream release of PCBs by the ongoing dredging. Completing dredging with stair-stepped side walls, rather than dredging multiple bucket depths at the same location, will reduce bank sloughing. Bank sloughing can be a significant source of generated residuals and resuspension/release.	

CHANGE TO RESIDUALS EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER EPS
4. CONFIRMATION MONITORING – DREDGE PRISM a. Once dredging is complete to the Design Dredge Elevation, use bathymetric surveys to confirm elevations. b. Set CU subunits (roughly 1-acre each) as the performance area for completing dredge design prisms. c. Redredging within a CU subunit is only required when less than 95% of its area is at or below the DoC Elevation, and only to the degree necessary to bring at least 95% of the area at or below the DoC Elevation. d. Remove the existing dredge removal tolerances on percentage of the 10 x 10 ft compliance grid cells.	Dredging should be conducted such that at least 95% of the post dredge surface within each CU subunit (approximately 1 acre) is at or below the DoC Elevation. See Item 2, Design Dredge Elevation, for adjustments to the vertical dredge tolerance associated with meeting this criterion. Do not include individual small contiguous areas of less than 3 sq ft each that protrude above the DoC Elevation in the calculation of achieving 95% of the post-dredge surface below the DoC Elevation.	Achieving the Phase 1 removal tolerance on a 10 x 10 ft grid was time consuming, and ultimately residual PCB concentrations determined the next step in any event. Small protrusions above the dredge surface, such as logs and rocks and even small ridges between bucket placements, will be detected by surveying techniques. However, the small isolated areas do not represent significant undredged material, and attempts to capture them with a redredge pass will further increase resuspension releases and delay the timely closure of CU areas.	Timely closing of CUs will reduce resuspension and increase productivity.
5. CONFIRMATION MONITORING – PCBs a. Sample the surface sediment (top 6”) immediately after reaching the Design Dredge Prism. b. Use post-dredge surface sediment chemistry results to determine whether a backfill or a cap is appropriate to complete the remedial action at the CU subunit. c. Use a composite sampling	The Panel recommends an 8-point composite sample of the post-dredge surface sediment (top 6”) for each CU subunit (approximately 1 acre). Submit the composite sample for PCB analyses. (Do not archive original samples for future analyses.) The PCB Confirmation Monitoring analyses can occur after placement of the Initial Sand Cover, provided the PCB monitoring program	Completing 40 discrete cores with multiple vertical subsections for confirmation monitoring within a CU was time-consuming and caused a backlog in the PCB analyses, which contributed to the extended length of time required to close a CU, without sufficient benefit of reduced residuals, reduced exposures, or reduced ecological risks. With	Timely closing CUs will reduce resuspension and increase productivity.

CHANGE TO RESIDUALS EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER EPS
approach for CU subunits. d. Set CU subunits as the performance area for PCB confirmation monitoring to be used to select backfill or capping.	penetrates through the sand layer into 6" of the post-dredging surface sediment. In that case 1 ft cores should be collected and the sand discarded prior to sub-sampling the top 6"of sediment to create the composite. PCB concentrations measured from the 8-point surface sediment composite within a subunit will establish whether backfilling or capping is required as the final action in a subunit as follows: • Backfill if less than or equal to 3 mg/kg Tri+PCBs. • Cap if greater than 3 mg/kg Tri+PCBs.	improved delineation of the DoC, the extensive coring program is no longer necessary. A composite sample provides an average PCB concentration that is more representative of the risk presented by the CU subunit after dredging. A 3 mg/kg Tri+PCBs criterion for capping is more achievable, practicable and representative of surrounding surficial sediments that are not being actively remediated. 3 mg/kg Tri+PCBs would not retard natural recovery of surrounding areas if the backfill were to erode.	
6. BACKFILL AND CAPPING a. As soon as practical after removing the sediments in the Design Dredge Prism, place an Initial Sand Cover to a depth of 3-6" over the surface of the dredged area. b. Select either backfilling or capping for the CU subunit based on the post-dredging PCB concentration in surface sediment. c. Complete backfilling and capping in a dredging season, working from upstream locations to downstream locations.	The Initial Sand Cover will be placed on a volume-per-area basis and not require sampling to verify the thickness of sand placed throughout the CU subunit.	The near-immediate placement of an Initial Sand Cover will reduce resuspension and redeposition outside the dredge prism and will provide risk reduction until a decision is made to cap or add more sand to complete backfilling after dredging of the CU and other upstream CUs is completed.	Timely closing of CUs will reduce resuspension and increase productivity. The Initial Sand Cover will reduce resuspension.

CHANGE TO RESIDUALS EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER EPS
7. MONITOR RESIDUALS OUTSIDE OF THE DREDGE PRISM a. EPA and GE should establish a residuals monitoring program that evaluates the potential for near-field PCB deposition outside of dredged CUs. b. The program should test the potential for near-field and far-field off site deposition and generation of dredged residuals in low-, moderate-, and high-flow areas downstream of dredged CUs. c. Continued monitoring requirements after Year 1 of the Phase 2 dredging program should be determined by EPA and GE, based on the Year 1 results. d. Adjustments to BMPs or dredge volumes should be considered if the risks associated with the generation of residuals compromise the benefits to be achieved in terms of reduced risks resulting from dredging.	The numerical criteria for off-CU residual deposits should be based on the following: The generation of off site (i.e., off-CU) residuals should not adversely increase baseline surface sediment Tri+PCB concentrations on off-CU areas, such that post-dredge recovery rates are slower than would be achieved via MNR. This evaluation should NOT be based on a comparison of cumulative loads, but instead should be based on long-term fish exposures associated with surface sediment deposits, and should include a combined assessment of dredged and undredged areas.	Phase 1 inadequately evaluated the generation of residuals outside of the dredge prisms and CUs. Near-field and far-field PCB deposition has the potential to adversely increase ecological and human health risks. EPA and GE should evaluate whether off-CU deposits have the potential to increase risks to levels that are unacceptable when compared to the reduced risks associated with dredging and backfilling. The sediment surface (e.g., top 0 - 2") should be characterized and the data used to determine if upstream releases are redepositing in depositional areas, and to determine whether redeposition results in unacceptable changes to the surface sediment in off-CU / off site areas.	No impact on the other EPS is anticipated unless the results indicate a need to change dredging plans, BMPs, and operations, which might decrease productivity and resuspension.

CHARGE QUESTION 4. If EPA and/or GE has proposed modifications to the monitoring and sampling program for Phase 2, are the proposed modifications adequate and practicable for determining whether the Phase 2 Engineering Performance Standards will be met?

Both EPA and GE proposed changes to the EPS with concurrent changes to the monitoring and sampling program for Phase 2. However, the Panel finds that it will not be practicable to consistently and simultaneously meet the EPS being proposed by either party and, thus, cannot make a finding regarding the monitoring and sampling programs relative to these proposed standards except for those items that have been specifically addressed under Charge Question 2, above. Rather, the Panel has addressed Question 4 relative to the modified EPS and processes recommended by the Panel in response to Charge Question 3.

Finding Rdl.4: The experience in Phase 1 shows that the monitoring and sampling program for residuals in Phase 2 will need more rapid characterization of surficial samples to determine whether dredging residuals—based on a proactive determination of the DoC—can be backfilled or should be capped.

Since risk is driven by average surficial contaminant concentrations, confirmation sampling of residuals for verifying attainment of the Residuals EPS should be based on surficial samples that are composited to represent an average surface. With a well-characterized DoC, dredging will remove the vast majority of the inventory, ideally leaving only generated residuals without any undredged inventory; therefore, there is little reason to monitor for contamination at depths below the top 6 inches of dredged sediment surface. Because risk reduction will be provided by the isolation created by the backfill or the cap covering all dredged areas, little benefit is gained from attempting to remove the small contaminant mass present in generated residuals. The average Tri+PCBs concentration in the composite of surficial residuals samples is compared with the residuals criteria to determine if the area can be backfilled or should be capped as described in Table 12. The Residuals EPS monitoring for removal of inventory should focus on determining whether a dredged area has removed the sediment down to the Design Dredge Elevation in 95 percent or more of the dredged area. PCB mass removal should be based on the DoC coring program results.

After the bathymetric survey results of the dredged subunit are verified, the 1-acre subunit should be sampled as soon as practicable to determine whether the residuals need to be backfilled or capped. Eight 6-inch-deep samples (1-foot samples followed by removal of the sand layer if collected after sand cover is placed) should be collected, composited into a single sample, and analyzed for Tri+PCBs concentration. If the 1-acre composite concentration is less than 3 ppm Tri+PCBs, then the subunit should be backfilled to close the area; otherwise, the subunit should be capped. 3 ppm Tri+PCBs is selected as the decision criterion because it is representative of the concentration achieved in Phase 1 at the end of the cleanup passes. It is also representative of the surficial concentration outside the dredge areas in TIP and, as such, is comparable to the concentration that would result from recontamination by surrounding undredged sediments.

In Phase 1, about 25 percent of the high-confidence areas had Tri+PCB concentrations greater than 3 mg/kg and the apparent DoC was off on average about 6 inches (GE Table 6.1-3). With improved DoC delineation and an allowance for vertical dredge tolerance in setting the Design Dredge Elevation, the Panel expects the inventory to be routinely removed.

However, based on the failure to correctly establish the DoC during Phase 1, and thus the lack of performance data to gauge the adequacy of the Panel's recommended DoC delineation approach, a limited confirmation monitoring program is recommended to verify the effectiveness of the updated DoC delineation approach. For example, following GE's development of the updated DoC for the first year of Phase 2, a limited number of cores may be collected to confirm that the DoC was adequately characterized, by analyzing the cores in 6-inch sections for Tri+PCB. The results of the confirmation samples may be used to adjust the coring density in subsequent years during Phase 2, particularly if the additional cores do not adequately validate the updated DoC for the first year of Phase 2.

The additional cores could be completed either before or after the dredging for the first year of Phase 2. If done after dredging, the Panel does not recommend redredging of any missed inventory, as doing so would adversely impact resuspension and productivity. As appropriate and as necessary for the design process, geotechnical testing (i.e., water content, organic matter, etc.) should also be performed on these core sections to permit better interpretation of the findings for the adaptive management process.

5 PRODUCTIVITY

CHARGE QUESTION 1. Does the experience in Phase 1 show that each of the Phase 1 Engineering Performance Standards can consistently be met individually and simultaneously?

The 2004 Phase 1 Productivity EPS for the Hudson River PCBs Superfund Site defines "productivity" as the volume of sediment in cubic yards (cy) that is removed from the waterway, processed, and shipped off site to an approved landfill for permanent disposal, per unit of time.

Specifically, the Productivity EPS states:

> *The minimum volume of sediment to be removed, processed, and shipped off site during Phase 1 shall be 200,000 cubic yards.*

The removal component in this report includes the dredging and haul barge transport to the processing site. Project productivity criteria are expressed as cy/day, cy/mo, and cy/yr.

Contrary to this definition, the GE and EPA Phase 1 Evaluation Reports both used *productivity* to refer to the output of a single operational component (i.e., removal). Thus, both reports incorrectly evaluated project performance by comparing dredging output to the Productivity EPS, which explicitly includes all 3 outputs (i.e., "The minimum volume of sediment to be removed, processed, and shipped off site…").

> *The maximum monthly dredging production rate achieved during Phase 1 was approximately 78,000 cy, only 12 percent less than the Phase 1 requirement of 89,000 cy.* (EPA's Phase 1 Evaluation Report, Hudson River PCBs Site, pg. ES-20).

> *The best 1-month production that was accomplished in Phase 1 was 77,300 cy. More typically, the weekly productivity rate during Phase 1 resulted in a monthly production of 64,000 to 77,000 cy. This rate is 15% to 30% lower than the production rate necessary to achieve the 89,000 cy per month target rate.* (GE's Phase 1 Evaluation Report, Hudson River PCBs Superfund Site, pp. ES-23 & -24).

In both cases, the "production rate" or "productivity rate" refers only to the achieved removal output, and did not account for processing or shipping outputs. Output is distinct from productivity as it relates to 3 individual components of the sediment remediation project; productivity represents the total volume of material that is handled by all 3 components over a specified time.

The project's Phase 2 annual productivity goal was set at 490,000 cy/yr. EPA's criteria for monthly productivity changed between the time EPA issued the 2004 EPS for Dredging and the start of Phase 1 dredging in 2009. EPA originally based its monthly productivity standard on a 7-month dredging season, which yielded a 70,000 cy/mo standard (490,000 cy divided by 7 months = 70,000 cy/mo). During remedial design, the planned dredging period was changed from 7 months to 5 ½ months. As a result, EPA revised its monthly productivity standard to 89,000 cy/mo (490,000 c y divided by 5.5 months = 89,090 cy/mo).

Finding P.1: Phase 1 did not achieve the 2004 Productivity EPS and the experience in Phase 1 does not show that the Productivity EPS can be met for Phase 2.

The Panel evaluated Phase 1 outputs reported in Appendix D[1] and Appendix E[2] of GE's *Phase 1 Evaluation Report, Hudson River PCBs Superfund Site* (March 2010) against EPA's 4 numerical productivity criteria. As shown in Table 13, Phase 1 did not meet any of the productivity criteria set forth in the 2004 EPS. Phase 1 also failed to demonstrate that the existing Productivity EPS could be met in Phase 2.

Table 13. Phase 1 productivity results vs. 2004 EPS for Dredging

Productivity Standard 2004 EPS for Dredging	Numerical EPS	Achieve EPS?	Actual Phase 1 Productivity	Discussion
1.a. Minimum Phase 1 volume to be removed, processed, and shipped off site during Phase 1.	200,000 cy	No	100,000 cy 50% of EPS	The total material removed and processed was 371,229 tons. Of that, 128,550 tons were shipped off site (35% of total). 35% of the total reported volume removed (286,354 cy) is 100,000 cy. 100,000 cy/yr is 50% of the 200,000 cy/yr EPS.
1.b Targeted Phase 1 volume to be removed , processed, and shipped off site during Phase 1.	265,000 cy	No	100,000 cy 38% of EPS	Only 100,000 cy was removed, processed and shipped off site during Phase 1. See discussion 1.a. 100,000 cy/yr is 38% of the 265,000 cy/yr EPS.
2. Minimum 1-month production rate, for removal, processing, and shipping off site.	89,000 cy/mo	No	42,400 cy/mo 48% of EPS	The peak 1-month productivity for Phase 1 removal, processing, and shipping off site was 42,400 cy/mo, achieved during the period ending October 17, 2009. It was controlled by the shipping output (42,400 cy/mo) which was less than the processing output (62,800 cy/mo) and less than the removal output (63,300 cy/mo) during that period. See Table 14. 42,400cy/mo is 48% of the 89,000 cy/mo EPS
3. All material removed and processed shall be shipped off site to final disposal by end of calendar year.	100% shipped off site	No	35% shipped 35% of EPS	See discussion 1.a above.

Finding P.1-1: Phase 1 achieved 50 percent (100,000 cy / 200,000 cy = 0.5) of the minimum volume specified in the Productivity EPS.

The minimum volume of 200,000 cy/yr applies to the volume of design inventory sediment that was removed, processed, and shipped off site during 2009 (see footnote 3 to EPA's Table 2-6 of the 2004 EPS). During Phase 1, transportation issues and delays constrained productivity to the extent that only 35 percent of the material that was removed and processed was actually shipped off site by the end of

[1] Appendix D is titled *Detailed Discussion of Productivity During Phase 1 Dredging*, and is referred to as "GE's Appendix D."

[2] Appendix E is titled *Detailed Discussion of Processing and Disposal During Phase 1 Dredging* and is referred to as "GE's Appendix E."

the calendar year. Specifically, of the total volume removed during 2009 (286,354 cy), only about 100,000 cy was shipped off site.[3]

GE reported delays in off site transport in July and August, relating to the cleaning and marking of empty rail cars, as well as later delays due to materials management problems at the disposal cell. Consequently only 2 unit trains[4] were shipped during June, July, and August of 2009 (1 unit train is approximately 8,350 tons, or about 6,400 cy of dredged material). Eleven unit trains were shipped off site from mid-September through the end of October (see GE's Table E-5), averaging just under 1.5 unit trains per week. During the following 6 weeks, there was no off site transportation. The year ended with 2 unit trains plus a partial train (2,900 tons) shipped off site during the last 2 weeks of December.

Finding P.1-2: Phase 1 achieved 38 percent (100,000 cy / 265,000 cy = 0.38) of the target volume specified in the Productivity EPS.

The Phase 1 Target Volume EPS of 265,000 cy/yr also applies to the volume of design inventory sediment that was removed processed, and shipped off site during 2009 (see footnote 3 to EPA's Table 2-6 of the 2004 EPS). Again, by the end of Phase 1 the transportation component constrained the total volume of material that was actually removed and processed and shipped off site, with approximately 100,000 cy shipped off site by the end of the calendar year.

GE reported a Phase 1 total dredging output of 286,354 cy, of which 144,438 cy was designated as design inventory, 119,964 cy as extra inventory, and 21,952 cy as residual dredging (GE's Table D-4). In accordance with the 2004 EPS for Dredging, only design inventory counts toward meeting the Production EPS (see footnote 3 to EPA's Table 2-6 of the 2004 EPS). During Phase 1, GE requested and EPA agreed to include the extra inventory (119,964 cy, GE's Table D-4) that was removed towards the productivity target. The actual Phase 1 removal output (design inventory plus extra inventory) is 264,402 cy/yr, which happens to approximate the productivity target volume of 265,000 cy. However, as discussed previously, removal output is not the same as volume removed, processed, and shipped off site, and thus does not represent achievement of the Phase 1 Productivity EPS.

Finding P.1.3: Phase 1 achieved 48 percent of the minimum monthly productivity (42,400/89,000 = 0.48) specified in the 2004 EPS for Dredging.

The Phase 1 Minimum 1-Month Productivity of 89,000 cy/mo applies to the volume of design inventory sediment that was removed, processed, and shipped off site for a continuous 1-month period during 2009 to "*verify the capabilities of the dredging operations, including the equipment and the sediment processing and transportation systems*" (pg. 66, 2004 EPS for Dredging).

Evaluation of the peak monthly production rate requires a tabulation of monthly removal output, monthly processing output, and monthly transportation output, to identify the peak monthly volume that was removed and processed and shipped off site (productivity). GE's Table D-5 presents a 30.66-day running total of monthly removal output by 3 categories: design inventory, residual and extra

[3] 128,550 tons was shipped during Phase 1 and 243,000 tons remained on site, which calculates as 35 percent of the material that was removed and processed (371,229 tons). The total volume reported as dredged (removed) by GE was 286,354 cy (Table D-4). 35 percent of 286,354 cy is approximately 100,000 cy removed, processed, and shipped off site.

[4] Unit train: 81 cars each carrying approximately 103 tons, or about 8,350 tons per unit train.

inventory, and total volume. Comparable cumulative 30.66-day running output tabulations were not found for sediment processing and sediment transportation off site.

GE's Table E-5 provides a weekly total output in tons/wk for sediment processing and shipping. The Panel estimated monthly outputs in cubic yards based on these data, as shown in Table 14.

- The running total of tonnage processed or shipped over the 4 weeks leading up to the noted week-ending dates on Table 14 were calculated and posted in the "4-week" column.
- The tonnage processed or shipped over a month (30.66 days) was approximated by multiplying the tonnage processed over 4 weeks by a time-based scaling factor (30.66 days / 28 days = 1.095) and posted in the "Month" column.
- The monthly tonnage estimates were converted to cubic yard outputs (rounded to 100 cy) by dividing the tonnage production by 1.3 tons / cy.

The 1.3 tons/cy factor is based on the reported 371,229 tons processed during Phase 1 divided by the reported 286,354 cy removed during Phase 1 (371,229 / 286,354 = 1.3).

The calculated outputs, in cubic yards per month, are presented on Table 14 under the header "Process/Ship Output (cy/mo)" for both processing and off site shipping. The last 2 columns of Table 14 present the monthly removal output from GE's Table D-5 for the week-ending dates listed. Note that GE's Table D-5 and Table 14 provide a different tonnage summary. Table D-5 provides a monthly cumulative removal output for every day of dredging from June 4, 2009 through October 31, 2009, while Table 14 only presents a monthly cumulative output calculation once per week.

Table 14. Phase 1 monthly output summary

		Tonnage Processed			Tonnage Shipped			Process Ship Output (cy mo)		Removal Output (cy mo)	
Week	Ending	GE Table E-5			GE Table E-5			at 1.3 tons cy		GE Table D-5	
		Week	4-week	Month	Week	4-week	Month	Processed	Ship	Design Inv	Total
1	5/9/2009	0			0						
2	5/16/2009	1330			0						
3	5/23/2009	1200			0						
4	5/30/2009	1300	3,830	4,195	0	0	0	3,200	0		
5	6/6/2009	4656	8,486	9,295	0	0	0	7,200	0	8,171	8,171
6	6/13/2009	8502	15,658	17,151	0	0	0	13,200	0	14,554	14,554
7	6/20/2009	13318	27,776	30,425	0	0	0	23,400	0	22,556	22,556
8	6/27/2009	12,231	38,707	42,398	8,447	8,447	9,252	32,600	7,100	31,172	31,172
9	7/4/2009	10,013	44,064	48,266	8,366	16,813	18,416	37,100	14,200	35,295	35,295
10	7/11/2009	13,480	49,042	53,719	0	16,813	18,416	41,300	14,200	40,105	40,105
11	7/18/2009	18,160	53,884	59,022	0	16,813	18,416	45,400	14,200	50,133	50,133
12	7/25/2009	22,432	64,085	70,196	0	8,366	9,164	54,000	7,000	58,052	58,533
13	8/1/2009	24,525	78,597	86,092	0	0	0	66,200	0	66,987	68,045
14	8/8/2009	22,321	87,438	95,776	0	0	0	73,700	0	**71,423**	**75,566**
15	8/15/2009	16,054	85,332	93,469	0	0	0	71,900	0	53,966	66,254
16	8/22/2009	24,543	87,443	95,781	0	0	0	**73,700**	0	39,856	65,326
17	8/29/2009	19,896	82,814	90,711	0	0	0	69,800	0	24,530	63,200
18	9/5/2009	18,746	79,239	86,795	0	0	0	66,800	0	11,940	59,199
19	9/12/2009	16,432	79,617	87,209	16,652	16,652	18,240	67,100	14,000	15,220	70,632
20	9/19/2009	18,171	73,245	80,229	0	16,652	18,240	61,700	14,000	16,592	72,897
21	9/26/2009	19,290	72,639	79,566	16,784	33,436	36,624	61,200	28,200	20,909	69,577
22	10/3/2009	18,861	72,754	79,692	8,430	41,866	45,858	61,300	35,300	17,826	67,926
23	10/10/2009	17,384	73,706	80,734	16,709	41,923	45,921	62,100	35,300	12,790	71,403
24	10/17/2009	18,989	74,524	81,630	8,382	50,305	55,102	62,800	**42,400**	8,198	63,267
25	10/24/2009	18,253	73,487	80,495	16,765	50,286	55,081	61,900	42,400	2,985	62,881
26	10/31/2009	11,367	65,993	72,286	8,392	50,248	55,040	55,600	42,300	0	51,897
27	11/7/2009	0	48,609	53,244	0	33,539	36,737	41,000	28,300	0	N/A

Table 14 presents an estimated monthly cumulative output (cy/mo) for removal, processing, and shipping as of the last day of each week of the project. The tabulation only goes through week 27 (11/7/2009), following the last week of Phase 1 dredging and processing.

The peak cumulative monthly output for each component (removal, processing, and shipping) is bolded and boxed on Table 14. The maximum cumulative removal output for both design inventory (71,423 cy/mo) and total volume (75,566 cy/mo) occurs during the month ending August 8, 2009.[5] During that same time period, the processing output is 73,700 cy/mo, the same as the maximum processing output for the period ending 8/22/2009. However, no shipping occurred in August. Consequently, the peak removal output reported in August cannot satisfy the Productivity EPS.

The maximum cumulative monthly productivity for removal, processing, and off site transportation is 42,400 cy/mo, recorded during the period ending October 17, 2009. During this period, the removal output was 63,267 cy/mo,[6] the processing output was 62,800 cy/mo, and the transportation output was 42,400 cy/mo.

Table 15 presents the peak individual monthly outputs achieved during Phase 1 for removal, processing, and shipping, based on the calculations presented in Table 14. Both removal and processing achieved a peak monthly output on the order of 70,000 to 75,000 cy/mo, while shipping off site peaked at 42,400 cy/mo. EPA's Phase 2 targeted monthly production rate of 89,000 cy/mo was not achieved by any of the 3 individual components (removal, processing, or shipping) during Phase 1.

Table 15. Peak Phase 1 monthly output rates

Production Component	Peak Output cy/mo	Period Ending
Removal		
Design Inventory	71,423	8/8/2009
Total Volume	75,566	8/8/2009
Processing	73,700	8/22/2009
Shipped off Site	42,400	10/17/2009

Finding P.1-4: Phase 1 achieved 35 percent of the off site shipping standard specified in the Productivity EPS.

The Productivity EPS requires that all material removed be processed and shipped off site for disposal by the end of the calendar year. Only 35 percent of the material removed and processed was actually shipped off site by the end of the calendar year.

[5] Note that GE's Table D-5 reports the maximum design inventory removal of 73,377 cy/mo and maximum total volume removed of 77,284 cy/mo on the period ending August 7, 2009. The discrepancy between Table 14. Phase 1 monthly output summary and GE's Table D-5 is due to GE's daily calculation of running totals; Table 14. Phase 1 monthly output summary shows the results of weekly calculations.

[6] 63,267 cy assumes that dredging production can include both design inventory and extra inventory volume to meet the standard. However, if meeting the standard can only be based on design inventory removal, as stated in the 2004 Productivity EPS, then the peak monthly production rate would be 20,909 cy/mo as achieved during the period ending 9/26/2009, when the processing rate was 61,200 cy/mo, the shipping rate was 28,200 cy/mo, and the design inventory removal rate was 20,909 cy/mo.

CHARGE QUESTION 2. If not, and if EPA and/or GE has proposed modified Engineering Performance Standards, does the experience in Phase 1 and any other evidence before the panel show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2?

Finding P.2: The experience in Phase 1 and other evidence before the Panel does not show that it will be practicable to consistently meet the Productivity EPS proposed for Phase 2 by EPA and GE.

Both EPA and GE have proposed changes to the Productivity EPS for Phase 2. Some of these changes have merit, as discussed below. However, collectively, the changes do not result in a consistently achievable EPS that meets the requirements of the ROD and facilitates simultaneous achievement of the Resuspension EPS and Residuals EPS. For example, EPA's proposed annual required and targeted productivity criteria are not practicable for Phase 2. The total volume to be removed, processed, and shipped is likely underestimated by EPA and consequently the annual and monthly productivity rates to complete the program in 5 years is likely underestimated. The annual and monthly productivity rates that are actually achievable are well below EPA's recommended required productivity rates. On the other hand, GE is essentially recommending that productivity be eliminated from the Phase 2 EPS, reflecting the Panel's concerns expressed during the public Peer Review meetings. This is certainly practicable, but may not be in keeping with the ROD.

Table 16. Summary of EPA's proposed modifications to the Productivity EPS

<table>
<tr><th>EPA's Proposed Change to Productivity EPS</th><th>EPA's Proposed Numerical Criteria</th></tr>
<tr><td>1. Add a provision to extend the timeframe for Phase 2 at the discretion of EPA.</td><td>Every reasonable effort will be made to maintain the 5-year duration of Phase 2. EPA may allow 1 or 2 additional years if conditions require.</td></tr>
<tr><td>2. Recalculate the annual required and target productivity volumes to reflect the revised Phase 2 removal volume.</td><td rowspan="2"><table><tr><th>Year</th><th>Required Vol., CY</th><th>Target Vol., CY</th></tr><tr><td>2</td><td>475,300</td><td>528,100</td></tr><tr><td>3</td><td>475,300</td><td>528,100</td></tr><tr><td>4</td><td>475,300</td><td>528,100</td></tr><tr><td>5</td><td>475,300</td><td>528,100</td></tr></table></td></tr>
<tr><td>3. Count sediment volumes removed during residuals dredging and when dredging missed inventory toward meeting required and target volumes listed in the Productivity EPS.</td></tr>
</table>

Table 17. Summary of GE's proposed modifications to the Productivity EPS

GE's Proposed Change to Productivity EPS	GE's Proposed Numerical Criteria
1. No firm Productivity EPS. Allocate the PCB load (revised Resuspension EPS) among CU areas, specifically targeting the CU areas where PCBs are, or may become, bioavailable.	Eliminate numerical criteria for target and required volumes. Productivity to be governed by the Resuspension and Residuals EPS criteria.
2. Implement Phase 2 such that the goal is to complete the removal within 5 years.	No numerical criteria. Project incorporating GE's approach can likely be completed within 5 years.
3. Change the metric for tracking productivity from sediment volume removed to area remediated	None presented. Area remediated is a measure of benefits achieved and an appropriate means to track production.

Finding P.2-1: There should be flexibility in the Phase 2 timeframe to accommodate anticipated and unanticipated conditions that will be encountered during the work.

EPA has proposed extending the timeframe for Phase 2 to adjust the project schedule if necessary to accommodate conditions beyond the control of EPA and GE, such as extreme flows, force majeure, or the discovery of significant additional inventory to be removed, as well as possible resuspension impacts. This proposal is consistent with the Phase 1 experience, which demonstrated that many factors were not understood or anticipated when Phase 1 dredging was initiated. Examples include:

- Phase 1 removed 83 percent more contaminated sediment than was anticipated by the design. According to GE's Table D-4, Phase 1 removed 144,438 cy of design inventory, and another 119,964 cy of extra inventory (119,964 cy / 144,438 cy = 0.83).
- Transportation and placement of processed material into a Texas landfill encountered significant complications and delays, with only 35 percent of the removed and processed material moved off site during 2009 (100,000 cy / 286,354 cy = 0.35).
- Dredges spent 24 percent of the available dredging time waiting for barges (GE's Figure D-15) due to numerous issues associated with the complexity of the project. The issues included shallow draft in some CUs, higher than normal river discharge, transfer time from mini hopper barges to deeper-draft hopper barges, controlling PCB volatilization, offloading, and processing variable material types from sand to silt to stiff clay.

Experience during Phase 1, as well as experience of the Panel members at other large complex sediment remediation projects, demonstrates the need for schedule flexibility to deal with the complexities and complications that arise during the remedial action.

Finding P.2-2: Extra inventory and residual dredging should be included as part of tracking productivity.

The 2004 EPS for Dredging explicitly states that only material included in the dredge prism of the final design (design inventory) will count toward meeting the Productivity EPS (see footnote 3 to Table 2-6 of the 2004 EPA for Dredging). EPA's revised Productivity EPS proposes to count all sediment volumes removed, including missed inventory, toward meeting required and target volume criteria. GE requested—and EPA approved—a change for Phase 1 to count extra inventory towards meeting the Productivity EPS. EPA has proposed that since there is some uncertainty in the remaining inventory to be removed for Phase 2, and since all dredging contributes to resuspension losses, the extra inventory

and residual dredging should count toward the Phase 2 Productivity EPS. The Panel concurs with this proposal.

Finding P.2-3: Depth of contamination (DoC) is not well-defined, leading to likely underestimates of total and annual required and target volumes.

Setting annual and project target volumes requires knowledge of the amount of material yet to be removed. EPA found that there are insufficient data available at present to complete a rigorous analysis to determine the remaining volume of material to be removed. Instead, EPA started with the original design estimate of volume to be removed in the remaining CUs (1,664,500[7] cy) and multiplied it by various scaling factors to update the estimate of volume to be removed during Phase 2.

EPA employed 3 methods to estimate the remaining volume. First, EPA multiplied the original design estimate of volume remaining by a factor of 1.6, which is the ratio of actual Phase 1 dredging (design plus extra inventory) divided by the design volume, with CU-1 excluded from the calculation.[8] This resulted in an estimate of remaining material to be removed of 2,663,000 cy.[9] Second, EPA applied a Phase 1 experience factor[10] to increase the assumed DoC by 1.13 feet beyond GE's design estimates, and applied it to the 442 acres yet to be dredged for an added increment of 805,800[11] cy and a total estimate of remaining material to be removed of 2,470,000 cy, which equates to a scaling factor of 1.5 times the original design volume.[12] Third, EPA started with the original ROD estimate of 2,650,000 cy total volume, subtracted their estimate of the Phase 1 dredging (273,600 cy) to come to an estimate of 2,376,500[13] cubic yards yet to be removed. This equates to a scaling factor of 1.43 times the original design volume, and EPA's current estimate of Phase 2 annual productivity of 475,300 cy/yr.

EPA used the 2,376,500 cy estimate of total volume yet to be removed to derive recommended changes to the annual production rate criteria: 475,300 cy/yr and a monthly average of 86,420 cy/mo.

The Panel finds significant shortcomings with these estimates, which do not account for changes in lithology between Phase 1 and Phase 2 areas, anticipated TIP deposits, and uncertainty associated with current DoC estimates. EPA's proposed Phase 2 annual productivity criterion is it is based on the low end of the estimated range of possible dredging volume remaining. However, if CU-1 is not excluded from the experience during Phase 1, then Phase 1 removed a volume equal to 1.83 times the design

[7] Following the May 2010 deliberation meeting, GE provided a table of the design volume for each of the remaining COs, totaling 1,664,500 cy.

[8] GE's Table D-4 identifies Phase 1 dredging as 144,438 cy of design inventory and 119,964 cy of extra inventory for a total "design plus extra inventory" volume of 264,402 cy. GE's Table D-10 identifies 34,363 cy of extra inventory dredging for CU-1 (sum of 1st, 2nd, 3rd, and 4th redredge passes). Subtracting the CU-1 extra inventory from the overall summation results in an estimate of total "design plus extra inventory" of 230,039 cy. EPA's scaling factor of 1.6 is calculated as 230,039/144,438 = 1.6.

[9] Multiplying 1,664,500 cy by 1.6 yields an estimate of 2,663,000 cy remaining to be dredged.

[10] Excluding CU-1 from the calculation, EPA reported that the net increase in volume dredged during Phase 1 was 82,100 cy over an area of 44.86 acres, or an average of 1.13 feet of increased dredging depth.

[11] See Section 3.3, pg III-25, of EPA's Phase 1 Evaluation Report, March 2010.

[12] Adding in 805,800 to the original design volume of 1,664,500 cy brings the estimate of volume remaining to 2,407,300 cy. The scaling factor is calculated as 2,470,300/1,664,500 = 1.5.

[13] See Section 3.3, pg iii-26, of EPA's Phase 1 Evaluation Report, March 2010.

volume (264,402 cy / 144,438 cy = 1.83). Applying a scaling factor of 1.83 to the design volume (1,664,500 cy) yields an estimate of 3,050,000 cy yet to be removed.

Until the DoC is better defined, EPA should recognize the potential that the estimate of material remaining to be removed could be significantly greater than anticipated. Based on the upper end of the range of values presented (i.e., 2,376,500 to 3,050,000 cy), the monthly productivity requirement is 86,400 to 111,000 cy/mo, based on a 5-year project with a 5.5 month dredging season.

Based on the results of Phase 1 and the Panel's productivity calculations (refer to the discussion under Charge Question 3, following), it is not expected that these rates can be practicably and consistently met during Phase 2**.**

Finding P.2-4: The Productivity EPS should not be eliminated.

GE's proposed the elimination of the Productivity EPS while applying a revised Resuspension EPS and Residuals EPS to constrain the volume of sediment to be removed during Phase 2. GE's proposal would likely require a ROD amendment as it deviates from a fundamental ROD requirement. This represents a significant shift in the remedial action objectives, and additional studies and evaluations would be required before such an approach could be approved. The Panel was not presented with sufficient evidence to support the need for eliminating productivity considerations entirely and strictly limiting the volume of sediment to be dredged, nor did the Panel's charge include an evaluation of the requirements of the ROD.

For the revised EPS, the Panel recommends that EPA and GE explicitly acknowledge that there are tangible and substantial trade-offs between dredging production rates and the potential for resuspension and residual generation. Thus, the Panel supports the use of productivity targets rather than standards, as strictly defined. In this sense, the productivity target would be informed by a more complete understanding of how operational activities contribute to sediment resuspension and residuals formation and what the short- and long-term environmental implications of resuspension and residuals are for achieving remedial objectives pertaining to both the upper and lower river.

The Panel understands that both GE and EPA are working to incorporate Phase 1 data into models that are expected to provide insight regarding the relationship between dredge productivity and resuspension/residuals. Iterative use of such modeling should be used in conjunction with onsite adaptive management to calibrate productivity, both within and between operational seasons, in a manner that preserves the integrity of the project's risk reduction objectives over the long term. This approach must recognize uncertainties associated with future operations, including conditions that cannot be predicted today and unanticipated operational adjustments that will be needed to accommodate those conditions.

In addition, the Panel recommends that the project team develop productivity targets for closing CUs in an efficient and rapid manner, as this particular aspect of the operation is most closely related to achieving remedial objectives for the upper river.

CHARGE QUESTION 3. If the experience in Phase 1 and other evidence before the Panel does not show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2, can the Phase 1 Engineering Performance Standards be modified so that they could consistently be met in Phase 2, and, if so, how?

Finding P.3: The Phase 1 Productivity EPS can be modified to be consistently met in Phase 2.

In order to evaluate the practicability of the existing and proposed Productivity EPS, and to develop practicable modifications, the Panel assessed likely annual productivity that could be achieved on the Upper Hudson. This involved estimating the possible annual output of dredging, processing, and transport from several perspectives, including:

- Peak monthly output achieved during Phase 1 for each component of the remedial action (i.e., dredging, processing, and transportation)
- Added dredging output that would have been achieved during Phase 1 if the dredges had all started at the beginning of the season and if the impacts from CU-1 were removed
- Removal output, assuming barge arrival and waiting times were improved
- Shipping output, assuming rail and landfill issues are resolved and no longer a significant productivity limitation

Through review of Phase 1 operations, the Panel did not discover any single factor that could be adjusted to significantly increase overall productivity. For example, neither increasing the number of barges in service nor increasing the offload rate at the processing facility provided a dramatic increase in productivity. Rather the Panel found multiple lines of evidence that indicated 350,000 cy/yr as a reasonable annual productivity estimate for the start of Phase 2.

The Panel's recommendations for modifying the Productivity EPS are summarized in Table 18, and discussed further below.

Table 18. Summary of recommended changes to the Productivity EPS

CHANGE TO PRODUCTIVITY EPS	NUMERICAL CRITERIA	RATIONALE	IMPACT ON OTHER STANDARDS
1. TOTAL VOLUME Eliminate total volume from the Productivity EPS.	None	EPA found that there are insufficient data available at present to determine the volume remaining to be removed in the remaining CUs. The Panel agrees, primarily because of incomplete DoC characterization. Estimates of material remaining range from about 2.3 million to 3.0 million cubic yards. Consequently, due to the real uncertainty about material remaining to be removed, the Total Volume should be eliminated as a productivity criterion. It does not make good sense to use an uncertain quantity to set a certain standard. Until DoC is better defined, the Panel recommends that EPA use at least 2,700,000 cy as planning-level estimate of material remaining to be removed.	Shift focus away from annual productivity to managing residuals and resuspension.
2. ANNUAL VOLUME Change annual volume to reflect Phase 1 experience, and adjust the volume annually, based on experience and appropriate adaptive management.	350,000 cy/yr base value, adjusted for site conditions and to meet the Resuspension EPS and Residuals EPS.	Since the total volume to be removed is not known, it is not reasonable to project what the annual production would be based on a 5-year schedule for Phase 2. The Panel's evaluations indicate that 350,000 cy/yr is a reasonable initial planning level production rate for the project, subject to modification due to changing site conditions during Phase 2, (such as different material types, longer barge-transport and lockage requirements, and annual variations in weather and river flow), and productivity modifications necessary to maintain the Resuspension EPS and Residuals EPS.	Shift focus away from annual productivity to managing residuals and resuspension.
3. PHASE 2 DURATION Shift focus away from the Phase 2 duration from the Productivity EPS while still taking into consideration the consequences of prolonged construction activities on the river.	None	Experience during Phase 1, as well as the experience of Panel members at other large complex sediment remediation projects, demonstrates the need for schedule flexibility to deal with the complications that arise during the remedial action. In addition, the productivity schedule should be subordinated to the Resuspension EPS and Residuals EPS. For planning purposes, the duration of Phase 2 can be roughly estimated by dividing the crude estimate of total volume remaining (2.3 to 3.0 million cy) by a planning level estimate of annual productivity (350,000 cy/yr). The resulting planning-level estimate of the duration of Phase 2 is 7 to 9 years.	Shift focus away from annual productivity to managing residuals and resuspension.

Finding P.3-1: Drop the Total Volume Productivity EPS criterion.

There are insufficient data available to determine the volume remaining to be removed in the remaining CUs, primarily because of incomplete DoC characterization. Existing estimates (with limited confidence) of material remaining range from about 2.3 million to 3.0 million cubic yards. Because it does not make sense to use an uncertain quantity to set a certain standard, the Panel recommends dropping the Productivity EPS criterion for Total Volume.

The Panel recommends establishing monthly and annual volume "targets," combined with established total and annual areas to be remediated. Area remediated reflects a substantial measure of environmental benefit and could be expressed as a specified number of CUs to close each year.

Tracking of total volume and mass of PCBs removed should continue, but the environmental benefit accrued should be based primarily on area remediated.

Finding P.3-2: Initially set the Annual Volume Productivity EPS criterion at 350,000 cy/yr.

Since the total volume to be removed is not known, it is not reasonable to project what the annual production would be based on a 5-year schedule for Phase 2. The Panel's evaluations, described below, indicate that 350,000 cy/yr is a reasonable initial planning level production rate for the project to be applied for the next dredging season. This rate is near to the peak monthly dredging or processing output achieved during Phase 1, and assumes there will be some net output improvement over Phase 1. Maintaining 350,000 cy of annual productivity will likely require that the removal and processing outputs be decoupled from the shipping output. The annual rate is also subject to modification due to changing site conditions during Phase 2, (such as different material types, longer barge-transport and lockage requirements, and annual variations in weather and river flow), and productivity modifications necessary to meet the Resuspension EPS and Residuals EPS.

Finding P.3-2.1: A reasonable target for Phase 2 removal output is 350,000 cy/yr.

Removal rates were evaluated from several perspectives to identify a practicable annual output estimate of 350,000 cy/yr for Phase 2. Considering the multiple factors of uncertainty at the site, the Panel considers 350,000 cy/yr to be a reasonable Phase 2 removal output target, until project experience during Phase 2 demonstrates otherwise. The annual productivity must be managed adaptively, from year to year, and should consider such factors as the revised approach to managing residuals (i.e., the elevation-based design paradigm), changes in barge travel distances and lock throughput requirements from year to year, changes to the sediment bed lithology as dredging progresses downstream, and increased experience and management of BMPs.

Phase 1 Peak Monthly Output

As presented in Table 14, the peak monthly removal output, calculated once per week, was 75,566 cy/mo, for the period ending 8/8/2009. The peak monthly removal output, calculated daily by GE, was 77,284 cy/mo for the period ending 8/7/2009. If these peak outputs are applied to a 5-month dredging season[14], the removal output would be in the range of 375,000 cy/yr to 385,000 cy/yr. It is not reasonable to consider achieving the peak monthly Phase 1 removal output during every month of

[14] Five months is considered a reasonable timeframe for calculating annual dredging output, considering the annual variability in weather and river flows.

Phase 2. A more reasonable estimate for Phase 2 removal would be based on 90 percent of the monthly peak rate, or on the order of 350,000 cy/yr.

Adjusted Phase 1 Total Output

The actual Phase 1 total removal output of 286,354 cy was adjusted upward by estimating the additional output that would have been achieved if all of the dredges started working in mid May (added 67,000 cy) and by factoring out the impacts of significant excess dredging at OU-1 (added another 17,000 cy), for a total adjusted removal output in Phase 1 of 370,000 cy/yr (286,000 cy + 67,000 cy + 17,000 cy).

Dredge Output Calculations

The Phase 1 dredging fleet was used as a basis for estimating dredging output during Phase 2. Considering the relatively small channel dimensions of the river, and the limited draft conditions in the river, it is not evident at this time that adding more dredges to the project will provide a proportional increase in overall dredge output. The peak output of the existing dredging fleet was estimated[15] to be in the range of 375,000 cy/yr to 400,000 cy/yr, assuming that the barge wait times experienced during Phase 1 were reduced considerably during Phase 2. The calculations were based on a dredge effective working time percentage in the range of 50-55 percent, which is reasonable for new-work projects in constricted work areas with multiple potential output constraints. In addition, considering that Phase 2 will have longer barge transport distances, with multiple locks to pass through, a reasonable output estimate would be on the order of 375,000 cy/yr for Phase 2 with the existing dredge fleet. Nonetheless, the Panel maintains that a target removal rate of 350,000 cy/yr is a reasonable estimate to commence Phase 2.

Finding P.3-2.2: A reasonable target for Phase 2 processing output is 330,000 cy/yr.

As presented in Table 15, the peak monthly processing output, calculated once per week, was 73,700 cy/mo, for the period ending 8/22/2009. If applied to a 5-month dredging season, the implied processing output would be 368,000 cy/yr if the peak output during Phase 1 was achieved every month during Phase 2. A more reasonable estimate for Phase 2 processing output would be based on 90 percent of the monthly peak rate, or on the order of 330,000 cy/yr for a 5-month season. The annual processing could be increased if some stockpiling was available to allow processing to occur for a period of time after dredging was completed.

Finding P.3-2.3: A reasonable target for Phase 2 shipping output is 380,000 cy/yr.

As presented in Table 15, the peak monthly shipping output, calculated once per week, was 42,400 cy/mo, for the period ending 10/17/2009. It was achieved by averaging 1.5 unit trains per week.

[15] Dredge output calculations for existing fleet: Dredge D385 (4 in fleet): 5 cy bucket, dredge 45cy/effective hr., cap 149 sy/effective hr.; Dredge D345 (1 in fleet): 2 cy bucket, dredge 24.4 cy/effective hr., cap 135 sy/effective hr.; Dredge D320 (7 in fleet): 1 cy bucket, dredge 16.1 cy/effective hr., cap 85 sy/effective hr.; 120 work days: 154 calendar days May 15 to October 15, 22 maintenance days (Sundays), 4 vacation days, 8 non-working days (high flows, resuspension, contingency). Assume 1 sy capping for every cy dredged based on Phase 1. Annual dredge output is approximately 345,000 cy/yr at 45 percent effective working time (EWT), 375,000 cy/yr at 50 percent EWT, and 400,000 cy/yr at 55 percent EWT. Effective working times higher than 55 percent are not considered appropriate for planning a new-work sediment remediation project with multiple and complex operational constraints, including but not limited to the presence of significant debris / rock substrate / clay substrate, shallow draft, small navigation channel, river locks, potential high river flows, and output constraints related to resuspension and air quality.

If applied to a 5-month season, 1.5 unit trains output per week would be about 210,000 cy/yr.[16] Recognizing that shipping can continue longer each year because it is not constrained by the river conditions, a 7-month shipping season would be about 295,000 cy/yr, and a 9-month shipping season would be about 380,000 cy/yr.

Phase 1 established that 2 unit trains could be loaded and shipped in a week's time. Two unit trains were shipped every other week from the week ending 9/12/2009 through the week ending 10/24/2009. GE's Appendix E indicated that a unit train could be loaded every 2 days. Provided that other site factors would not limit the ability to ship 2 unit trains per week, the monthly shipping output at 2 trains per week would be approximately 55,000 cy/mo. This would equate to 275,000 cy/yr for a 5-month season, 385,000 cy/yr for a 7-month season, and 495,000 cy/mo for a 9-month season. If the duration of shipping is decoupled from the dredging season, which would be reasonable to do, then the annual shipping output can match the estimated annual dredging and production outputs.

Finding P.3-3: The project, as designed, cannot be completed in 5 years.

For initial Phase 2 planning purposes, the duration of Phase 2 can be roughly estimated by dividing the current estimate of total volume remaining (2.3 to 3.0 million cy) to be removed by a planning level estimate of annual productivity (350,000 cy/yr). The resulting planning-level estimate of the duration of Phase 2 is 7 to 9 years. As the DoC is refined with improved coring results, and as annual productivity is demonstrated, the total volume estimate and duration of Phase 2 can be refined accordingly.

Experience during Phase 1, as well as the experience of Panel members at other large sediment remediation projects, demonstrates the need for schedule flexibility to deal with the complications that arise during the remedial action, as discussed above. In addition, the productivity schedule should be subordinated to the Resuspension EPS and Residuals EPS. Consequently the 5-year productivity criterion should be dropped in favor of providing more flexibility to complete the work in a manner that protects the integrity of the project and its risk reduction objectives.

[16] 1.5 unit trains per week, at 8,350 tons per unit train, is 12,500 tons/week, or 54,750 tons / mo, which is roughly 42,000 cy/ mo (assuming 1.3 tons/cy), or 210,000 cy over 5 months.

CHARGE QUESTION 4. If EPA and/or GE has proposed modifications to the monitoring and sampling program for Phase 2, are the proposed modifications adequate and practicable for determining whether the Phase 2 Engineering Performance Standards will be met?

Both EPA and GE proposed changes to the EPS with concurrent changes to the monitoring and sampling program for Phase 2. However, the Panel finds that it will not be practicable to consistently and simultaneously meet the EPS being proposed by either party and, thus, cannot make a finding regarding the monitoring and sampling programs relative to these proposed standards except for those items that have been specifically addressed under Charge Question 2, above. Rather, the Panel has addressed Question 4 relative to the modified EPS and processes recommended by the Panel in response to Charge Question 3.

Improving the efficiency and timeliness of closing CUs will require a significant improvement in the accurate definition of DoC before dredging is initiated. It will also require the implementation of an ongoing adaptive management program where various “best management practices” for removal are evaluated with regard to productivity, resuspension, and residuals generation, and then either accepted, modified, or rejected. The Panel’s response to Charge Question 3 for Residuals provides additional discussion of the need for improved DoC characterization, as well as a discussion of monitoring programs to support adaptive management of the removal activity to reduce resuspension and generation of residuals.

6 SUMMARY BY CHARGE QUESTION

The preceding sections present the Panel's detailed review of the charge questions, with each section devoted to a different EPS. This section reorganizes the information presented in the preceding sections and addresses the charge questions in order. This section is intended to provide a synopsis, and the summary information presented herein should not be used as a substitute for the detailed findings and recommendations presented in Sections 3 through 5.

6.1 Summary of Response to Charge Question 1

CHARGE QUESTION 1. Does the experience in Phase 1 show that each of the Phase 1 Engineering Performance Standards can consistently be met individually and simultaneously?

The experience in Phase 1 does not show that each of the Phase 1 EPS can be met consistently, individually, and simultaneously. None of the Phase 1 EPS were met consistently during Phase 1. The EPA and GE evaluations of the Phase 1 experience do not provide evidence that the EPS could be met consistently and simultaneously if applied without modification during Phase 2.

The Resuspension EPS were not achieved in Phase 1. Total PCB concentrations and total and Tri+PCB loads were not met consistently. Suspended solids concentration requirements were met; however, the Panel does not consider this parameter relevant to understanding PCB resuspension and release. Resuspension was likely due to a combination of factors including dredge operations and the management of the CUs. Evidence from Phase 1 does not suggest that this standard could be met without modification during Phase 2.

The Residuals EPS were not achieved in Phase 1. The Residuals EPS were developed based on the assumption that all inventory would be removed with a maximum of 2 passes, followed by additional passes to remove dredge-generated residuals. However, the EPS did not work as envisioned in Phase 1, mainly because inventory was improperly characterized, requiring multiple production passes and leaving CUs open longer than intended. Similar issues would be expected if the Residuals EPS were to be applied without modification during Phase 2.

The Productivity EPS were not achieved in Phase 1. None of the 4 numerical productivity criteria (i.e., minimum removal, target removal, maximum monthly rate, and transportation of all material off site by the end of the year) was achieved. The goal of transportation and disposal of all Phase 1 dewatered sediment by the end of 2009 was not accomplished. While ramping up of individual unit processes is possible, the project cannot be scaled up to meet the anticipated inventory using the current design data.

Phase 1 demonstrated that the 3 EPS were not and cannot consistently be met simultaneously. In the attempt to meet the Residuals EPS under the conditions of inadequately characterized DoC, CUs were left open longer than intended. As a result, disturbed residuals layers were left exposed and subject to erosion by currents and vessel traffic. Erosion of the residuals layer was likely a significant source of resuspension. The 3 EPS cannot be consistently met simultaneously without significant modifications that take into account the complex interactions among operational factors and release mechanisms.

6.2 Summary of Response to Charge Question 2

CHARGE QUESTION 2. If not, and if EPA and/or Settling Defendant has proposed modified Engineering Performance Standards, does the experience in Phase 1 and any other evidence before the panel show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2?

Both EPA and GE proposed changes to the EPS. Based on the Panel's review of EPA and GE's evaluations of Phase 1 and experience with environmental dredging, the Panel finds that it will not be practicable to consistently and simultaneously meet the EPS being proposed by either party for Phase 2. Phase 1 demonstrated that the 3 EPS interact in complex ways and that in order for the EPS to work individually and simultaneously, these interactions need to be better understood and addressed. Neither proposal provides a framework to generate the information needed to better understand these interactions and adapt the implementation of EPS so they can be met individually and simultaneously.

The first step toward achieving an integrated set of EPS would be to revise the Residuals EPS and Productivity EPS by better characterizing the DoC and creating an elevation-based design that would allow for a simplified decision process, less redredging, and the timely closure of CUs. EPA's proposal attempts to simplify the process but still relies too heavily on redredging and a complex decision process for closing CUs. In addition, EPA's proposed modifications would not provide the information needed to better understand PCB release mechanisms and the implications of productivity and residuals decisions on resuspension and implications for downstream risk to fish.

GE's proposed modifications to the EPS are based on an assertion that downstream loading is tied directly to dredging. Based on this, GE strongly recommends closing CUs with single-pass dredging in high-confidence areas, 2-pass dredging in low-confidence areas, and limiting the mass of PCBs dredged. The Panel finds that delayed closure of CUs was likely a major contributor to downstream loading and, thus, supports an approach that minimizes dredge passes and provides for quick CU closure. However, such an approach would need to be predicated on better characterization of the DoC and use of a target dredge elevation that takes into account the vertical accuracy of the dredge.

The Panel does not support placing an absolute limit on the mass of PCBs to be dredged, as proposed by GE, because the mass of PCBs to be removed is unknown and constraining the remedy to such a limit appears to be contrary to the ROD.

6.3 Summary of Response to Charge Question 3

CHARGE QUESTION 3. If the experience in Phase 1 and other evidence before the panel does not show that it will be practicable to consistently and simultaneously meet the Engineering Performance Standards that are being proposed for Phase 2, can the Phase 1 Engineering Performance Standards be modified so that they could consistently be met in Phase 2, and, if so, how?

Based on the Panel's review of Phase 1 evaluations and the Panel members' collective experience, the Panel finds that the Phase 1 EPS can be modified so that they could be consistently be met in Phase 2.

However, modifications to the EPS would not be enough to successfully complete the project; changes to the overall management of the project and its objectives would also be necessary.

In terms of objectives, the Panel recommends the following: focus must be placed on achieving rapid CU closure to minimize resuspension and release; productivity should be measured with regard to the remediated footprint (i.e., equal focus on the area remediated as well as inventory removed); and the decision to backfill or cap must be made and implemented more immediately based on the residual concentration of PCBs. These combined objectives could be achieved by: improved characterization of the DoC; using this information to establish Design Dredge Elevations that more accurately capture the target inventory; dredging the inventory based on updated Design Dredge Elevations, not residuals chemistry; and closing the CUs as quickly as possible.

Specifically, the Panel recommends the following framework for dredging and residuals management (see Section 4 for more detailed recommendations):

- Perform recoring of all low-confidence samples and recommends confirmation of 20 percent of high-confidence samples.
- Remodel the DoC using all high-confidence cores to establish the topography of the DoC (the "DoC Elevation") throughout each CU.
- Update the design with a Design Dredge Elevation based on the remodeled DoC Elevation.
- Set the Design Dredge Elevation initially to 4 inches below the modeled DoC Elevation to account for the vertical accuracy of the dredge.
- Establish BMPs to limit sediment resuspension and release.
- Perform confirmation sampling in each 1-acre sub-CU as soon as possible after attainment of the DoC Elevation in 95 percent or more of the area is confirmed by EPA.
- Place a 3-6 inch sand cover over sub-CU as soon as possible after confirmation samples are collected (before PCB analytical results are obtained).
- Use PCB analytical results of composited surface samples to determine whether an area will be backfilled or capped and install final layers accordingly.

6.4 Summary of Response to Charge Question 4

CHARGE QUESTION 4. If EPA and/or Settling Defendant has proposed modifications to the monitoring and sampling program for Phase 2, are the proposed modifications adequate and practicable for determining whether the Phase 2 Engineering Performance Standards will be met?

Both EPA and GE proposed changes to the EPS with concurrent changes to the monitoring and sampling program for Phase 2. However, the Panel finds that it will not be practicable to consistently and simultaneously meet the EPS being proposed by either party and, thus, cannot make a finding regarding the monitoring and sampling programs relative to these proposed standards except for those items that have been specifically addressed under Charge Question 2, above. Rather, the Panel has addressed Question 4 relative to the modified EPS and processes recommended by the Panel in response to Charge Question 3.

Achieving all 3 proposed EPS in Phase 2 consistently and simultaneously according to the proposed approach outlined herein will require a sampling and monitoring program that will provide accurate determination of the DoC for all CUs and post-removal composite sampling to determine whether the CU requires backfilling or a cap.

Further, the interaction of the dredge operations and release mechanisms is not well understood, and this issue is not sufficiently addressed in the current monitoring program. While to date there is insufficient information to demonstrate that transported PCB load outside the currently planned CUs in the Upper and Lower Hudson is causing increased PCB concentrations in bedded-sediment concentrations, the Panel believes that expected benefits of the removal action must be demonstrated in the off site areas. If significant increases are occurring that compromise the expected risk reductions, further changes to the removal program would need to occur. Sufficient monitoring must be conducted to assess whether such increases are occurring and provide the information necessary to effectively modify the removal program.

7 CONCLUDING REMARKS

Phase 1 showed that the 2004 EPS for Resuspension, Residuals, and Productivity were not met individually or simultaneously during Phase 1 and cannot be met under Phase 2 without substantive changes. EPA and GE proposed changes to the EPS but the Panel finds that the new proposed standards from either party would not contribute to the successful execution of Phase 2. However, Phase 2 can remove the bulk of the PCB inventory if coring data and the resulting DoC model results are improved and focus is placed on quick closure of CUs. The Panel developed an approach along with modified EPS to maximize removal of PCB inventory in a careful balance with resuspension and residuals goals, while achieving an acceptable level of productivity.

The Panel also recommends building upon the adaptive practices and approaches that have been employed to date by developing a more comprehensive and formalized adaptive management approach to all EPS that includes the annual reassessment of the EPS based on each prior year's data. The challenges encountered during Phase 1, and the adaptations employed by EPA and GE to address those challenges, demonstrate the need for flexibility during Phase 2. This was evidenced in the records of the management meetings to achieve CU closure during Phase 1, and especially by the commitment to this Peer Review process, seeking to refine and improve the EPS and in-field practices. During Year 1 of Phase 2, the Panel recommends collecting additional data to support the further refinement of relevant performance standards to be applied for the remainder of the project's duration. Additional review between Years 1 and 2 of Phase 2, and each subsequent year of the project, should allow for ongoing modification of the EPS to optimize remedial operations, while limiting unintended consequences and adverse environmental impacts from these operations.

Phase 1 demonstrated that the Residuals EPS had a substantial impact on the operational success of the project as well as a tangible interaction with productivity and resuspension processes and their respective EPS. A key obstacle to simultaneously achieving the performance standards involved incomplete, inaccurate, and imprecise DoC characterization combined with disagreement on how to interpret and attain target levels. This directly affected both the Resuspension EPS and Productivity EPS. The repeated dredge passes and prolonged exposure of sediments in the CUs resulted in increased PCB resuspension and release. The unexpected increase in inventory due to incomplete DoC characterization had the greatest effect on the Productivity EPS in terms of numbers of CUs remediated. The Panel presents revised EPS that accelerate CU closure by establishing an elevation-focused dredge design paradigm, thereby reducing resuspension, effectively managing residuals, and accelerating productivity without compromising the goals of the ROD with respect to overall recovery of the river.

The Panel proposes an elevation-focused design of the dredge prism design that builds on accurate, high-precision characterization of the DoC elevation, a 4-inch overdredge based on vertical tolerance of the dredge and precision of the DoC that ensures rapid achievement of the target elevation (the elevation of the DoC not including the overdredge) across at least 95 percent of the CU area or subunit area, verification of the target elevation based on high-precision bathymetry, and rapid closure of CU or subunit areas following EPA validation of confirmed elevations

This approach does not involve redredging to remove dredge-generated residuals or address redefined inventory based on post-dredge confirmation sampling. The CU would be closed based on the results of the residuals sampling results. The CU (or sub-CU) should be backfilled if the average residuals

concentration is less than or equal to 3 mg/kg Tri+PCBs and capped if the average residuals concentration is greater than 3 mg/kg Tri+PCBs. The backfill or cap eliminates the risk from any residual PCBs in the sediments.

This revised removal and closure approach is the first step toward integrating the Residuals, Resuspension, and Productivity EPS. Through better characterization of the DoC and establishing an elevation-based dredging prism design, Resuspension and Productivity EPS also can be revised to be consistent with the updated dredge depths and volumes. For Year 1 of Phase 2, the Panel proposes Resuspension EPS and Productivity EPS based on metrics consistent with Phase 1: for resuspension, target levels are 2 percent and 1 percent of the dredged PCB mass, measured at TIP and Waterford, respectively; for productivity, target volumes are 350,000 CY per year. Both of these targets (i.e., for resuspension and productivity) should help guide BMPs, but should not lead to shutting down operations. In other words, the Panel does not recommend interrupting dredging activities if the targets are not achieved during Year 1 of Phase 2; the goal of the interim standards is to establish baseline targets during Year 1 of Phase 2 and to allow dredging to recommence in 2011, while near-field and far-field data are collected.

Based on the results of Year 1 of Phase 2, combined with the Phase 1 results, EPA and GE should refine the performance criteria to establish practicable targets that can be achieved for all 3 EPS. In addition to evaluating the performance of the modified Residuals EPS, the focus between Years 1 and 2 of Phase 2 should be the Resuspension EPS to manage near-field and far-field resuspension, release, and deposition processes, based on an understanding of whether there are increased risks associated with surface sediment deposits containing PCBs released during dredging. The Productivity EPS should also be updated based on a revised volume estimate derived from the elevation-based dredging paradigm. In addition to an annual volume productivity standard, the Panel advances an additional EPS metric; annual areas to be remediated. Area remediated reflects a substantial measure of environmental benefit and could be expressed as a specified number of CUs to close each year. Tracking of total volume and mass of PCBs removed should continue, but the environmental benefit accrued should be based both on mass removal and on area remediated. Eventually, an area-based standard could supplant the volume-based productivity standard, if appropriately tied to the elevation-based design.

The Panel found that the models used to develop the 2004 Resuspension EPS cannot be used to adapt revised standards for moving forward. The Panel believes that to do so requires a new model that must be developed collectively by EPA and GE. The GE model may be a useful foundation for this model, and both model structure and parameters must be agreed upon by both EPA and GE. The model must be peer reviewed by an expert panel once EPA and GE complete its development. Similar arrangements have been established at other Superfund Sites, including the Passaic River, the Lower Duwamish Waterway (WA), and the Lower Willamette River (OR). The fate, transport, and risk model must enable EPA and GE to understand the implications of operational changes on long-term recovery rates to support EPA and GE in making to appropriate and meaningful risk management decisions about dredging productivity, BMPs, and the long-term fate and transport of PCB residuals and resuspension/ release.

The Panel evaluated the results from Phase 1 in order to assess a practicable annual production rate. The evaluation included a detailed review of peak monthly output for each component of the remedial

action (i.e., dredging, processing, transportation), dredging and removal output (i.e., numbers and cycle times for dredges and barges), and shipping output to the landfill. The Panel did not discover any single factor that could be adjusted to significantly increase overall productivity. For example, neither increasing the number of barges in service nor increasing the offload rate at the processing facility provided a substantive increase in productivity. Rather, the Panel found multiple lines of evidence supporting 350,000 cy/yr as a reasonable annual productivity estimate for the start of Phase 2. The Panel also found that the productivity schedule should be subordinated to the Resuspension EPS and Residuals EPS. Consequently the 5-year productivity criterion should be dropped to provide more flexibility to complete the work in a manner that protects the integrity of the project and its risk reduction objectives.

8 REFERENCES

Bridges, T. S., S. Ells, D. F. Hayes, D. Mount, S. C. Nadeau, M. R. Palermo, C. Patmont, and P. R. Schroeder. 2008. "The Four Rs of Environmental Dredging: Resuspension, Release, Residual, and Risk," Technical Report ERDC/EL TR-08-4, U.S. Army Engineer Research and Development Center, Vicksburg, MS.

Connolly, J., V. Chang, and L. McShea. 2006. Grasse River Remedial Options Pilot Study (ROPS) Findings from Dredging Activities. Presentation at the Second Meeting on Sediment Dredging at Superfund Megasites, June 7, 2006, Irvine, CA.

Hayes, D. and P. Wu. 2001. Simple approach to TSS source strength estimates. Proceedings, 21st Annual Meeting of the Western Dredging Association (WEDA XXI) and 33d Annual Texas A&M Dredging Seminar, Houston, TX.

Palermo, M. R., P. R. Schroeder, T. J. Estes, and N. R. Francingues. 2008. "Technical Guidelines for Environmental Dredging of Contaminated Sediments," Technical Report ERDC/EL TR-08-29, U.S. Army Engineer Research and Development Center, Vicksburg, MS.

Patmont, C. and M. Palermo. 2007. Case studies of environmental dredging residuals and management implications. Proceedings, 4th International Conference on Remediation of Contaminated Sediments, January 22-25, Savannah, GA.

Pennekamp, J. G. S., R. J. C. Eskamp, W. F. Rosenbrand, A. Mullie, G. L. Wessel, T. Arts, and I. K. Decibel. 1996. Turbidity caused by dredging; viewed in perspective. Terra et Aqua, 64:10-17.

Steuer, J. J. 2000. A mass-balance approach for assessing PCB movement during remediation of a PCB-contaminated deposit on the Fox River, Wisconsin. U.S. Geological Survey Water-Resources Investigations Report 00-4245. December 2000.

APPENDIX A: DOCUMENTS PROVIDED TO THE PEER REVIEW PANEL

Background Documents Provided January 2010

From EPA

- *Transmittal of Phase 2 Dredge Area Delineation Report*, December 17, 2007, from John Haggard, General Electric to Doug Garbarini, EPA Region 2
- Hudson River PCBs Site Phase 2 Dredge Area Delineation Report, Figures, and Appendices, December 17, 2007
- Report on the Peer Review of the U.S. Environmental Protection Agency's "Draft Engineering Performance Standards—Peer Review Copy" for the Hudson River PCBs Superfund Site, March 4, 2004.
- Appendices: Report on the Peer Review of the U.S. Environmental Protection Agency's "Draft Engineering Performance Standards—Peer Review Copy" for the Hudson River PCBs Superfund Site. March 4, 2004.
- Volume 1. Statement of the Engineering Performance Standards for Dredging, April 2004.

From General Electric

- Archeological Reports
  - NYSDEC boat ramp (area L) testing report
  - Phase 1 ARA report
  - Terrestrial survey report
  - Terrestrial testing report
  - Underwater resource testing report
  - Underwater survey report appendices
  - Underwater survey report
  - Work support marina survey report
  - Work support marina testing report
  - WSM data recovery report
- Consent Decree
  - Appendices B-D
  - Modification transmittal letter, Attachment A, and Consent Decree Mod
- Performance Standard Documents
  - Hudson River Quality of Life Performance Standards (full report). May 2004
  - Engineering Performance Standards: Volumes 1-5
- Phase 1 Design (this folder contains multiple files related to design and construction specifications)

- Phase 1 Contract Documents
    1. Contract 1: Facility site work construction
    2. Contract 2: Rail Yard Construction
    3. Contract 3A: Processing facility construction
    4. Contract 3B: Processing facility operations
    5. Contract 4: Dredging Operations
    6. Contract 5: Habitat Construction
    7. Contract 6: Rail Yard Operations
- Phase 1 Dredge Area Delineation Report. Prepared for GE by Anchor QEA. February 28, 2005. (Includes tables, figures and appendices)
- Phase 1 Final Design Report. March 21, 2006 (includes attachments A-K, figures and tables).

- Phase 2 Intermediate Design Report
    - Text of Phase 2 Intermediate Design Report
    - Logistics model output data
    - Phase 2 shoreline photos
    - Phase 2 IDR approval and response to comments
    - Appendix 1 drawings
    - Appendix 2 specifications
    - Attachments A-M
    - Tables and Figures
- Quality Assurance Project Plans
    - 5/28/2005 BMP QAPP text
    - 5/28/2005 BMP QAPP appendices
    - 5/28/2005 BMP QAPP figures
    - 5/28/2005 BMP QAPP tables
    - RAM QAPP appendices
    - RAM QAPP final 5/12/2009 tables and figures
    - RAM QAPP final 5/12/2009 text only document
- Remedial Action Work Plans
- 2002 ROD and responsiveness summary
- Phase 1 Data Compilation Hudson River PCBs Superfund Site. Prepared for GE by Anchor QEA. November 2009
- Appendices to Phase 1 Data Compilation Report.
- Supplement to Phase 1 Data Compilation Hudson River PCBs Superfund Site. Prepared for GE by Anchor QEA. January 2010.
- Appendices to Supplement to Phase 1 Data Compilation Hudson River PCBs Superfund Site.

Items Provided Independent of Panel Requests following February 17 – 18 Introductory Session

- Hard copy of GE Phase 1 Evaluation Report, Tables, and Figures and CD of Appendices (provided March 10, 2010)
- Hard copy of EPA Phase 1 Evaluation Report, Tables, Figures, and Appendices and CD of same (March 16, 2010)
- EPA Oversight Team Phase 1 Observations Report (sent via email March 24, 2010)
- Addendum to EPA Phase 1 Evaluation Report (sent via email to the Panel May 2, 2010, and provided in hard copy at Peer Review Panel Meeting, May 4, 2010)

Items Provided in Response to Panel Supplemental Information Requests following February 17 – 18 Introductory Session

(submitted to EPA March 2, 2010 and forwarded by EPA to GE March 10, 2010)

- March 26, 2010 GE Initial Response to Panel Supplemental Information Requests from Introductory Session (provided to the Panel March 30), including:
  - CD with maps showing bucket prints from each pass
  - Table showing number of closed buckets per pass, per CU
  - Directions on finding simple CU coring maps within GE Data Compilation Report and GE Final Phase 1 Evaluation Report
  - Directions to locating productivity dredge data in Appendix D of GE's Final Phase 1 Evaluation Report
  - Directions to finding pre-RA cores and grabs collected during the design phase in Appendix R of GE's Final Phase 1 Evaluation Report
  - A CD file for Residual data, including sample descriptions for each residual sample that was analyzed for PCBs
  - 4 DVDs with electronic dredge pack data, including the software drivers needed to view the data
- March 29, 2010 EPA Response letter to Panel Information Requests following Introductory Session
- April 8, 2010 GE Additional Response to Panel Supplemental Information Requests and Questions Received after the Introductory Session. This response included the following additional items
  - Hudson River Dredging Project Phase 1 Summer 2009 (DVD)
  - Operation, Maintenance, and Monitoring Plan for Phase 1 Caps and Habitat Replacement/Reconstruction, March 4, 2010 (CD)
- April 21, 2010 GE Response to Supplemental Panel requests (submitted to EPA April 14, 2010 and forwarded by EPA to GE April 19, 2010). Includes:
  - Response to request for raw field logs (directions provided to located information in SSAP database in Appendix R of GE Phase 1 Evaluation Report)

  - Dredging elutriate test (DRET) data (directions and link provided Treatability Studies Report which was one of separately bound appendices of the Phase 1 Intermediate Design Report submitted to EPA August 22, 2005)
  - Phase 1 Dredging daily summaries (instruction to locating information in November 2009 GE Data Compilation Report and January 2010 Supplement
  - Dredging contractor's daily reports and weekly reports (summarized in Appendices A can DC of January 2010 Supplement)
  - CD of GE's Weekly Productivity Summary Reports
  - Construction management contractor's barge reports (Appendix P of Data Compilation Report and Appendix P and January 2010 Supplement contain PDFs of scanned barge reports
  - Additional GE Monthly RA Reports/Monthly Progress Reports (Appendix F of Data Compilation Report contains monthly RA reports for May – September 2009)
- Public comments on EPA and GE Phase 1 Evaluation Reports (CDs received from EPA April 28 and provided to the Panel on April 29)
- April 29, 2010 GE Response to Supplemental Panel requests (remaining request from March 2 and additional requests from April 23), including:
  - summary of GE's proposed changes to Engineering Performance Standards
  - GE cost data for Phase 1
  - GIS shapefiles of the study area
  - Attachment A –Technical Memorandum Allowable Load Calculations for Hudson River Dredging Project
  - Attachment B – Repost on PCB Expenditures 1990 – 2009
  - Attachment C – An Overview of the Upper Hudson River PCB Modeling System
  - Attachment D – Relevance of EPA's Contaminated Sediment Remediation Guidance to the Engineering Performance Standards
  - Attachment E – 2010 High Flow Event Technical Memorandum

Information Provide to the Panel in Response to Information Requests following the May 4-6 Peer Review Panel Meeting

- Written public comments submitted at May 4-6 Peer Review Panel meeting (sent via email 5/11/)
- EPA response to 5/11 Panel request for updated table of proposed modifications to the resuspension standard (received from EPA 5/18 and provided to the Panel 5/18)
- CD with public comments on EPA Report Addendum and materials presented at May 4-6 Peer Review Panel meeting, including GE's comments on same (provided to the Panel 5/19)
- GE response to 5/12 Panel request for table of estimated Tri+PCB mass for all Phase 2 CUs (received from GE 5/19 and provided to the Panel 5/19)

- GE response to 5/25 Panel request for modified table of estimated Tri+PCB mass for all Phase 2 CUs to include average existing surface sediment PCB concentrations per CU and design inventory volume per CU (received from GE 5/26 and provided to Panel 5/26)
- EPA response to 5/18 Panel request for clarification on "new model analysis" and "a model simulation of the Upper Hudson" referred to in EPA's proposed modifications to the resuspension standard table provided on 5/18 (received from EPA 6/2 and provided to the Panel 6/2)
- GE response to 6/1 Panel request for maps and excel spreadsheet that identify surface sediment PCB concentrations in areas not targeted for dredging, upstream of Waterford and figures for Section 5 of Phase 2 DAD (GE replied with letter and accompanying CD dated 6/2 and provided to the Panel 6/3)
- GE response to 6/2 Panel request for clarification of estimate of the total PCBs to be removed for the duration of the project (received from GE 6/3 and provided to the Panel 6/3)
- EPA response to 6/3 Panel request for:
  - Pre-Phase 1 Surface Weighted Average Concentration by CU, and then for the entire Thompson Island Pool
  - Post-Phase 1 Surface Weighted Average Concentration by CU, and then for the entire Thompson Island Pool
  - Post-completion of all the CUs in the TIP (a) estimate of the SWAC by CUs, and for the entire TIP
  - Pre-Phase 2 SWAC for the Rest of River by CU and then by Segment
  - Post Phase 2 SWAC for the Rest of River by CU and then by Segment
  - Estimate of the Phase 1 SWAC after completion of each individual pass

  (received from EPA 6/9 and provided to the Panel 6/10)
- EPA response to 6/2 Panel request for clarification showing how EPA derived the PCB mass to be removed for the remainder of the project, and how EPA derived the Control Level of Tri+PCB of 670 kg (received from EPA 6/11 and provided to the Panel 6/11)
- GE Response to 6/3 Panel request to provide the GIS layers associated with Figures 6-2a-s and 6-3a-s from the April 29, 2010 Upper Hudson Model Technical Memorandum (received letter and CD containing GIS surface PCB concentration figures and Phase 2 Dredge Area Delineation Chapter 5 figures from GE 6/7 and provided to the Panel 6/7)
- GE Response to 6/23 Panel request to provide information on how much material has been sent to the landfill and whether the landfill problems have been resolved (received response from GE 6/25 and provided to the Panel 6/15)

Attachment T

Letter from Dr. Robert Haddad, NOAA, to Robert Sussman, EPA

U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
National Ocean Service
Office of Response and Restoration
Silver Spring, Maryland 20910

December 2, 2010

Via Email and Fed Ex

Robert Sussman
Senior Policy Advisor
U.S. Environmental Protection Agency
Ariel Rios Building, Mail Code 1101A
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Re: Phase 2 Remediation, Hudson River PCB Superfund Site

Dear Mr. Sussman:

The National Oceanic and Atmospheric Administration's Office of Response and Restoration (NOAA OR&R), on behalf of the Department of Commerce, thanks you for meeting with us on November 30, 2010 to discuss our recommendations regarding the Hudson River PCB Superfund Site Phase 2 remediation. The intent of this letter is to reiterate the points that Tom Brosnan and Lisa Rosman of my staff raised on Tuesday and to provide you with the documentation supporting those points.

NOAA, in its natural resource trustee capacity, works to protect and restore coastal resources from threats related to releases of hazardous substances and oil spills. NOAA has a long history of working with the US Environmental Protection Agency (EPA) to maximize the cleanup and ultimate restoration of the Hudson River PCB Site.

As relayed previously, NOAA has significant concerns regarding the scope and design of the Phase 2 remedy that EPA will present to GE for its opt in/opt out decision, and the condition of the river that will be left behind for the natural resource trustees to restore. Our analyses indicate that:

- Average surface PCB contamination in River Sections 2 and 3 are 5-10 times greater and sediment natural recovery is much slower (verging on negligible) than what was believed when the ROD was originally issued in 2002 (Figure 1).

- Average surface (maximum in top 12 inches) PCB concentrations in River Sections 1 and 2 are equally elevated (>100 ppm total PCBs), but the surface clean up trigger in River Sections 2 and 3 (~90 ppm) is about 3 times higher than in River Section 1 (~30 ppm total PCBs).

- Given these facts, following remedy implementation, approximately 5 times higher concentrations of bioavailable PCBs will be left behind in surface sediments in River Sections 2 and 3 than the ROD envisioned in 2002 (Figure 2). This should translate into a proportional

increase in fish tissue PCBs and a delay in the projected recovery of the river and the natural resources services that the river supports. There is no evidence to suggest that Hudson River fish will behave differently from the theoretical response.

- To our knowledge, none of these points are disputed by EPA. NOAA suggests that EPA conduct an analysis on the impacts of these findings on changes to risk to fish, wildlife or humans relative to the risks originally projected by the 2002 remedy, and an evaluation of the potential need for a change in the scope of the remedy. GE's untested model seems to be the only basis for EPA to believe that fish concentrations will achieve target levels in the time frame envisioned by the ROD, rather than a more protracted time frame.

- These concerns are further compounded by the proposed one pass approach to dredging that has the potential to leave dredgeable inventory in-place and surface PCBs above 1 ppm Tri+ PCBs because remediation would be to a prescribed elevation. The one pass approach could result in substantial and unnecessary capping of the river bottom, much more than envisioned by the ROD and the Engineering Performance Standards since depth of contamination is not adequately characterized and the overcut is insufficient to address depth of contamination uncertainties.

- From NOAA's perspective, EPA should continue to minimize the amount of capping allowed consistent with the 2004 Engineering Performance Standards.

- Finally, significant problems encountered during Phase 1 habitat reconstruction led to unsuccessful habitat mitigation. Many of these problems have not been adequately addressed in the Phase 2 design to ensure effective reconstruction of high quality, sustainable and resilient habitat.

The impacts of maintaining the current course of action is clear and troubling to NOAA:

- A series of Superfund-caliber sites will be left behind due to the level and extent of unremediated surface sediment PCBs;

- These elevated post-construction concentrations are often adjacent to the cut lines. This will result in the high likelihood of remediated areas becoming recontaminated;

- Restoration with the appropriate nexus to the locations of the ecological injuries as directed by the NRDA process will not be feasible due to the remaining contamination and projects may need to be relocated further from the site of injury;

- Recovery of the Hudson River will be further delayed, due to remaining PCBs and the improper and insufficient habitat reconstruction, resulting in a loss of ecosystem productivity;

- This will set a national precedent as the Hudson River remediation is being closely watched by PRPs, EPA, trustees and NGOs nationwide. This is a precedent NOAA doesn't want repeated.

NOAA urges EPA to seek to achieve the original risk-based goals of the ROD, by trying to achieve surface concentrations closer to what the ROD envisioned. This can be accomplished by applying River Section 1 surface criteria to River Sections 2 and 3. Most of the highly contaminated surface sediment remaining in River Sections 2 and 3 after Phase 2 remediation is in close proximity to Phase 2 dredge

prisms. NOAA strongly recommends additional removal of highly contaminated sediments especially within 100 to 200 feet of the dredge lines (Figure 3; Figures distributed at meeting not transmitted). This enhancement to the design would result in the additional removal of 80-100 acres (15-18 % increase in sediment volume) (Tables 1 and 2) and is within the volume of sediment removal envisioned in the original ROD. Our recommendation of a uniform surface criterion across all three River Sections would significantly reduce bioavailable PCBs.

NOAA also wants to ensure that EPA realizes that the Trustees plan to communicate our analysis of the post-remediation surface sediment PCBs to the public shortly so that they understand the challenges the Trustees will face in identifying and implementing in-kind/in-place restoration in the Upper Hudson due to much more bioavailable sediment PCB contamination left behind than we consider acceptable.

NOAA thanks EPA for the opportunity to express these concerns and would be happy to present these concerns to other EPA Senior Leadership, if so desired. Finally, NOAA urges EPA to seriously evaluate the merits of our proposal to expand the dredge prism boundaries to remove high concentrations in adjacent sediments and to improve restoration of habitats impacted by the remedy.

Sincerely,

Robert Haddad, Ph.D.,
Chief, Assessment & Restoration Division
Office of Response and Restoration
National Oceanic & Atmospheric Administration

Cc: Judith Enck, EPA
Walter Mugdan, EPA
Eric Schaaf, EPA
Paul Simon, EPA
Mathy Stanislaus, EPA
Jim Woolford, EPA
Betsy Sutherland, EPA
Craig O'Connor, NOAA
Tom Brosnan, NOAA
Lisa Rosman, NOAA
Bob Foley, USFWS
Wendi Weber, USFWS
Brian Donahue, USDOJ
Peter Kautsky, USDOJ
Stuart Gruskin, NYSDEC
Alison Crocker, NYSDEC
Kevin Farrar, NYSDEC
Andy Gugliemli, NYSDEC
John Davis, NYS AG
Eugene Leff, NYS AG

Figure 1. A comparison of estimated pre-remediation surface (top 2 inches) Tri+ PCBs. Blue bars are modeled concentrations reported in the Feasibility Study and used in selection of the remedy. Red bars are post-ROD data collected for the design. This illustrates that average surface PCB contamination in River Sections 2 and 3 are 5-10x greater than what was believed when the ROD was originally issued in 2002. (Total PCBs = ~3x Tri+ PCBs)

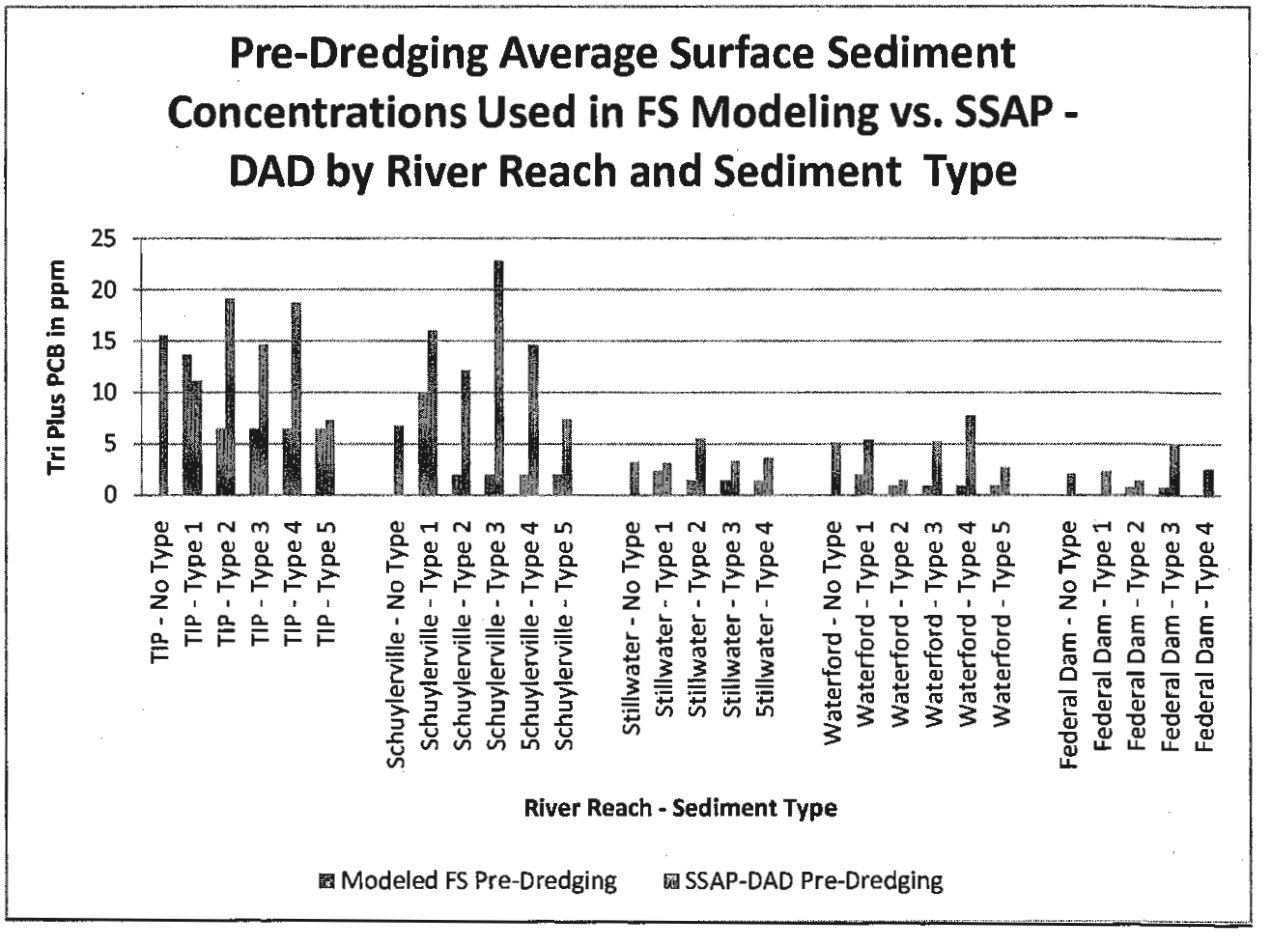

Figure 2. A comparison of estimated post-remediation surface (top 2 inches) Tri+ PCBs. Blue bars are modeled concentrations reported in the Feasibility Study and used in selection of the remedy. Red bar are based upon post-ROD design data. This illustrates that after the remedy is implemented. approximately 5x higher concentrations of bioavailable PCBs will be left behind in surface sediments in River Sections 2 and 3 than was envisioned in the 2002 ROD. (Total PCBs = ~3x Tri+ PCBs)

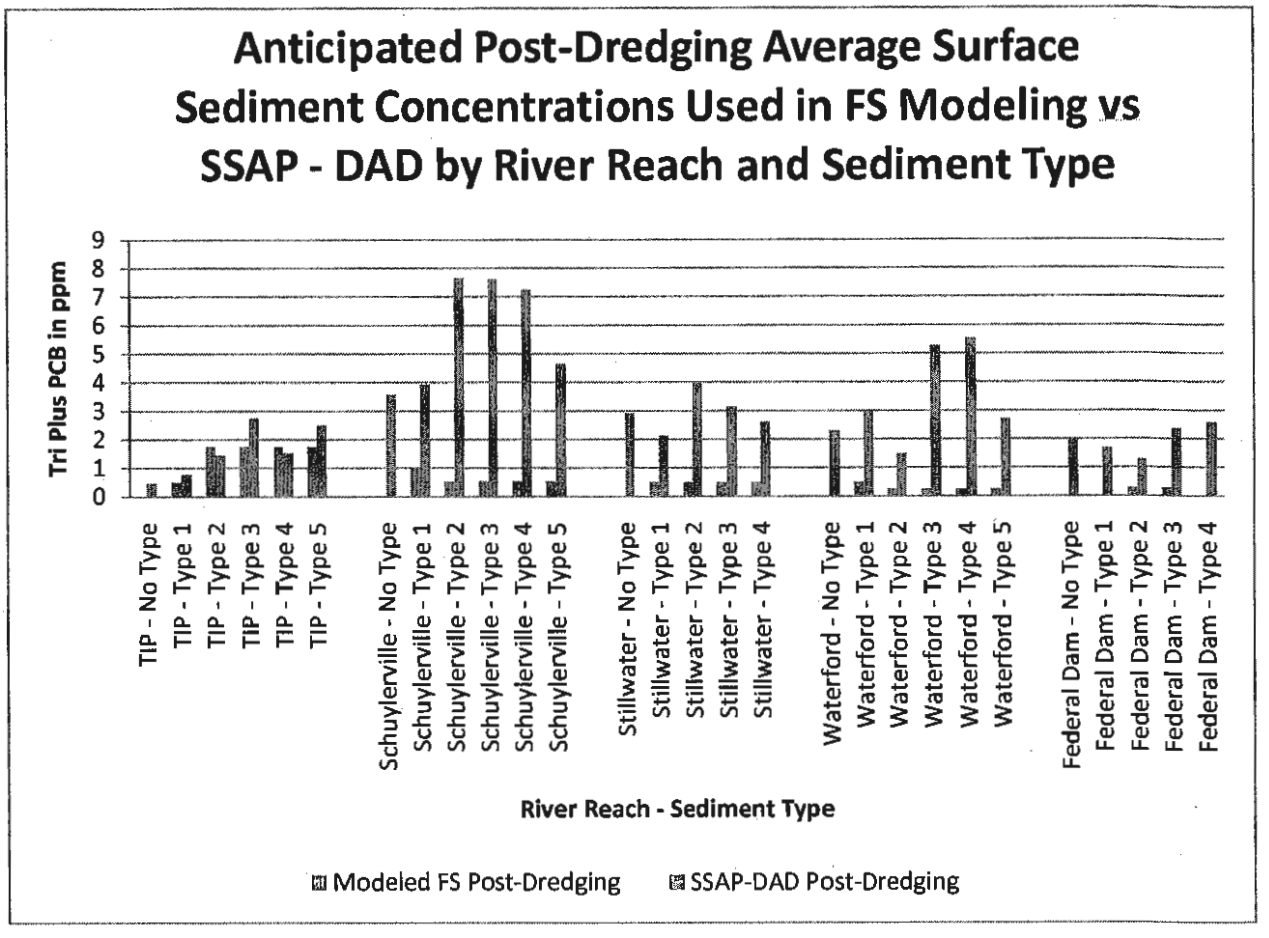

Figure 3. Additional reductions in estimated surface (top 2 inches) Tri+ PCBs achieved with additional removal in River Sections 2 and 3 through the application of the River Section 1 surface sediment cleanup trigger. Red bars indicate no addition removal from current Phase 2 design. Blue and green bars represent River Section 1 criteria within a 100 ft and 200 ft buffer respectively, of the existing dredge prism. The black bar applied River Section 1 surface trigger to River Sections 2 and 3. (Total PCBs = ~3x Tri+PCBs)

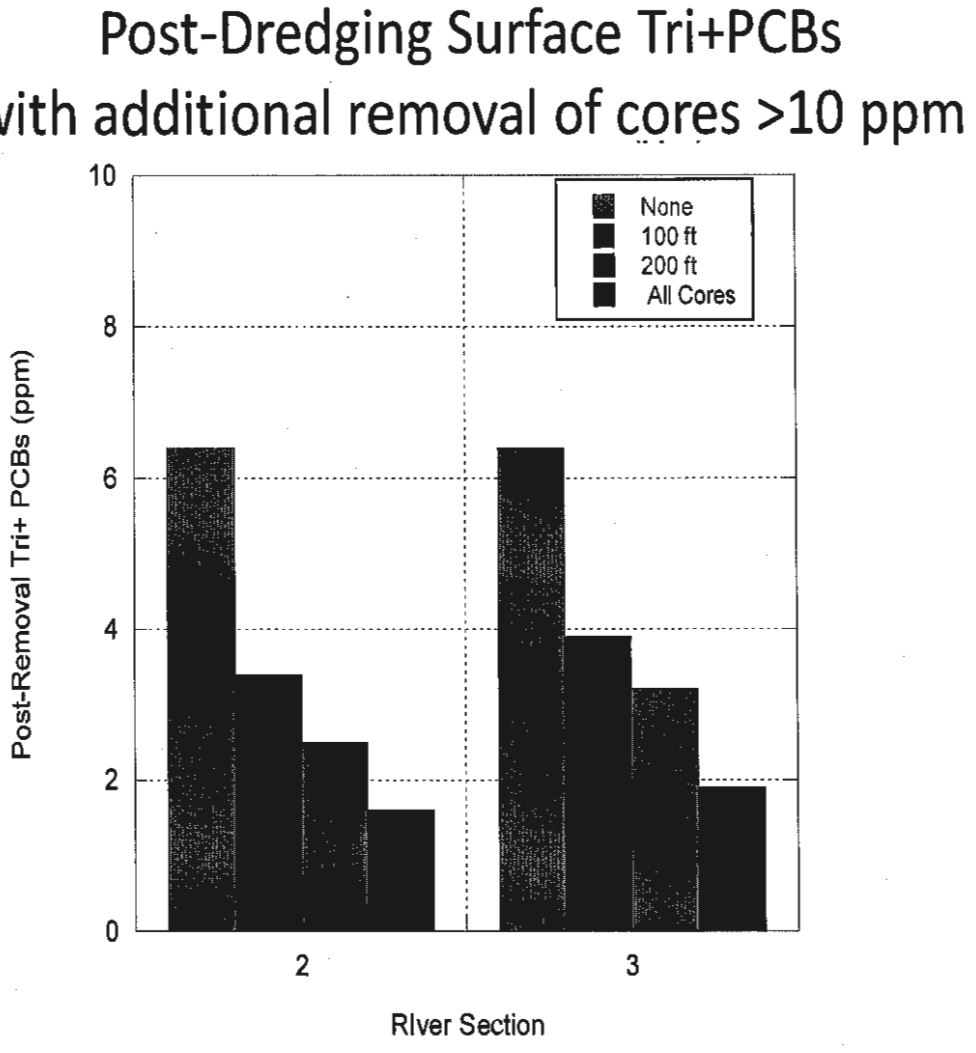

Table 1. Estimated number of acres based on distance beyond dredge footprint and estimated post-remedial surface Tri+ PCB concentrations.

River Section	Total Number of Acres with Surface Tri+ PCB >10 ppm			Estimated Tri+ PCB (ppm) in Surface Following Additional Removal of cores with Surface Tri+ PCB >10 ppm			
	Within 100 ft of dredge prism	Within 200 ft of dredge prism	Removing All Cores Outside Dredge Prism	Within 100 ft of dredge prism	Within 200 ft of dredge prism	Removing All Cores Outside Dredge Prism	No Additional Removal
RS2	29.0	37.0	44.9	3.4	2.5	1.6	6.4
RS3	51.4	62.1	91.0	3.9	3.2	1.9	6.4

Note: Basis for the acreage estimate: one core=1/8 acre from Garvey personal communication 2010. Surface definition is consistent with EPA July 26, 2004 Final Decision DAD Dispute.

Table 2. Volume estimates for different enhanced removal scenarios.

Estimated PCB Post Remediation	Estimated Area (acres) RS2+RS3	Estimated Additional Removal Volume (1000 cy)				
		Assuming 1ft DoC	Assuming 2ft DoC	Assuming 3ft DoC	Assuming 4ft DoC	Assuming 5ft DoC
All Cores within 100 ft >10ppmTri+	80	129	258	387	516	645
All Cores within 200 ft >10ppmTri+	99	160	319	479	639	799
All cores >10ppmTri+	136	219	439	658	878	1097

Attachment U

New York State Attorney
General Letter
Sept. 16, 2016

STATE OF NEW YORK
OFFICE OF THE ATTORNEY GENERAL

ERIC T. SCHNEIDERMAN
ATTORNEY GENERAL

DIVISION OF SOCIAL JUSTICE
ENVIRONMENTAL PROTECTION BUREAU

September 16, 2016

By Electronic Mail

Judith Enck, Regional Administrator
United States Environmental Protection Agency
290 Broadway
New York, New York 10007-1866

Walter Mugdan, Director
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United States Environmental Protection Agency
290 Broadway
New York, New York 10007-1866

Re: *Hudson River Superfund Site: EPA's Five Year Review and Certificate of Completion of Remedial Action*

Dear Administrator Enck and Mr. Mugdan:

Please accept this letter on behalf of the New York Attorney General s Office as a part of our ongoing dialogue with EPA regarding the Hudson River Superfund Site. We believe that additional steps are necessary to assure that the remedial action objectives set forth in EPA s 2002 Record of Decision (ROD) are timely met and that the remedy is fully protective of human health and the environment. Completion of those steps is necessary before EPA issues a certificate of completion of the remedial action to GE pursuant to 42 U.S.C. 9622(f)(3) and the November 2006 Consent Decree between EPA and GE.

EPA s issuance of the certificate of completion must comply with the statutory requirements of CERCLA Section 122(f), 42 U.S.C. 9622(f), insofar as it will give rise to a covenant not to sue and a release from liability for GE. EPA should not predicate the certificate of completion solely upon completion of the technical engineering performance tasks undertaken pursuant to the Consent Decree. Rather, it should be issued only upon completion of the remedial action in accordance with CERCLA and only upon EPA s finding after a comprehensive review that the remedy is protective of human health and the environment, as contemplated by the ROD. The covenant that arises upon issuance of the certificate of completion is in direct conflict with CERCLA s intent absent compliance with the remedial action objectives in the ROD and completion of the remedy contemplated, and absent a fully

supported finding that the remedy is protective of human health and the environment. 42 U.S.C. 9622(f).

Accordingly, we request that EPA, at a minimum, ensure that the remedial action objectives of the ROD and the requirements of CERCLA are met by taking the following actions:

(1) Defer issuance of a certificate of completion of the remedial action until EPA finds that the remedy is completed and is fully protective of human health and the environment in compliance with 42 U.S.C. 9622(f)(3), (5);

(2) Determine with reasonable certainty how long it will take for the PCB concentrations in fish to drop to the levels necessary to achieve those objectives and to enable lifting human consumption health advisories for PCBs in all contaminated River reaches for all affected species;

(3) Undertake a comprehensive fish consumption survey along all contaminated reaches of the River, from Hudson Falls to the Battery, to quantify present and future human exposure to PCBs from the consumption of contaminated fish; and

(4) Define in writing the scope and objectives of the Five Year Review and the participation and respective roles of the review team members, including non-EPA members.

These issues are discussed in detail below.

(1) Defer Issuance of a Certificate of Completion of the Remedial Action until EPA Finds that the Remedy is Protective of Human Health and the Environment in Compliance with 42 U.S.C. § 9622(f)

We understand that EPA is considering issuing to GE a certificate of completion of the remedial action, notwithstanding evidence establishing the ineffectiveness of the remedy in achieving the ROD s remedial action objectives, and despite EPA s on-going Five Year Review to determine the effectiveness of the remedial action. It is inconsistent with the requirements of CERCLA Section 122(f)(3) to issue the certificate of completion without finding that the ROD s remedial action objectives have been achieved and that the remedial work necessary to achieve those objectives is complete. 42 U.S.C. 9622(f)(3). That provision prohibits a covenant not to sue from taking effect unless EPA certifies that the remedial action has been completed in accordance with the requirements of this chapter at the facility that is the subject of such covenant. 42 U.S.C. 9622(f)(3). The question presented here is whether EPA can certify that the Hudson River remedial action has been completed in accordance with the requirements of CERCLA.

EPA s issuance of a certificate of completion is contrary to CERCLA s statutory scheme in the absence of a finding that the remedial action objectives of the ROD have been achieved and the remedy is protective of human health and the environment. For the Hudson River, the preeminent remedial action objective in the ROD is the reduction of the concentration of PCBs in fish within specific time-frames. *See* ROD, p. 73. It is now clear that the ROD s express remedial action objective to reach 0.4 mg/kg of PCBs in fish by 2016 has not been achieved. Based on the most recent data available, the 2014 PCB fish concentration is 2.71 mg/kg, which is more than 600% greater than the remedial objective of 0.4 mg/kg for 2016. No reasonable observer expects the upcoming 2015 or 2016 data to demonstrate that the 0.4 mg/kg concentration has now been achieved. The ROD s objective of achieving more dramatic reductions (0.05 mg/kg) later also is questionable, as EPA recognized in its 2012 Five-Year Review. *See* First Five Year Review Report for Hudson River PCBs Superfund Site, p. 34 (2012).

In addition to the ROD s remedial action objective for PCB fish concentrations not being met, EPA has failed to provide a revised projected time-frame by which they will be met. EPA has not publicly amended the ROD or explained the significant difference between its objectives and the current status of PCB concentrations in fish. Because EPA cannot conclude that the ROD s remedial action objectives have been met, it cannot conclude that the remedial action has been completed in accordance with CERCLA s requirements. 42 U.S.C. 122(f)(3). Consequently, a certification of completion and the associated covenant not to sue would be improper.[1]

Furthermore, a certificate of completion is premature before EPA completes its Five Year Review, which is presently underway. If EPA finds that the remedy is not protective (or if it improperly defers a protectiveness finding because of the absence of data showing declining PCBs in fish),[2] EPA may have limited recourse against GE once the certificate is issued. Indeed, EPA s finding that the remedial action has not met the ROD s standard of protective would be

[1] Unlike numerous other consent decrees for Superfund sites, the 2006 Hudson River Consent Decree does not specify that certification of the remedial action shall be issued in compliance with CERCLA Section 122(f), 42 U.S.C. 9622(f)(3). *See, e.g., U.S. v. Atlantic Richfield Co.*, CV-83-317-HLN-SHE (D. Montana, 2008), Consent Decree, Clark Fork River Operable Unit (p. 16) (Certification of Completion of the Remedial Action shall mean EPA s certification, in consultation with the State, pursuant to Section 122(f)(3) of CERCLA, 42 U.S.C. 9622(f)(3), that the Remedial Action . . . have been completed . . . in accordance with the requirements of CERCLA, the NCP, and the ROD . . . including certification that Performance Standards have been attained.); *U.S. v. NCR Corp., et. al.,* CV-10-C-910 (E.D.Wisc. 2010), Notice of Lodging of Consent Decree (p. 14) (. . . these covenants shall take effect upon certification of completion of the remedial action by EPA pursuant to 42 U.S.C. 9622(f)(3)). The 2006 Hudson River Consent Decree, however, must be read as incorporating that statutory requirement.

[2] EPA s guidance indicates that deferral of a protectiveness finding in the Hudson River s Five Year Review is not appropriate because exposure pathways are well-known, no new exposure pathways have been identified, no new contaminants have been identified, and an ecological risk assessment has been done. *See* OSWER Memo 9200.2-111: Clarifying the Use of Protectiveness Determinations for CERCLA Five-Year Reviews, p. 4 (Sept. 13, 2012).

well-supported because there continues to be human exposure to PCBs from fish consumption and the migration of PCBs down-River.

The Five Year Review is intended to assure the protectiveness of the remedy in situations where contamination remains. 42 U.S.C. □9621(c). The purpose of the Review is directly related to determining whether a responsible party may be granted a covenant not to sue. A remedy that is not protective of human health and the environment may not be deemed by EPA as complete, and granting a covenant not to sue in those circumstances is not appropriate under the statute.

Moreover, GE has not met a fundamental requirement for a covenant not to sue under CERCLA Section 122(f). As required by CERCLA Section 122(f)(5), 42 U.S.C. □9622(f)(5), GE has not completed all outstanding obligations under the 2006 Consent Decree including, but not limited to, work related to (1) successfully restoring all River habitat damaged during implementation of remedial work, and (2) decommissioning and decontaminating the contaminated sediment processing facility.

A certification of completion of the remedial action should not issue to GE absent EPA s finding that the ROD s remedial action objectives have been met, that the remedy is *protective,* and that GE is in compliance with, and has *completed,* all obligations under the Consent Decree within the meaning of CERCLA Section 122(f), 42 U.S.C. □9622(f).

(2) EPA Should Determine with Reasonable Certainty the Time-Frame Necessary to Achieve the Remedial Action Objectives for the Reduction of PCB Concentrations in Fish

As discussed above, it is now clear that the remedy has not met the remedial action objective of reducing PCB concentrations in fish to 0.4 mg/kg by 2016, and may not reach the ROD s more dramatic reductions to 0.05 mg/kg. Accordingly, EPA must determine with reasonable certainty the time-frame by which there will be a reduction of PCB concentrations in fish so that fish consumption advisories for PCBs may be lifted in all contaminated River reaches of the Hudson River for all species.

New York s concurrence in the ROD was premised upon timely achieving the stated remedial action objectives for reducing PCB concentrations in fish. The State has been prejudiced not only by the failure to achieve those objectives timely, but by the current lack of certainty regarding when they will be achieved so that Hudson River fish can be safely consumed. We request that as a part of the Five Year Review process, EPA clearly define the time-frame for achieving the remedial action objectives set forth in the ROD.

In evaluating that time-frame, EPA must take into account the change in fish tissue sampling that occurred during GE s implementation of the baseline and remedial fish monitoring. The consequence of the fish data being collected in a manner that was inconsistent with what New York believed was required is important to answering the question of the time-frame for achieving the remedial objectives. This issue requires credible review by EPA.

(3) EPA's Determination of the Remedy's Protectiveness Must Be Supported By a Comprehensive Fish Consumption Survey to Quantify Current and Potential Future Human Exposure

The short- and long-term effectiveness of fish consumption advisories as an institutional control of human exposure to PCBs in edible fish is questionable. Despite the New York Department of Health s (NYSDOH s) annual issuance of the advisories, the public is still consuming fish from the Hudson River, a circumstance of which EPA is aware. Human consumption and exposure need to be quantified and evaluated for all contaminated River reaches in order to determine whether the advisories are sufficiently protective over both the short- and long-term. In addition, the localized effects of human exposure in certain more contaminated areas of the River also should be evaluated as part of EPA s Five Year Review and protectiveness determination.

In 1996, the NYSDOH conducted a survey and found that the public was still consuming Hudson River fish in significant amounts despite extensive public outreach and widespread knowledge of the levels of PCB contamination in fish (Survey Report attached). The public s level of fish consumption and exposure that is documented in NYSDOH s Survey Report may be greater today in light of the public s understandable - but incorrect - perception that the River has been cleaned up and that the fish may safely be eaten.

An updated survey of fish consumption along all contaminated reaches of the River from Hudson Falls to the Battery should be undertaken to accurately assess current and future human exposure and the efficacy of fish consumption advisories as short- and long-term institutional controls to protect human health.

(4) The Scope and Objectives of the Five-Year Review

Presently, EPA is undertaking a second Five Year Review for the Hudson River remedy in which it is required to make a finding that human health and the environment are being protected. CERCLA 121(c) provides:

> If the President selects a remedial action that results in any hazardous substances, pollutants, or contaminants remaining at the site, the President shall review such remedial action no less often than each 5 years after the initiation of such remedial action *to assure that human health and the environment are being protected by the remedial action* being implemented.

42 U.S.C. 9621(c) (emphasis added). Thus, fundamental to the Five Year Review is EPA s required finding that the remedy is protective.

Under applicable guidance, a protectiveness determination requires EPA to find that the remedy is functioning as intended by the ROD; that the exposure assumptions, toxicity data, cleanup levels, and remedial action objectives used at the time the remedy was selected are still valid; and that no information has come to light that could call the protectiveness of the remedy

into question. *See* EPA s Comprehensive Five Year Review Guidance, p. 4-1 (June 2001). Given this criteria, EPA cannot make those findings now because of the flaws in the models on which the ROD was based, and because of the lack of data quantifying current human exposure.

We understand that EPA expects to complete its Five Year Review by April 2017. However, the contemplated schedule does not provide sufficient time for EPA to make the necessary finding that human health and the environment are protected, particularly in the absence of (1) fish data showing current, post-dredging PCBs levels in fish, and (2) results of a fish consumption survey quantifying current human exposure. EPA should issue the Five Year Review only if it can be well-supported. EPA s Guidance indicates that the Five Year Review team be a multi-disciplinary team with relevant technical expertise to properly review the protectiveness of the remedy. *Id*. at p. 3-1, 3-2. Rather than issue a determination that lacks sufficient data and information, EPA should expedite the generation of necessary information, such as post-dredging fish and sediment data, should immediately initiate a fish consumption survey, and should involve independent experts in evaluating the flawed models.

EPA s Five Year Review process would benefit from greater formality, such as a written scope of work identifying objectives, the participants in the process and their areas of expertise, the tasks to be undertaken, areas of responsibility, a timetable for completing the tasks, and criteria for transparency in the process. We suggest that EPA s scope of work be issued for public comment so that interested parties understand how the process will proceed.

Thank you for your consideration of the foregoing. We look forward to your response and to continuing our discussions regarding the Hudson River.

Very truly yours,

Maureen F. Leary
James Woods
Brittany Haner
Assistant Attorneys General
John D. Davis
Environmental Scientist
Environmental Protection Bureau
Office of the Attorney General

Attachment

cc: Mathy Stanislaus
Brian Donohue
Peter Kautsky
Douglas Fisher
Thomas Hill

Attachment V

EPA IRIS for PCBs

Polychlorinated biphenyls (PCBs); CASRN 1336-36-3

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR PCBs

File First On-Line 05/01/1989

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	message	06/01/1994
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	yes	10/01/1996

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Polychlorinated biphenyls (PCBs)
CASRN — 1336-36-3

I.A.1. Oral RfD Summary

Please check the following individual aroclor files for RfD assessments: Aroclor 1016, Aroclor 1248, and Aroclor 1254.

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Polychlorinated biphenyls (PCBs)
CASRN — 1336-36-3

Not available at this time.

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Polychlorinated biphenyls (PCBs)
CASRN — 1336-36-3
Last Revised — 10/01/1996

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — B2; probable human carcinogen

Basis — A 1996 study found liver tumors in female rats exposed to Aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to 1260. These mixtures contain overlapping groups of congeners that, together, span the range of congeners most often found in environmental mixtures. Earlier studies found high, statistically significant incidences of liver tumors in rats ingesting Aroclor 1260 or Clophen A 60 (Kimbrough et al., 1975; Norback and Weltman, 1985; Schaeffer et al., 1984). Mechanistic studies are beginning to identify several congeners that have dioxin-like activity and may promote tumors by different modes of action. PCBs are absorbed

through ingestion, inhalation, and dermal exposure, after which they are transported similarly through the circulation. This provides a reasonable basis for expecting similar internal effects from different routes of environmental exposure. Information on relative absorption rates suggests that differences in toxicity across exposure routes are small. The human studies are being updated; currently available evidence is inadequate, but suggestive.

II.A.2. Human Carcinogenicity Data

Inadequate. A cohort study by Bertazzi et al. (1987) analyzed cancer mortality among workers at a capacitor manufacturing plant in Italy. PCB mixtures with 54%, then 42% chlorine were used through 1980. The cohort included 2100 workers (544 males and 1556 females) employed at least 1 week. At the end of follow-up in 1982, there were 64 deaths reported, 26 from cancer. In males, a statistically significant increase in death from gastrointestinal tract cancer was reported, compared with national and local rates (6 observed, 1.7 expected using national rates, SMR=346, CI=141-721; 2.2 expected using local rates, SMR=274, CI=112-572). In females, a statistically significant excess risk of death from hematologic cancer was reported, compared with local, but not national, rates (4 observed, 1.1 expected, SMR=377, CI=115- 877). Analyses by exposure duration, latency, and year of first exposure revealed no trend; however, the numbers are small.

A cohort study by Brown (1987) analyzed cancer mortality among workers at two capacitor manufacturing plants in New York and Massachusetts. At both plants the Aroclor mixture being used changed twice, from 1254 to 1242 to 1016. The cohort included 2588 workers (1270 males and 1318 females) employed at least 3 months in areas of the plants considered to have potential for heavy exposure to PCBs. At the end of follow-up in 1982, there were 295 deaths reported, 62 from cancer. Compared with national rates, a statistically significant increase in death from cancer of the liver, gall bladder, and biliary tract was reported (5 observed, 1.9 expected, SMR=263, $p<0.05$). Four of these five occurred among females employed at the Massachusetts plant. Analyses by time since first employment or length of employment revealed no trend; however, the numbers are small.

A cohort study by Sinks et al. (1992) analyzed cancer mortality among workers at a capacitor manufacturing plant in Indiana. Aroclor 1242, then 1016, had been used. The cohort included 3588 workers (2742 white males and 846 white females) employed at least 1 day. At the end of follow-up in 1986, there were 192 deaths reported, 54 from cancer. Workers were classified into five exposure zones based on distance from the impregnation ovens. Compared with national rates, a statistically significant excess risk of death from skin cancer was reported (8 observed, 2.0 expected, SMR=410, CI=180-800); all were malignant melanomas. A proportional hazards analysis revealed no pattern of association with exposure zone; however, the numbers are small.

Other occupational studies by NIOSH (1977), Gustavsson et al. (1986) and Shalat et al. (1989) looked for an association between occupational PCB exposure and cancer mortality. Because of small sample sizes, brief follow-up periods, and confounding exposures to other potential carcinogens, these studies are inconclusive.

Accidental ingestion: Serious adverse health effects, including liver cancer and skin disorders, have been observed in humans who consumed rice oil contaminated with PCBs in the "Yusho" incident in Japan or the "Yu-Cheng" incident in Taiwan. These effects have been attributed, at least in part, to heating of the PCBs and rice oil, causing formation of chlorinated dibenzofurans, which have the same mode of action as some PCB congeners (ATSDR, 1993; Safe, 1994).

II.A.3. Animal Carcinogenicity Data

Sufficient. Brunner et al. (1996) compared carcinogenicity across different Aroclors, dose levels, and sexes. Groups of 50 male or female Sprague-Dawley rats were fed diets with 25, 50, or 100 ppm Aroclor 1260 or 1254; 50 or 100 ppm Aroclor 1242; or 50, 100, or 200 ppm Aroclor 1016. There were 100 controls of each sex. The animals were killed at 104 weeks, after which a complete histopathologic evaluation was performed for control and high-dose groups; histopathologic evaluations of liver, brain, mammary gland, and male thyroid gland were also performed for low- and mid-dose groups.

Statistically significant increased incidences of liver adenomas or carcinomas were found in female rats for all Aroclors and in male rats for Aroclor 1260. Some of these tumors were hepatocholangiomas, a rare bile duct tumor seldom seen in control rats.

To investigate tumor progression after exposure has stopped, groups of 24 female rats were exposed for 52 weeks, then exposure was discontinued for an additional 52 weeks before the rats were killed. For Aroclors 1254 and 1242, tumor incidences from the stop study were approximately half those of the lifetime study; that is, nearly proportional to exposure duration. In contrast, stop-study tumor incidences were zero for Aroclor 1016, while for Aroclor 1260 they were generally greater than half those of the lifetime study. For 100 ppm Aroclor 1260, the stop study incidence was greater than that of the lifetime study, 71 vs. 48 percent.

Thyroid gland follicular cell adenomas or carcinomas were increased in males for all Aroclors; significant dose trends were noted for Aroclors 1254 and 1242. The increases did not continue proportionately above the lowest dose. No trends were apparent in females.

In female rats, the incidence of mammary tumors was decreased with lifetime exposure to Aroclor 1254 and, to a lesser extent, to 1260 or 1242; this result was not observed for Aroclor

1016. Decreases did not occur for any Aroclor in the stop study. The first mammary tumor was observed at a later age in the dosed groups.

Kimbrough et al. (1975) fed groups of 200 female Sherman rats diets with 0 or 100 ppm Aroclor 1260 for about 21 months. Six weeks later the rats were killed and their tissues were examined. Hepatocellular carcinomas and neoplastic nodules were significantly increased in rats fed Aroclor 1260.

The National Cancer Institute (NCI, 1978) fed groups of 24 male or female Fischer 344 rats diets with 0, 25, 50, or 100 ppm Aroclor 1254 for 104-105 weeks (24 months). Then the rats were killed and their tissues were examined. The combined incidence of leukemia and lymphoma in males was significantly increased by the Cochran-Armitage trend test; however, since Fisher exact tests were not also significant, NCI did not consider this result clearly related to Aroclor 1254. Hepatocellular adenomas and carcinomas were increased. Morgan et al. (1981) and Ward (1985) reevaluated gastric lesions from this study and found 6 adenocarcinomas in 144 exposed rats. This result is statistically significant, as gastric adenocarcinomas had occurred in only 1 of 3548 control male and female Fischer 344 rats in the NCI testing program. Intestinal metaplasia in exposed rats differed morphologically from controls, suggesting Aroclor 1254 can act as a tumor initiator.

Schaeffer et al. (1984) fed male weanling Wistar rats a standard diet for 8 weeks, then divided them into three groups. One group was fed the basic diet; for the other groups 100 ppm Clophen A 30 or A 60 was added. Rats were killed at 801 832 days (26.3 27.3 months) and were examined for lesions in the liver and some other tissues. For both mixtures, preneoplastic liver lesions were observed after 500 days (16.4 months) and hepatocellular carcinomas after 700 days (23 months) in rats dying before the end of the study. The investigators concluded, "Clophen A 60 had a definite, and Clophen A 30 a weak, carcinogenic effect on rat liver."

Norback and Weltman (1985) fed groups of male and female Sprague-Dawley rats diets of 0 or 100 ppm Aroclor 1260 for 16 months; the latter dose was reduced to 50 ppm for 8 more months. After 5 additional months on the control diet, the rats were killed and their livers were examined. Partial hepatectomy was performed on some rats at 1, 3, 6, 9, 12, 15, 18, and 24 months to evaluate sequential morphologic changes. In males and females fed Aroclor 1260, liver foci appeared at 3 months, area lesions at 6 months, neoplastic nodules at 12 months, trabecular carcinomas at 15 months, and adenocarcinomas at 24 months, demonstrating progression of liver lesions to carcinomas. By 29 months, 91% of females had liver carcinomas and 95% had carcinomas or neoplastic nodules; incidences in males were smaller, 4% and 15%, respectively. Vater et al. (1995) obtained individual animal results to determine whether the partial hepatectomies, which exert a strong proliferative effect on the remaining tissue, affected the incidence of liver tumors. They reported that the hepatectomies did not increase the tumor

incidence. Among females fed Aroclor 1260, liver tumors developed in 4 of 7 animals with hepatectomies and 37 of 39 without hepatectomies; no liver tumors developed in controls or males with hepatectomies.

Moore et al. (1994) reevaluated the preceding rat liver findings (Kimbrough et al., 1975; NCI, 1978; Schaeffer et al., 1984; Norback and Weltman, 1985) using criteria and nomenclature that had changed to reflect new understanding of mechanisms of toxicity and carcinogenesis. The reevaluation found somewhat fewer tumors than did the original investigators. The apparent increase for Clophen A 30 (Schaeffer et al., 1984) is no longer statistically significant.

II.A.4. Supporting Data for Carcinogenicity

Several studies of less-than-lifetime exposure are supportive of a carcinogenic response (Kimbrough et al., 1972; Kimbrough and Linder, 1974; Kimura and Baba, 1973; Ito et al., 1973, 1974; Rao and Banerji, 1988).

PCBs give generally negative results in tests of genetic activity (ATSDR, 1993), implying that PCBs induce tumors primarily through modes of action that do not involve gene mutation. Initiation-promotion studies for several commercial PCB mixtures and congeners show tumor promoting activity in liver and lung; these studies are beginning to identify a subset of mixture components that may be significant contributors to cancer induction (Silberhorn et al., 1990). Toxicity of some PCB congeners is correlated with induction of mixed-function oxidases; some congeners are phenobarbital-type inducers, others are 3-methylcholanthrene-type inducers, and some have mixed inducing properties (McFarland and Clarke, 1989). The latter two groups most resemble 2,3,7,8-tetrachlorodibenzo-p-dioxin in structure and toxicity.

Studies of structurally related agents: Studies of 2,3,7,8- tetrachlorodibenzo-p-dioxin and a polybrominated biphenyl (PBB) mixture are summarized here because the pattern of tumors found by Brunner et al. (1996) mimics the tumors induced in rats by these structurally related agents. The National Toxicology Program (NTP, 1982) exposed groups of 50 male or female Osborne-Mendel rats by gavage to 0, 1.4, 7.1, or 71 ng/kg-day 2,3,7,8- tetrachlorodibenzo-p-dioxin for 2 years. Similar to the Brunner et al. (1996) study, liver tumors were increased in female rats and thyroid gland follicular cell tumors were increased in male rats. Mammary tumors were not, however, decreased in dosed female rats. In another study, NTP (1983) exposed groups of 51 male or female Fischer 344/N rats by gavage to 0, 0.1, 0.3, 1, 3, or 10 mg/kg-day of a PBB mixture ("Firemaster FF 1") for 6 months, then exposure was discontinued for 23 months before the animals were killed. Statistically significant increased incidences of liver tumors were found in male and female rats. Dose-related increased incidences of cholangiocarcinomas were found in male and female rats.

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

II.B.1. Summary of Risk Estimates

Oral Slope Factor — See txt

Drinking Water Unit Risk — See txt

Extrapolation Method — Linear extrapolation below LED10s (U.S. EPA, 1996b)

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	See txt
E-5 (1 in 100,000)	See txt
E-6 (1 in 1,000,000)	See txt

II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)

Tumor Type — Liver hepatocellular adenomas, carcinomas, cholangiomas, or cholangiocarcinomas
Test animals — Female Sprague-Dawley rats
Route — Diet
Reference — Brunner et al., 1996; Norback and Weltman, 1985

	Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
Aroclor 1260	0 25 50 100	0 0.35 0.72 1.52	1/85 10/49 11/45 24/50

	Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
Aroclor 1254	0 25 50 100	0 0.35 0.76 1.59	1/85 19/45 28/49 28/49
Aroclor 1242	0 50 100	0 0.75 1.53	1/85 11/49 15/45
Aroclor 1016	0 50 100 200	0 0.72 1.43 2.99	1/85 1/48 7/45 6/50
Aroclor 1260 (Norback and Weltman, 1985)	0 100/50/0	0.75 1.3	1/45 41/46

II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)

The cancer potency of PCB mixtures is determined using a tiered approach that depends on the information available. The following tier descriptions discuss all environmental exposure routes:

TIERS OF HUMAN SLOPE FACTORS FOR ENVIRONMENTAL PCBs

HIGH RISK AND PERSISTENCE

Upper-bound slope factor: 2.0 per (mg/kg)/day
Central-estimate slope factor: 1.0 per (mg/kg)/day

Criteria for use:
- Food chain exposure
- Sediment or soil ingestion
- Dust or aerosol inhalation

- Dermal exposure, if an absorption factor has been applied
- Presence of dioxin-like, tumor-promoting, or persistent congeners
- Early-life exposure (all pathways and mixtures)

LOW RISK AND PERSISTENCE

Upper-bound slope factor: 0.4 per (mg/kg)/day
Central-estimate slope factor: 0.3 per (mg/kg)/day

Criteria for use:
- Ingestion of water-soluble congeners
- Inhalation of evaporated congeners
- Dermal exposure, if no absorption factor has been applied

LOWEST RISK AND PERSISTENCE

Upper-bound slope factor: 0.07 per (mg/kg)/day
Central-estimate slope factor: 0.04 per (mg/kg)/day

Criteria for use: Congener or isomer analyses verify that congeners with more than 4 chlorines comprise less than 1/2% of total PCBs.

Slope factors are multiplied by lifetime average daily doses to estimate the cancer risk. SAMPLE CALCULATIONS ARE GIVEN IN U.S. EPA (1996a). Although PCB exposures are often characterized in terms of Aroclors, this can be both imprecise and inappropriate. Total PCBs or congener or isomer analyses are recommended.

When congener concentrations are available, the slope-factor approach can be supplemented by analysis of dioxin TEQs to evaluate dioxin-like toxicity. Risks from dioxin-like congeners (evaluated using dioxin TEQs) would be added to risks from the rest of the mixture (evaluated using slope factors applied to total PCBs reduced by the amount of dioxin-like congeners). SAMPLE CALCULATIONS ARE GIVEN IN U.S. EPA (1996a).

Depending on the specific application, either central estimates or upper bounds can be appropriate. Central estimates describe a typical individual's risk, while upper bounds provide assurance that this risk is not likely to be underestimated if the underlying model is correct. The upper bounds calculated in this assessment reflect study design and provide no information about sensitive individuals or groups. Central estimates are useful for estimating aggregate risk across a population. Central estimates are used for comparing or ranking environmental hazards, while upper bounds provide information about the precision of the comparison or ranking.

Some PCBs persist in the body and retain biological activity after exposure stops (Anderson et al., 1991a). Compared with the current default practice of assuming that less-than-lifetime effects are proportional to exposure duration, rats exposed to a persistent mixture (Aroclor 1260) had more tumors, while rats exposed to a less persistent mixture (Aroclor 1016) had fewer tumors (Brunner et al., 1996). Thus there may be greater-than- proportional effects from less-than-lifetime exposure, especially for persistent mixtures and for early-life exposures.

Highly exposed populations include some nursing infants and consumers of game fish, game animals, or products of animals contaminated through the food chain. Highly sensitive populations include people with decreased liver function and infants (Calabrese and Sorenson, 1977).

Because of the potential magnitude of early-life exposures (ATSDR, 1993; Dewailly et al., 1991, 1994), the possibility of greater perinatal sensitivity (Calabrese and Sorenson, 1977; Rao and Banerji, 1988), and the likelihood of interactions among thyroid and hormonal development, it is reasonable to conclude that early-life exposures may be associated with increased risks. Due to this potential for higher sensitivity early in life, the "high risk" tier is used for all early-life exposure.

It is crucial to recognize that commercial PCBs tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain (that is, the rats were fed Aroclor mixtures, not environmental mixtures that had been bioaccumulated). Bioaccumulated PCBs appear to be more toxic than commercial PCBs (Aulerich et al., 1986; Hornshaw et al., 1983) and appear to be more persistent in the body (Hovinga et al., 1992). For exposure through the food chain, risks can be higher than those estimated in this assessment.

In calculating these estimates, administered doses were expressed as a lifetime daily average calculated from weekly body weight measurements and food consumption estimates (Keenan and Stickney, 1996). Doses were scaled from rats to humans using a factor based on the 3/4 power of relative body weight.

UNIT RISK ESTIMATE AND DRINKING WATER CONCENTRATIONS

For ingestion of water-soluble congeners, the middle-tier slope factor can be converted to a unit risk estimate and drinking water concentrations associated with specified risk levels.

Upper-bound slope factor: 0.4 per (mg/kg)/day
Upper-bound unit risk: 1 x 10-5 per ug/L

Drinking water concentration associated with a risk of:

1 in 10,000	10 ug/L
1 in 100,000	1 ug/L
1 in 1,000,000	0.1 ug/L

These estimates should not be used if drinking water concentrations exceed 1000 ug/L, since above this concentration the dose-response curve in the experimental range may provide better estimates.

For food chain exposure or ingestion that includes contaminated sediment or soil, the slope factor for "high risk and persistence" should be used instead.

II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)

Joint consideration of cancer studies and environmental processes leads to a conclusion that environmental PCB mixtures are highly likely to pose a risk of cancer to humans. Although environmental mixtures have not been tested in cancer assays, this conclusion is supported by several complementary sources of information. Statistically significant, dose-related, increased incidences of liver tumors were induced in female rats by Aroclors 1260, 1254, 1242, and 1016 (Brunner et al., 1996). These mixtures contain overlapping groups of congeners that, together, span the range of congeners most frequently found in environmental mixtures. Several congeners have dioxin-like activity (Safe, 1994) and may promote tumors by different modes of action (Silberhorn et al., 1990); these congeners are found in environmental samples and in a variety of organisms, including humans (McFarland and Clarke, 1989).

The range of potency observed for commercial mixtures is used to represent the potency of environmental mixtures. The range reflects experimental uncertainty and variability of commercial mixtures, but not human heterogeneity or differences between commercial and environmental mixtures. Environmental processes alter mixtures through partitioning, transformation, and bioaccumulation, thereby decreasing or increasing toxicity. The overall effect can be considerable, and the range observed for commercial mixtures may underestimate the true range for environmental mixtures (Hutzinger et al., 1974; Callahan et al., 1979). Limiting the potency of environmental mixtures to the range observed for commercial mixtures reflects a decision to base potency estimates on experimental results, however uncertain, rather than apply safety factors to compensate for lack of information.

A tiered approach allows use of different kinds of information in estimating the potency of environmental mixtures. When congener information is limited, exposure pathway is used to indicate whether environmental processes have decreased or increased a mixture's potency. Partitioning, transformation, and bioaccumulation have been extensively studied (Hutzinger et al., 1974; Callahan et al., 1979) and can be associated with exposure pathway, thus the use of exposure pathway to represent environmental processes increases confidence in the risks inferred for environmental mixtures. For example, evaporated or dissolved congeners tend to be lower in chlorine content than the original mixture; they tend also to be more inclined to metabolism and elimination and lower in persistence and toxicity. On the other hand, congeners adsorbed to sediment or soil tend to be higher in chlorine content and persistence, and bioaccumulated congeners ingested through the food chain tend to be highest of all. Rates of these processes vary over several orders of magnitude (Hutzinger et al., 1974; Callahan et al., 1979). When available, congener information is an important tool for refining a potency estimate that was based on exposure pathway.

Extrapolation to environmental levels is based on models that are linear at low doses. Low-dose-linear models are appropriate when a carcinogen acts in concert with other exposures and processes that cause a background incidence of cancer (Crump et al, 1976; Lutz, 1990). Even when the mode of action indicates a nonlinear dose-response curve in homogeneous animal populations, the presence of genetic and lifestyle factors in a heterogeneous human population tends to make the dose-response curve more linear (Lutz, 1990). This is because genetic and lifestyle factors contribute to a wider spread of human sensitivity, which extends and straightens the dose-response curve over a wider range.

Uncertainty around these estimates extends in both directions. The slope factor ranges primarily reflect mixture variability, and so are not necessarily appropriate for probabilistic analyses that attempt to describe model uncertainty and parameter uncertainty. Estimates based on animal studies benefit from controlled exposures and absence of confounding factors; however, there is uncertainty in extrapolating dose and response rates across species. Information is lacking to evaluate high-to-low-dose differences. PCBs are absorbed through ingestion, inhalation, and dermal exposure, after which they are transported similarly through the circulation (ATSDR, 1993). This provides a reasonable basis for expecting similar internal effects from different routes of environmental exposure. Information on relative absorption rates suggests that differences in toxicity across exposure routes are small. The principal uncertainty, though, is using commercial mixtures to make inferences about environmental mixtures.

When exposure involves the food chain, uncertainty extends principally in one direction: through the food chain, living organisms selectively bioaccumulate persistent congeners, but commercial mixtures tested in laboratory animals were not subject to prior selective retention of persistent congeners. Bioaccumulated PCBs appear to be more toxic than commercial PCBs (Aulerich et

al., 1986; Hornshaw et al., 1983) and appear to be more persistent in the body (Hovinga et al., 1992). For exposure through the food chain, risks can be higher than those estimated in this assessment. Two highly exposed populations, nursing infants and consumers of contaminated game animals, are exposed through the food chain.

The dioxin-like nature of some PCBs raises a concern for cumulative exposure, as dioxin-like congeners add to background exposure of other dioxin- like compounds and augment processes associated with dioxin toxicity. This weighs against considering PCB exposure in isolation or as an increment to a background exposure of zero. Confidence in this assessment's use of low-dose-linear models is enhanced when there is additivity to background exposures and processes (Crump et al, 1976; Lutz, 1990).

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

II.C.1. Summary of Risk Estimates

Inhalation Unit Risk — See txt

Extrapolation Method — Linear extrapolation below LED10s (U.S. EPA, 1996b)

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	See txt
E-5 (1 in 100,000)	See txt
E-6 (1 in 1,000,000)	See txt

II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

See Dose-Response Data for oral exposure.

II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

See Additional Comments for oral exposure.

For inhalation of evaporated congeners, the middle-tier slope factor can be converted to a unit risk estimate and ambient air concentrations associated with specified risk levels.

Upper-bound slope factor: 0.4 per (mg/kg)/day
Upper-bound unit risk: 1 x 10-4 per ug/cu.m

Ambient air concentration associated with a risk of:

1 in 10,000	1 ug/cu.m
1 in 100,000	0.1 ug/cu.m
1 in 1,000,000	0.01 ug/cu.m

These estimates should not be used if ambient air concentrations exceed 100 ug/cu.m, since above this concentration the dose-response curve in the experimental range may provide better estimates.

For inhalation of an aerosol or dust contaminated with PCBs, the slope factor for "high risk and persistence" should be used instead.

II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

See Discussion of Confidence for oral exposure. Information on relative absorption rates suggests that differences in toxicity across exposure routes are small.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1996a [Available from the IRIS Hotline, Telephone: (202)566-1676; FAX (202)566-1749)].

The source document and IRIS Summary were considered at a public, external peer review workshop in May 1996. A workshop report was written by the review panel (U.S. EPA, 1996c). All comments have been carefully evaluated and considered in this IRIS Summary. A record of these comments is summarized in the IRIS documentation files.

Other EPA Documentation — U.S. EPA, 1988

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 08/22/1996

Verification Date — 08/22/1996

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris epa.gov (internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Polychlorinated biphenyls (PCBs)
CASRN — 1336-36-3

VI.A. Oral RfD References

None

VI.B. Inhalation RfC References

None

VI.C. Carcinogenicity Assessment References

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VII. Revision History

Substance Name — Polychlorinated biphenyls (PCBs)
CASRN — 1336-36-3

Date	Section	Description
05/01/1989	II.	Carcinogen summary on-line
06/01/1994	I.A.	Message only
01/01/1996	II.	Note added to assessment
10/01/1996	II.	File replaced; cancer potency of mixtures addressed

VIII. Synonyms

Substance Name — Polychlorinated biphenyls (PCBs)
CASRN — 1336-36-3
Last Revised — -- 05/01/1989

- 1336-36-3
- AROCLOR
- AROCLOR 1221
- AROCLOR 1232
- AROCLOR 1242
- AROCLOR 1248
- AROCLOR 1254
- AROCLOR 1260
- AROCLOR 1262
- AROCLOR 1268
- AROCLOR 2565
- AROCLOR 4465
- AROCLOR 5442
- BIPHENYL, POLYCHLORO-
- CHLOPHEN
- CHLOREXTOL
- CHLORINATED BIPHENYL
- CHLORINATED DIPHENYL
- CHLORINATED DIPHENYLENE
- CHLORO BIPHENYL
- CHLORO 1,1-BIPHENYL
- CLOPHEN
- DYKANOL
- FENCLOR
- INERTEEN
- KANECHLOR
- KANECHLOR 300
- KANECHLOR 400
- MONTAR
- NOFLAMOL
- PCB
- PCBs
- PHENOCHLOR
- PHENOCLOR
- POLYCHLORINATED BIPHENYL
- Polychlorinated Biphenyls
- POLYCHLOROBIPHENYL

- PYRALENE
- PYRANOL
- SANTOTHERM
- SANTOTHERM FR
- SOVOL
- THERMINOL FR-1
- UN 2315

Attachment W

Exposure to and Health Effects of Volatile PCBs

David O. Carpenter*

Exposure to and health effects of volatile PCBs

Abstract

Introduction: Polychlorinated biphenyls (PCBs) are persistent, lipophilic contaminants that are known to increase risk of a number of human diseases. Although ingestion of animal fats is a major route of exposure, there is increasing evidence that inhalation of vapor-phase PCBs is also important and may be as or even more important than ingestion under some circumstances.

Methods: The evidence that inhalation of PCBs may cause cancer, heart disease, hypertension, and diabetes is reviewed and presented in this report.

Results: PCBs are known human carcinogens. A husband and wife, occupationally required to 'smell' PCB-containing oils, both developed thyroid cancer, malignant melanoma/severely melanocytic dysplastic nevus (a precursor to malignant melanoma) and the husband, a non-smoker, developed and died of lung cancer. The serum of both had highly elevated concentrations of lower chlorinated, volatile PCB congeners. In other studies, residents living near PCB-containing hazardous waste sites, and thus breathing PCB-contaminated air, have elevated rates of hospitalization for cardiovascular disease, hypertension, diabetes and reduced cognitive performance, whereas other studies in defined populations show that there is an elevated risk of all of these diseases in individuals with elevated serum PCBs.

Conclusions: These results are consistent with the conclusion that inhaled PCBs can increase risk of cancer, cardiovascular disease, hypertension, diabetes and reduce cognitive function.

Keywords: cancer; cardiovascular disease; diabetes; hypertension; PCB exposure; volatile PCBs.

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Introduction

Polychlorinated biphenyls (PCBs) were manufactured in many countries from the late 1920s until they were found to be persistent and toxic in the late 1970s, when their manufacture and use was stopped in most developed countries. It is reported, however, that they are still being manufactured in North Korea, and even in the US, many transformers and capacitors that are still being used contain PCBs.

PCBs consist of mixtures of up to 209 individual congeners, which vary depending on how many chlorines are on the biphenyl rings and where they are located on the molecule. Figure 1 shows the PCB molecule and the convention for identifying different congeners based on the location of chlorines. PCBs were manufactured in many countries as commercial mixtures through the chlorination of biphenyl with anhydrous chlorine in the presence of a catalyst, usually iron. The duration of the reaction determined the average degree of chlorination. In the US, almost all PCBs were manufactured by Monsanto, who sold commercial mixtures under the trade name 'Aroclor'. Aroclor 1242 was 42% chlorine by weight, whereas Aroclor 1260 was 60% chlorine. However, all commercial products contained a variety of PCB congeners, with the exception of Aroclor 1271, which was pure PCB 209 that contained chlorine groups at all 10 sites.

Most widely used commercial PCB mixtures are oils, and the greater the degree of chlorination, the more viscous the oil. They had many useful purposes. However, they had major uses in capacitors and light ballasts given because they are relatively nonflammable and nonconductive. They were widely used as hydraulic fluids, as solvents for paints or caulking, in carbonless copy paper, and in other products requiring a lipophilic solvent.

Although all PCB congeners have some common properties, they also have significant differences in physical properties and routes of exposure to humans. In general, PCBs have low water solubility and volatility. However, those congeners containing fewer chlorines are more water soluble and more volatile than those with more chlorines (1, 2). Table 1 (3) shows vapor pressure, water solubility, log octanol/water partition coefficient (log K_{ow}), and approximate evaporation rates as a function of the number of chlorines on the PCB molecule.

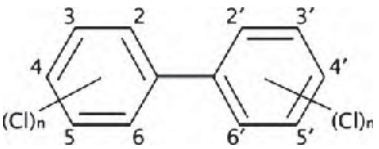

Figure 1: The structure of PCBs. There can be any number of chlorines around the biphenyl ring between one and ten. The convention for labelling the position is shown by the numbers, where the 2 and 6 positions are *ortho,* the 3 and 5 positions are *meta*, and the 4 position is *para*. The prime sign distinguishes in which ring the chlorines are located.

Table 1: Physical characteristics of PCBs by homologue groups at 25°.

PCB homologue group	Vapor pressure, Pa	Water solubility, g/m³	Log octanol/ Water coefficient	Evaporation rate, g/m³/h
Monochloro	1.1	4.0	4.7	0.25
Dichloro	0.24	1.6	5.1	0.065
Tetrachloro	0.012	0.26	5.9	4.2×10^{-3}
Hexachloro	5.8×10^{-4}	0.038	6.7	2.5×10^{-4}
Octachloro	2.8×10^{-5}	5.5×10^{-4}	7.5	1.5×10^{-5}
Decachloro	1.4×10^{-6}	7.6×10^{-4}	8.3	8.5×10^{-7}

Data from Ref (3).

Even commercial mixtures with primarily highly chlorinated congeners contain lower chlorinated congeners at low concentrations. Figure 2 shows the congener pattern of Aroclor 1260 (60% chlorine by weight) and that of PCBs in the vapor phase, resulting from blowing air over the commercial mixture. Clearly, even this highly chlorinated mixture contains lower chlorinated PCBs that volatize. There is also some volatilization of moderately chlorinated congeners, but the overall profile in the vapor phase shifts markedly to the left, indicating that lower chlorinated congeners are more volatile.

PCBs can volatilize from a variety of sources, including commercial mixtures, water, landfills, and commercial products. As lower chlorinated PCBs are more water soluble and more volatile (Table 1) they will selectively dissolve in water and then move from a soluble aqueous phase into the air. PCBs evaporate along with the water (4, 5), and this process is very temperature dependent (6). Volatile loss of PCBs from Lake Superior was calculated to be about 1900 kg per year (7). Outdoor air concentrations of PCBs near New Bedford Harbor, a highly contaminated body of water, ranged from 0.4 to 53 ng/m³ (8); these are significantly higher than those at a comparison site. PCB fluxes to air along the contaminated Hudson River ranged from 0.5 to 13 μg/m²/day (9).

The greater water solubility of lower chlorinated PCBs has implications for drinking water quality. The majority of the higher chlorinated congeners will be bound to particulates in water and then removed by standard drinking water treatments. However, those that are dissolved are more difficult to remove and may be an important route of human exposure, especially if contaminated surface water is used for municipal drinking water.

PCBs will also volatilize from contaminated soils and sediments. As from water, the PCBs volatize with water, and dry sediments lose fewer PCBs to the air as compared with wet sediments or soils (4). PCBs can also volatilize from landfills, depending upon how tightly they are covered (10). Hermanson et al. (11) studied air PCB concentrations near a Monsanto landfill in Anniston, Alabama, the site of a PCB synthesis factory, and compared results to those from a nearby site that had superficial soil PCB contamination. They found less dependence on surface temperature for PCB release to air from the landfill, and suggested that most of the sources of PCBs from the landfill site were materials buried within the landfill.

In addition to the differences in physical properties, congeners have both differences in rates of metabolism in the human body and major differences in mechanisms of action and health effects in humans. PCBs, like most chlorinated compounds, are poorly metabolized and are thus persistent. In general the half-life increases with number of chlorines but other factors like location of the chlorines around the ring also influence rates of metabolism. The half-lives in humans of several individual PCB congeners are shown in Table 2 (12). *Ortho* chlorine substitution usually increases the half-life relative to that of a PCB with the same number of chlorines but with none in the *ortho* position (13).

Many of the volatile mono-, di-, and tri-chloro congeners are metabolized within hours in rats (14). Hu et al. (15) found that labeled PCB 11 (3,3′-dichloro biphenyl) had a half-life of 12 h in male rats. Although human metabolism is generally not as rapid as in rodents, it is sufficiently rapid such that lower chlorinated congeners are rarely found at significant concentrations in human blood. Long half-life makes it convenient to determine the exposure of a person to PCBs in the past, but there is often the assumption that long half-life is indicative of greater health effect. This assumption is not necessary correct. This is because even those congeners that are more rapidly metabolized may have significant toxicity, especially if there is prolonged exposure, as would be the case if they were inhaled on a daily basis.

The major metabolism of PCBs is through cytochrome P450s in the liver and other organs (13). This results in

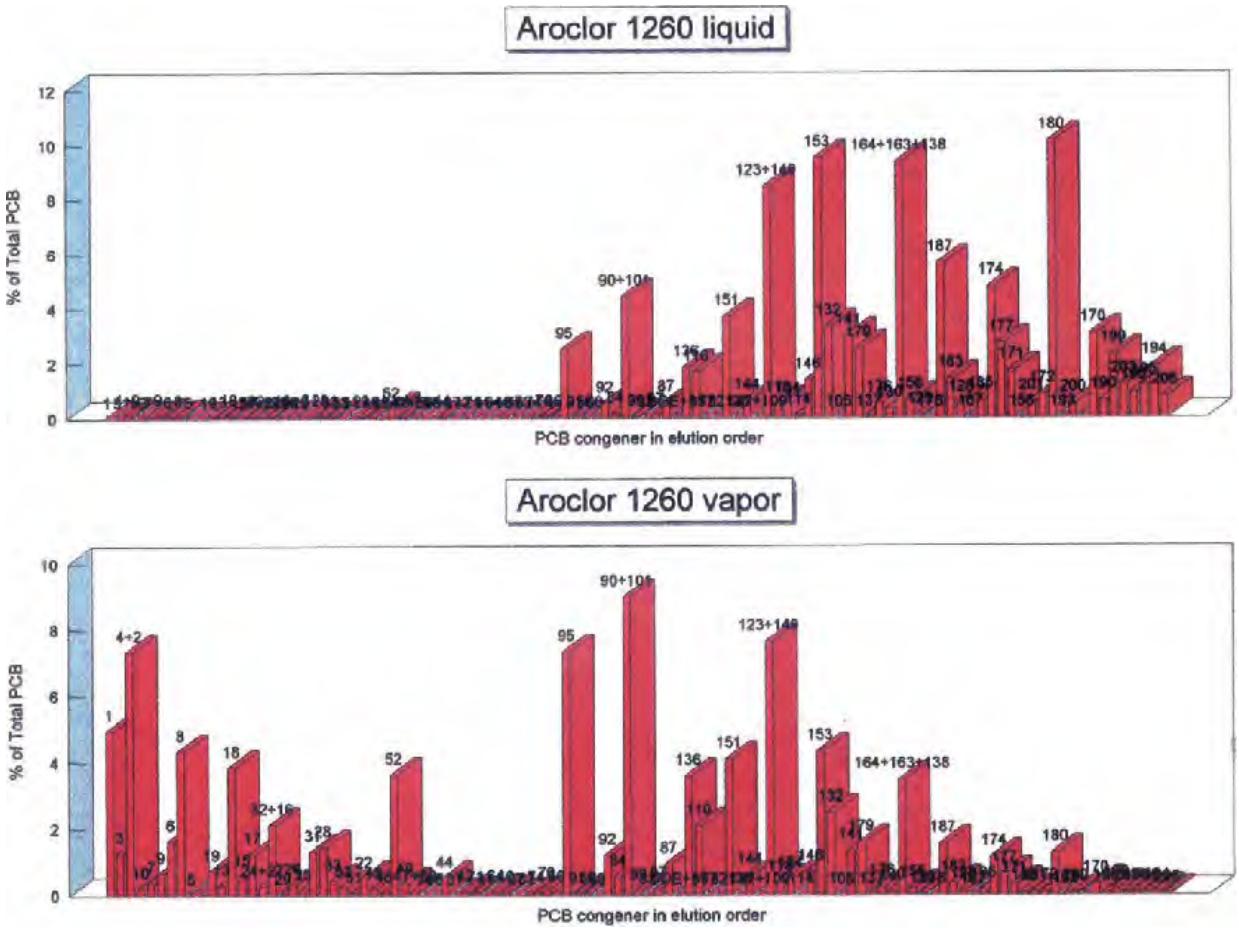

Figure 2: The congener patterns in Aroclor 1260 liquid (top) and the congener pattern seen when passing air over the liquid and collecting and analyzing the vapor-phase PCBs.
Peaks are shown in the order they elute from the column. The numbers above the peaks identify individual congeners or groups of congeners. Those peaks to the left have fewer chlorines.

Table 2: Half-lives of single PCB congeners in the human adult body.

PCB number	PCB structure	Half-life, years
28	2,4,4′ Trichlorobiphenyl	5.5
52	2,2′,5,5′ Tetrachlorobiphenyl	2.6
105	2,3,3′4,4′ Pentachlorobiphenyl	5.2
118	2,3′4,4′,5 Pentachlorobiphenyl	9.3
138	2,2′,3,4,4′,5′ Hexahlorobiphenyl	10.8
153	2,2′,4,4′,5,5′ Hexachlorobiphenyl	14.4
170	2,2′,3,3′,4,4′,5 Heptachlororbiphenyl	15.5
180	2,2′,3,4,4′,5,5′ Heptachlorobiphenyl	11.5

Data from Ref (4).

introduction of oxygen onto the molecule, which then allows for further metabolism by other transferases. Many of the hydroxylated or methyl sulfonated metabolites are somewhat persistent and have biologic activity (16). The position of the chlorines around the PCB molecule influences the rate of metabolism (17). This is why different PCB congeners with the same number of chlorines have different half-lives, as shown in Table 2. In addition, different congeners are targets of different P450s. Many studies have focused on PCB congeners that have dioxin-like activity as well as those that bind to the aryl hydrocarbon receptor, induce P4501A and then induce many different genes (18). Other congeners induce different P450s and many genes, but with a different pattern (19). To make matters even more complex, the profile of genes that are induced may vary from one tissue to another (20). Many of the adverse health effects reported in humans are likely a consequence of different patterns of gene induction.

Despite the more rapid metabolism of lower chlorinated PCBs, evidence for inhalation exposure can be obtained from serum samples. Our group has studied PCB exposure in a Native American population for many years. Many older

adults have a pattern of congeners dominated by a few highly chlorinated and persistent congeners like PCBs 138, 153, 170, and 180. However, we have been able to identify a pattern of lower chlorinated PCBs in the serum of younger Mohawks, which matched closely to the pattern of the PCB profile in air over a contaminated site (21) Figure 3. The pattern could not be observed clearly in older individuals because serum levels increase with age and the PCBs from ingestion obscure those more readily metabolized PCBs.

Herrick et al. (22) measured serum PCB levels in teachers working in a school that had elevated PCBs in indoor air, and found significantly higher concentrations of lower

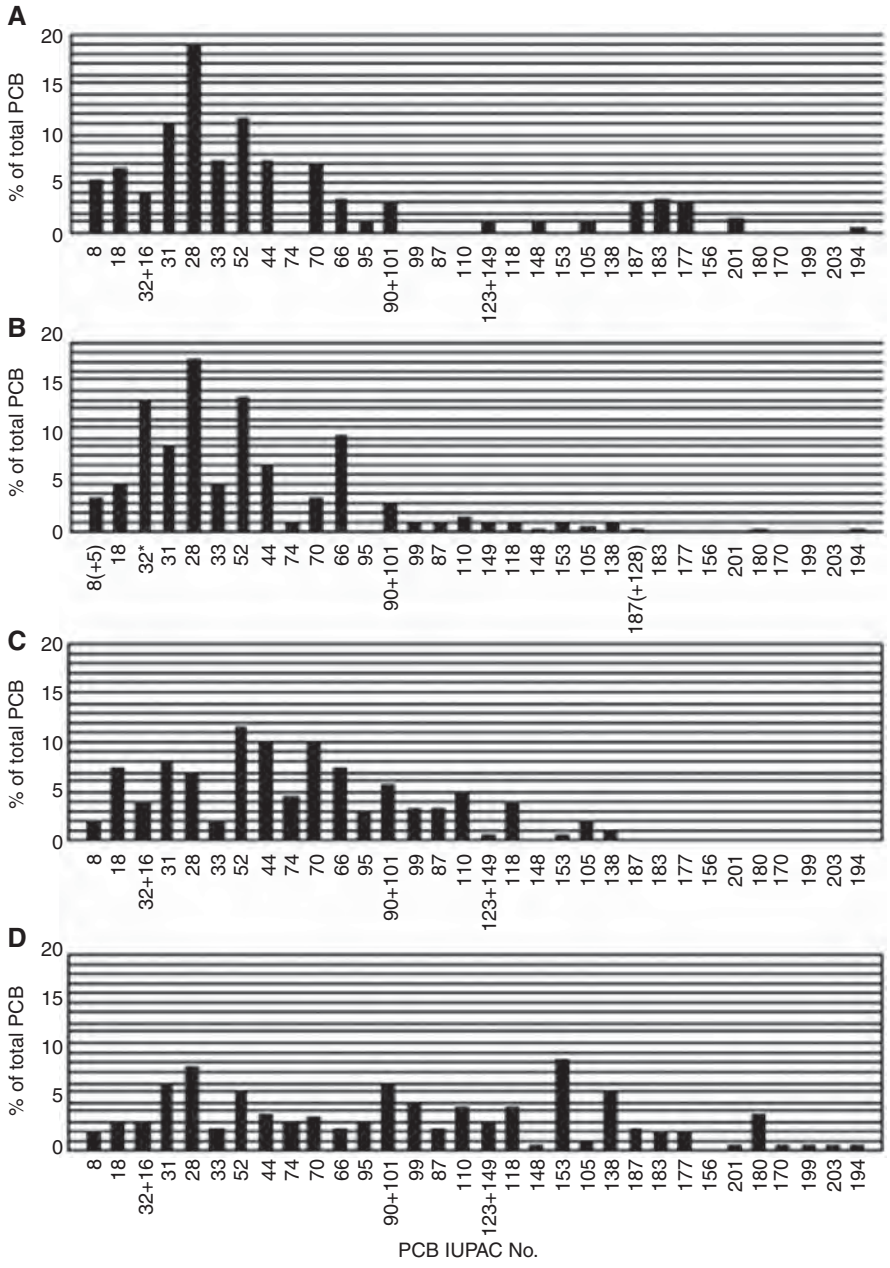

Figure 3: Congener compositions of (A) End-member (EM)-1 as determined by polytopic vector analysis (PVA) of serum PCB congener data for 702 adult Mohawks, (B) air sampled above "Contaminant Cove" at the western boundary of Akwesasne in summer 1993,[17] (C) native commercial A1248 liquid, and (D) serum from the subject with the highest proportion (46.2%) of EM-1. For profiles not generated in the authors' laboratory (i.e., B), the same congener elution order as that in the other samples is presented to facilitate comparisons. Differences in congener coelutions between samples are indicated by brackets; congeners analyzed in the authors' laboratory but not by others are shown in italics. For brevity, CB 138 is listed alone although it coelutes with CBs 163 and 164 for all samples. In addition, CB 32 coelutes with CBs 11, 12, and 13 for the sample shown in (B). *Reprinted from DeCaprio et al.[21] with permission from Elsevier B.V.*

chlorinated congeners (PCBs 6–74) than those found in unexposed teachers. Meyer et al. (23) obtained serum PCB measurements from 134 residents of a flat with high concentrations of PCBs in the indoor air, and compared levels to those of 139 unexposed persons. Levels of 27 congeners, especially lower chlorinated congeners, were found to be four times higher in the serum of the exposed individuals.

The goal of this paper is to review the evidence that the inhalation of PCBs can lead to adverse health effects in humans. The paper will focus on a few specific diseases for which evidence exists to support the conclusion that inhalation is an important route of exposure. The problem is that most scientists who are investigating health effects of PCBs use serum PCB concentration as their exposure assessment measure. Given that most of the more volatile congeners are rapidly metabolized, they are not present in high concentrations in serum samples and, thus, they are usually not considered. However, the typical source of inhaled PCBs is indoor air in homes, schools and offices, places where people spend many hours a day. Under these circumstances, people may be more or less continuously exposed and affected by the lower chlorinated congeners.

Cancer

PCBs have been identified as Group 1, known human carcinogens, by the International Agency for Research on Cancer (24). The specific cancer with the strongest evidence is malignant melanoma. There are, however, many of types of cancer for which strong associations with serum PCB levels have been found (25). However, there is little direct evidence for cancer in humans resulting from inhalation exposure to PCBs.

Until the recent IARC identification of all PCBs being carcinogenic, there was a widespread belief that only dioxin-like PCBs had carcinogenic activity. This is despite clear evidence presented by van der Plas et al. (26). They reported that majority (about 80%) of the tumor-promoting activity of PCBs can be found the in 2–4 *ortho*-substituted congener groups, which have little or no dioxin-like activity. Sandal et al. (27) compared the genotoxic activities of PCB 52 (2,2′,5,5′tetrachloro biphenyl, a non-dioxin-like congener) and PCB 77 (3,3′,4,4′ tetrachlorobiphenyl, a dioxin-like congener) on cultured human lymphocytes. They found that both congeners caused DNA damage as monitored by the comet assay, but that PCB 52 is significantly more potent. Both PCB 9 (2,5 dichlorobiphenyl) (28) and PCB 11 (29) generate reactive oxygen species, known to be a risk factor for cell damage and death. Ludewig et al. (30) found that PCB 3 (4-monochlorobiphenyl) and/or its metabolites increase mutations in rat liver. Tan et al. (31) found that PCBs 8 (2,4 dichlorobiphenyl), 28, 47 (2,2′4,4′-tetrachlorobiphenyl), and 52 are cytotoxic to both neurons and thymocytes, but the dioxin-like congeners PCBs 77, 80 (3,3′,5,5′-tetrachlorobiphenyl) and 81 (3,4,4′,5-tetrachlorobiphenyl) are not. Although not all of these effects are necessarily directly related to cancer, they clearly demonstrate toxicity of lower chlorinated, non-dioxin-like congeners.

Case study

Company X was an analytic services laboratory that provided analysis of fluids from electric transformers. Up until 1977, when their manufacture and new use was outlawed by the US Environmental Protection Agency (EPA) due to their persistence and toxicity, most electric transformers were filled with commercial mixtures of PCBs. However, old transformers that have not been serviced still contain PCBs. Now EPA requires that the fluid from transformers being serviced or discarded be tested to determine whether PCBs are present; if they are, then the EPA requires that the fluid be removed and the transformer cleaned and filled with a non-toxic substitute. All PCB-containing fluids at concentrations <50 ppm are to be treated as hazardous waste, and rules have been established to regulate disposal of oils containing PCBs at concentrations between 2 and 50 ppm.

JM, a relatively dark-skinned Hispanic, was employed by company X between 1994 and 2003 as a laboratory technician. His job was to analyze 100–150 transformer oil samples per day to determine whether they contained PCBs. It was known that 10%–20% of those samples would have PCBs at concentrations ranging from 50 to 499 ppm, and another 10% would have even higher concentrations, some being 100% commercial PCBs. JM was told to smell the fluid to determine whether or not it contained high concentrations of PCBs. PCBs have a subtle but distinctive odor. The reason for smelling the fluids before analyzing them was that running a sample with a high PCB concentration in the gas chromatograph would result in contamination that would then take time to wash out. Thus, if samples with high concentrations could be identified before being run, they could be serially diluted to the point that they would not require extra time to be taken to wash out the gas chromatograph.

JM was born in 1967 and did not smoke nor drink to excess. His medical history was unremarkable except for hypertension, and elevated LDL with a slightly low HDL. On December 14, 2001 he was found to have a greatly

reduced thyroid stimulating hormone (TSH) level, and highly elevated thyroxine (T4) level. On February 28, 2003 he was treated with radioactive ^{131}I, which resulted in a decrease in his TSH level. On March 3, 2003 a large papillary thyroid carcinoma was removed in a subtotal thyroidectomy. The tumor surrounded the vagus nerve and it was difficult to remove. On August 26, 2003 he was found to still have an abnormally elevated uptake of ^{131}I, which was suggestive of recurrent disease. Although he continued to work at company X after his surgery, he was no longer required to analyze for PCBs. In March, 2011 JM had a malignant melanoma removed from his back. In March, 2013 JM was diagnosed with lung cancer, which on biopsy, proved to be a poorly differentiated adenocarcinoma, not a metastasis from the melanoma. JM died later in 2013 with massive hemorrhagic brain metastases.

GM, wife of JM, was born in 1968 and hired by company X in 1996. Her job was to dump oils that were in the GC sampling vials that had been analyzed into 55 gallon drums, separating those with and without high concentrations of PCBs, and ensure that any liquids containing PCBs were not allowed down the drain. She also was required to wash the glassware. She worked in a 50 sq ft room with a hood and waste basin but without windows or air conditioning, and was told to keep the door closed. When the oils were to be dumped, she was told to sniff each sample in order to determine which 55 gallon drum the material should be placed in. If it smelled like PCBs, it would go into one drum, but if not then it should go into the other. The glassware contaminated with PCBs was to be washed with toluene and acetone, followed by soap and water. She was never provided with a lab coat, gloves, or a mask.

GM was also diagnosed with thyroid cancer in May of 2003, after which she stopped working at company X. She had a total thyroidectomy in July, 2003. She completed a course of 100 mC ^{131}I on September, 2003. She had some abnormal uptake of the isotope on August 26, 2003, but there was no evidence of recurrent disease by March, 2004. In 2011, she was diagnosed with a compound melanocytic dysplastic nevus, a highly dangerous mole that is a precursor to melanoma. This was removed. She also had abnormal liver function tests, perhaps a fatty liver, diabetes, and hypertension. She does not drink and does not have hepatitis.

Serum samples were obtained in the late summer and fall of 2005 for measurement of PCBs, and analysis was done by ERGO Forschungsgesellschaft mbH in Hamburg, Germany. The results for six PCB congeners are shown in Table 3.

Table 3: PCB concentrations (μg/kg or ppb wet weight) in serum samples from JM and GM.

PCB congener	JM	GM
28	1.82	3.47
52	1.22	1.60
101	nd	0.33
138	nd	0.22
153	0.17	0.23
180	0.16	0.44
Sum	3.37	6.28

nd, not detected.

There are several remarkable findings in this tragic story. For two persons who are not blood relatives to both develop two relatively rare cancers of the same type (thyroid and melanoma) by chance is extraordinarily unlikely. Malignant melanoma is the cancer for which there is the strongest evidence for causation by PCBs. This is reflected in the recent report from the International Agency for Research on Cancer, which declared PCBs to be Group 1, known human carcinogen, based primarily of occupational studies (24). Although the route of occupational exposure is uncertain in these reports, inhalation is certainly a major component.

Thyroid cancer has been reported in rats exposed to commercial PCB mixtures (32, 33). An elevation in lung cancer has been reported in one occupational cohort after control for other factors (34). Animal studies have shown that exposure of mice to Kanechlor-400 (a Japanese PCB product) resulted in various kinds of lung neoplasms (35). JM was a non-smoker living in an area where radon is not a major problem, and it is likely that his lung cancer was also a consequence of inhaling PCBs.

The pattern of PCB congeners found in the serum sample is striking. In the general population, PCB 153, 138, and 180 are found at much higher concentrations than PCBs 28 and 52. However because PCBs 28 and 52 have fewer chlorines, are much more volatile. In the 2003–04 NHANES, mean concentrations of PCB 28 in adults over 20 was 0.031 and the 95th percentile was 0.067 ppb. For PCB 52, the mean concentration was 0.016 and the 95th percentile was 0.043 ppb. Hence, the concentrations of both congeners are two orders of magnitude higher in both JM and GM. For PCB 153, the levels in both JM and GM are within the background concentrations found among the individuals in the 2003–2004 NHANES (mean, 0.148 ppb, 95th percentile, 0.671 ppb). This pattern of PCBs in serum alone is convincing evidence that the major route of exposure for both JM and GM was inhalation of volatile PCBs.

There is other evidence consistent with the conclusion that lower chlorinated, more volatile PCBs are

carcinogenic. Although those congeners with fewer chlorines are more rapidly metabolized, they generate hydroxylated and other metabolic progeny that exhibit genotoxicity (36) and oxidative stress (29). Maddox et al. (37) demonstrated a non-significant two-fold increase in spontaneous mutations induced by PCB 3 (4 monochloro biphenyl) and 4-OH-PCB 3 in rat lung. Xie et al. (38) showed that PCB 3 is converted to quinones which are very efficient in inducing gene mutations and chromosomal breaks.

Studies using hospitalization diagnoses to assess diseases from inhalation of PCBs

My colleagues and I have performed a series of studies using New York State (NYS) hospitalization data to examine residences near hazardous waste sites containing identified chemicals, particularly PCBs. In NYS, the diseases diagnosed in every patient admitted as an inpatient to a state-regulated hospital (all except federal hospitals like Veterans' Administration and Indian Health Services) must be reported to the Department of Health upon discharge. The data available to us include sex, age, race, method of payment and zip code of residence, as well as up to 15 different disease diagnoses. The data are limited in that we do not know names or street addresses, and do not have any information about personal habits. We do have access to behavioral characteristics by county from the Behavioral Risk Factor Surveillance System (BRFSS), and we have information on median household income and population density by zip code from the US Census. We have matched rates of hospitalization for specific diseases to residence in zip codes that either contain or do not contain a hazardous waste site. The Department of Environmental Conservation lists 814 such sites in NYS and identifies those containing PCBs. Our hypothesis behind these studies is that living near a PCB-contaminated site increases exposure, and that such exposure must be primarily by inhalation. There is no reason to assume that dietary exposure would be different depending upon where you live, and it is unlikely that most people are going to have significant dermal exposure.

There are some important limitations in ecologic studies of this sort, particularly with regards socioeconomic status (SES). Poverty is well known to be an important risk for disease, but we adjust for this the best we can using the BRFSS, which provides some information on personal habits in the locale and census data, from which we can obtain median household income in the zip code. The exposure assessment is also very limited, being only the zip code of residence. We cannot distinguish multiple hospitalizations by one person from those of different individuals. However, despite these limitations, there are some other major strengths. For example, there are 2.5 million hospitalizations each year in NYS, and we have data from 1993 through 2008. We have used results of these studies to generate hypotheses, which we then tested in smaller populations wherein we have better exposure assessment.

Cardiovascular disease

Sergeev and Carpenter (39) examined rates of hospitalization for coronary heart disease and myocardial infarction in NYS residents living in a zip code wherein a PCB hazardous waste site was located, and compared these rates with those living in a zip code without any hazardous waste site after adjustment for age, sex, race, income, and health insurance coverage. They found an odds ratio (OR) of 1.15 (95% confidence interval=1.03–1.29) for coronary heart disease and an OR of 1.20 (1.03–1.39) for myocardial infarction. They then examined a sub-set of the PCB zip codes, that being those along the 200 miles of the contaminated Hudson River. Average income is higher in these zip codes, and BRFSS data show more exercise, less smoking, and greater consumption of fruits and vegetables in these counties than in the rest of NYS. Despite living a healthier life style, the ORs for coronary heart disease and myocardial infarction in these zip codes were 1.36 (1.19–1.56) and OR=1.39 (1.19–1.63), respectively. Thus, living in a zip code containing a PCB hazardous waste site (either a landfill or a contaminated body of water) is associated with an increased risk of coronary heart disease and myocardial infarction, and this is unlikely due to inadequate adjustment for socio-economic status because the elevations in ORs are even higher along the Hudson.

Strokes ('brain attacks') are closely related to myocardial infarctions ('heart attacks'). Shcherbatykh et al. (40) used the same hospitalization records for stroke. They found significant elevations for ischemic stroke for individuals living in PCB-contaminated zip code (OR=1.17, 1.04–1.39) and a slightly greater elevation for individuals living along the Hudson River (OR=1.20, 1.10–1.32) as compared with zip codes without any hazardous waste site.

The above ecologic studies support the hypothesis that exposure to PCBs increases the risk of cardiovascular disease. In order to test this hypothesis, we performed

studies in two PCB-exposed populations where we have measured serum PCB concentrations. We suspect that the route of exposure for those individuals living near PCB hazardous waste sites is inhalation of lower chlorine congeners which are not very persistent. Hence, it is not clear whether the associations seen with measurement of total serum PCBs will give exactly the same results.

Goncharov et al. (41) determined self-reported rates of cardiovascular disease among the Mohawks at Akwesasne, a Native American group living at the US-Canadian border, in relation to measured serum PCBs and serum lipids. They found significantly elevated risk of self-reported cardiovascular disease, but found this to be an indirect effect via an elevation in serum cholesterol and triglycerides. Aminov et al. (42) investigated these same relationships in 575 residents of Anniston, Alabama who live near the Monsanto plant that manufactured PCBs. They also found that increased total serum PCB concentrations was significantly associated with elevated concentrations of total cholesterol and triglycerides, but found no effect on HDL or LDL cholesterol. Thus, there is a clear association between elevation in serum lipids, a major risk factor for cardiovascular disease, and more highly chlorinated PCBs, whereas the ecologic results support the conclusion that the lower chlorinated congeners are also important. At present, the relative importance of lower and higher chlorinated congeners on cardiovascular disease remains to be fully determined. Hennig et al. (43) have demonstrated pro-inflammatory changes induced by PCBs on endothelial cells, which may combine with elevations in serum lipids to increase the risk of cardiovascular disease. Ha et al. (44) have reported that there is a dose-dependent relationship between serum PCB concentrations and cardiovascular disease using data from the National Health and Nutrition Examination Survey (NHANES).

Hypertension

Hypertension is not usually considered to be an environmental disease. However, using the hospitalization data set, Huang et al. (45) reported a significantly elevated OR=1.19 (1.09–1.31) for hospitalization diagnosis of hypertension among individuals living in a zip code with a PCB hazardous waste site. They also found elevated hospitalization for hypertension (OR=1.14; 1.05–1.23) for residents living along the Hudson River.

We have determined the associations between serum PCB levels and blood pressure in 351 residents of Anniston who were not on anti-hypertensive medication. Three measurements of blood pressure were taken in individuals where serum PCBs levels had been measured. We found striking associations between rates of hypertension and serum PCB concentrations (46). After adjustment was age, sex, BMI, serum lipids, smoking and exercise the OR for lowest to highest tertile of PCB concentration was 4.09 (1.3–12.7) for clinical hypertension and 5.28 (1.0–25.8) for both systolic and diastolic hypertension. Even within the normotensive range of blood pressure, there were significant associations with total PCB concentration (47). Serum PCB concentration showed a stronger association than any other factor but age, including BMI, total lipids, sex, race, smoking, and exercise. Associations between serum PCBs and hypertension have also been reported using NHANES data (48, 49).

Diabetes

Kouznetsova et al. (50) analyzed NYS hospitalization data for adult inpatient admissions for diabetes in relation to residence in a zip code containing a PCB-contaminated waste site. Living in a PCB-contaminated zip code was associated with a 23% elevated chance of hospitalization for diabetes as compared with rates for individuals living in a zip code that did not contain a hazardous waste site (OR=1.23; 1.15–1.32), after adjustment for age, race, sex, median household income, and urban/rural residence. Living along the Hudson River was associated with an even greater elevation (OR=1.36; 1.25–1.42). As with the above diseases, the most likely exposure must have come from inhalation.

We have examined rates of physician-diagnosed diabetes in relation to serum PCB concentrations in the Mohawk population at Akwesasne. In a preliminary study, Codru et al. (51) reported that after adjustment for sex, age, BMI and smoking, individuals in the top tertile PCB concentration had a significant 3.9-fold elevated risk of diabetes (95% CI=1.5–10.6). Only two individual congeners were reported, PCBs 74 (2,4,4′,5-tetrachlorobiphenyl) and 153. When adjusted for all other contaminants in addition to the factors listed above, only PCB 74 showed a significant association. We have followed-up on this study (52) with a more complete single congener analysis and with adjustment for all other contaminants but the one under investigation. These results indicate that the only significant association with diabetes is with non- or mono-*ortho* PCB congeners that do not have dioxin-like activity. This is an important observation because these are the lower-chlorinated, volatile congeners. This provides strong support for the hypothesis developed from the hospitalization studies (50), which concluded that

the association between diabetes and living near a PCB-contaminated site is secondary to inhalation of lower chlorinated PCBs.

Discussion and conclusions

These results are consistent with the conclusion that inhalation of PCBs is not only an important route of exposure, but that it can also result in serious disease. PCB exposure is well documented to increase the risk of the diseases reviewed here, namely, cancer, cardiovascular disease, hypertension and diabetes, based on documentation that incidence of these diseases increased with serum concentrations of PCBs. However, the majority of the PCBs found in serum are the more persistent congeners, often with half-lives of a decade or more. These are the congeners found in the higher chlorinated commercial mixtures, and are the ones commonly found in animal fats, which is an important route of exposure to humans. From the point of view of research, the persistence of these higher chlorinated congeners is helpful for establishing associations because a blood sample will provide information about PCB exposure after many years have passed.

This review has focused on only four diseases, chosen because of at least some evidence for elevated risk coming from inhalation exposure. However, these are certainly not the only diseases for which exposure to PCBs is known to increase risk. PCBs are known to cause deficits in learning and memory (53, 54), and there is evidence from animal studies indicating that lowered chlorinated congeners are more neurotoxic than more highly chlorinated congeners (55). Fitzgerald et al. (56) reported decrements of verbal learning and an increase in depressive symptoms in adults living near the contaminated Hudson River, but serum concentrations are not significantly different from those in a comparison population (57). This finding is consistent with inhalation of lower chlorinated, more rapidly metabolized PCBs as the critical factor. PCBs are structurally somewhat similar to T4, and exposure has been shown to suppress thyroid function (58). PCBs also alter sex hormone function, with many congeners and hydroxylated metabolites having estrogenic activity (59). Elevated PCB exposure results in earlier puberty in girls (60) and a reduction in testosterone levels in men and boys (61, 62). PCBs suppress the immune system, leading to increased respiratory infections in children (63, 64) and elevations in cases of asthma (64, 65). PCB exposure to mothers is associated with lower birth weight of infants (66, 67). The relative role of inhalation of lower chlorinated PCBs, to date, has not been studied with regards these diseases and effects.

The PCB congeners that volatilize easily are less highly chlorinated, and most of them are much more rapidly metabolized in the human body. Some, like PCBs 28 and 52, are somewhat more persistent than others, and are frequently found at low concentrations in human serum, although the majority of those congeners with four of fewer chlorines are often not present at detectable concentrations. However, just because they are more rapidly metabolized and do not accumulate does not mean that they do not have adverse health effects. This is particularly the case if the concentrations of these lower chlorinated congeners in air are significant in places where people spend long periods of time (e.g., at home, school, or work). Under these circumstances exposure can be almost continuous, but would not be reflected in high levels of PCBs. Although the specific mechanisms whereby serum PCBs cause neurotoxicity are still uncertain, animal studies have shown that PCB, like lead, are effective in reducing long-term potentiation, an electrophysiologic marker of learning and memory (68).

The most extreme demonstration of the hazards of inhalation of PCBs is the cases of JM and GM, workers occupationally instructed to inhale PCB vapors. Both developed multiple cancers of the same type, and JM died of cancer. Their serum contained elevated concentrations of the lower chlorinated, more volatile PCBs, and only background concentrations of more highly chlorinated congeners that are less volatile.

The ecologic studies showing elevations of cardiovascular disease, hypertension, and diabetes in relation to residences near PCB-contaminated waste sites strongly suggest that inhalation is the route of exposure. However, there are significant limitations to ecologic studies, and they must be viewed as being hypothesis generating. Therefore, we have performed other investigations in defined populations where we have good exposure assessment (albeit with the limitations discussed above for lower chlorinated congeners), as well as access to medical and clinical chemistry information. These studies confirm the hypothesis that PCB exposure is associated with elevated risks of all three diseases. Thus, these studies provide support for the conclusion that inhalation of PCBs is the major cause of the elevated rates of hospitalization.

The implications of these studies are significant for several reasons. First, these results suggest that living near a PCB-contaminated waste site poses risk to health, and by extrapolation this applies also to attending a school with elevated PCBs in the air due to PCB-containing light ballasts or caulk (69–73), working in a contaminated building

(74, 75), working as a fireman around certain house fires (76), and living downwind of sewage sludge drying plants (77). Lower chlorinated PCBs are found in current retail paints, and would be expected to volatilize into room air (78). Urban areas are likely to have more hot spots with higher concentrations than in rural areas, as has been demonstrated in Chicago and Cleveland (79). Thus, many people are being unknowingly exposed to these sources via inhalation. Scientists from the USEPA have recently published a report calling for greater evaluation of health risks from inhaled PCBs (80).

PCBs are dangerous chemicals, but the danger is not restricted to dioxin-like congeners or persistent congeners. These findings reinforce the conclusion that it is imperative to find ways of removing these contaminants from the environment. Furthermore, it is important that risk assessment methodologies no longer rely only on measurement of serum PCB levels and their associations with various diseases, but rather consider air concentrations and the evidence that even low concentrations of PCBs in air constitute an important route of exposure and disease, especially if the exposure is prolonged.

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Attachment X

Increased Rate of Hospitalization for Diabetes and Residential Proximity of Hazardous Waste Sites

Increased Rate of Hospitalization for Diabetes and Residential Proximity of Hazardous Waste Sites

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Background: Epidemiologic studies suggest that there may be an association between environmental exposure to persistent organic pollutants (POPs) and diabetes.

Objective: The aim of this study was to test the hypothesis that residential proximity to POP-contaminated waste sites result in increased rates of hospitalization for diabetes.

Methods: We determined the number of hospitalized patients 25–74 years of age diagnosed with diabetes in New York State exclusive of New York City for the years 1993–2000. Descriptive statistics and negative binomial regression were used to compare diabetes hospitalization rates in individuals who resided in ZIP codes containing or abutting hazardous waste sites containing POPs ("POP" sites); ZIP codes containing hazardous waste sites but with wastes other than POPs ("other" sites); and ZIP codes without any identified hazardous waste sites ("clean" sites).

Results: Compared with the hospitalization rates for diabetes in clean sites, the rate ratios for diabetes discharges for people residing in POP sites and "other" sites, after adjustment for potential confounders were 1.23 [95% confidence interval (CI), 1.15–1.32] and 1.25 (95% CI, 1.16–1.34), respectively. In a subset of POP sites along the Hudson River, where there is higher income, less smoking, better diet, and more exercise, the rate ratio was 1.36 (95% CI, 1.26–1.47) compared to clean sites.

Conclusions: After controlling for major confounders, we found a statistically significant increase in the rate of hospitalization for diabetes among the population residing in the ZIP codes containing toxic waste sites.

Key words: behavior, diabetes mellitus, dioxins, negative binomial regression, PCBs, persistent pesticides, polychlorinated biphenyls, SES, socioeconomic status, ZIP codes. *Environ Health Perspect* 115:75–79 (2007). doi:10.1289/ehp.9223 available via *http://dx.doi.org/* [Online 18 August 2006]

Diabetes is one of the leading causes of death and one of the most costly diseases in developed countries. During 1980–2002 the number of people with physician-diagnosed diabetes in the United States increased more than 2-fold, from 5.8 million to 13.3 million. An estimated 5.2 million cases remain undiagnosed. In 2002, total direct and indirect health care costs for people with diabetes amounted to $132 billion [Centers for Disease Control and Prevention (CDC) 2003]. The prevalence of diabetes of all types was 6.3% in the United States in 2002, of which approximately 90–95% of cases is adult-onset, type 2 diabetes (CDC 2003).

Established risk factors for diabetes include age, hyperinsulinemia (a marker for insulin resistance), obesity, genetic factors, and a sedentary lifestyle [Haffner 1998; World Health Organization (WHO) 1994]. Socioeconomic status (SES) is also a risk factor, in that lower income is associated with an increased risk of obesity and sedentary life style (Brancati et al. 1996). The National Health Interview Survey (National Center for Health Statistics 2003) found race, sex, obesity, and age to be effect modifiers for the prevalence of diabetes. Diabetes generally increased more rapidly with obesity among women than among men, but there was no other consistent sex difference. African-American race was a strong risk factor for diabetes, especially among individuals of low SES. After adjustments for racial differences in age, SES, weight, and central adiposity, African Americans remained over twice as likely to have diabetes as whites [odds ratio (OR) = 2.35; 95% confidence interval (CI), 1.49–3.73; $p = 0.0003$] (Brancati et al. 1996).

In addition, recent epidemiologic evidence suggests associations between diabetes and several environmental exposures, including cigarette smoke (Will et al. 2001) and arsenic (Tsai et al. 1999). Dioxin-exposed populations have been found to be at increased risk of diabetes (Bertazzi et al. 1998; Cranmer et al. 2000; Henriksen et al. 1997), and recent studies suggest an association with polychlorinated biphenyl (PCB) exposure (Longnecker and Daniels 2001; Radikova et al. 2004). Some PCB congeners activate the aryl hydrocarbon receptor, and thus are dioxin-like in activity, whereas other congeners have different modes of action (Giesy and Kannan 1998).

Persistent organic pollutants (POPs), such as dioxins, furans, PCBs, and chlorinated pesticides, are complex mixtures of organic molecules that vary in the degree of chlorination. Whereas dioxins and furans are unintended products of incineration and by-products of some industrial processes, PCBs were manufactured and used primarily as coolants and lubricants in electrical equipment and as hydraulic fluids. The production of PCBs in the United States was discontinued in the late 1970s due to evidence that they, like dioxins and furans, persist in the environment and can cause toxic effects (Agency for Toxic Substances and Disease Registry 2000). The manufacture of most chlorinated pesticides was also stopped in developed countries in the late 1970s or early 1980s. The major routes of exposure to these compounds are ingestion of fish (especially sport fish caught in polluted lakes or rivers), meat and dairy products (Dellinger et al. 1996; Falk et al. 1999), and inhalation of contaminated air near hazardous waste sites (DeCaprio et al. 2005).

The objective of this study was to assess the potential association between residence near hazardous waste sites and hospitalization rates for diabetes among adult residents of New York State (NYS).

Materials and Methods

Study population. We used the New York Statewide Planning and Research Cooperative System (SPARCS) to obtain data on diabetes diagnosis among inpatients from 1993–2000. All hospitals regulated by and located in NYS are required to report every diagnosis (up to 15) for each inpatient, upon discharge, to the NYS Department of Health, based on the *International Classification of Disease, 9th Revision, Clinical Modification* (ICD-9-CM; National Center for Health Statistics 1980). The SPARCS data used includes patient age, sex, race, and ZIP code of current residence. New York City (NYC) maintains a separate data set and therefore was not included in this study. The SPARCS data does not identify individuals with multiple hospitalizations or patients in federally regulated hospitals, nor does it include out-of-state health care treatment received by NYS residents. It lists

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only current, not previous, residences, as previously reported (Sergeev and Carpenter 2005).

There are other important confounders for which information is not contained in the SPARCS dataset. Median household income by ZIP code was obtained from Claritas, Inc. (San Diego, CA) and was used as a proxy for SES. Rates of smoking, consumption of fruits and vegetables, and frequency of exercise (as surrogates for obesity) were obtained for counties (not ZIP codes) along the Hudson River from the Behavioral Risk Factor Surveillance System (BRFSS), as previously reported (Huang et al. 2006).

In this study we examined only two racial groups (Caucasians and African Americans) to reduce variability. These groups comprise 95% of diabetes hospitalizations in NYS exclusive of NYC. We identified all of the hospitalizations that included any of the ICD-9 codes for diabetes mellitus (ICD-9 code 250), which includes all forms of diabetes, and we studied patients 25–74 years of age.

Table 1. Distribution of characteristics in the study population.

Characteristic	Diabetic subjects No. (%)	Total person-years[a] No. (%)
Exposure	125,283 (37.3)	8,966,252 (41.6)
Total POPs	119,821 (35.7)	7,112,176 (33.0)
Hudson POPs	54,942 (45.9)[b]	2,871,808 (40.4)[b]
Other	90,448 (27.0)	5,491,372 (25.5)
Clean	125,283 (37.3)	8,966,252 (41.6)
Age (years)		
65–74	151,701 (45.2)	2,771,652 (12.8)
55–64	91,605 (27.3)	3,194,612 (14.8)
45–54	54,784 (16.3)	4,485,828 (20.8)
35–44	25,559 (7.6)	5,778,532 (26.8)
25–34	11,903 (3.5)	5,339,176 (24.8)
Race		
African American	41,543 (12.4)	1,487,372 (6.9)
Caucasian	294,009 (87.6)	20,082,428 (93.1)
Sex		
Male	164,909 (49.1)	10,517,272 (48.8)
Female	170,643 (50.9)	11,052,528 (51.2)
Income (US$)		
31,107.00–33,708.50	78,334 (23.3)	4,340,736 (20.1)
33,708.50–37,687.50	82,939 (24.7)	4,807,208 (22.3)
37,687.50–42,500.00	85,159 (25.4)	5,662,812 (26.3)
42,500.00–51,482.00 (reference)	89,120 (26.6)	6,759,044 (31.3)

[a]Sum of the population by ZIP code, 1993–2000. [b]Percentage of total POPs.

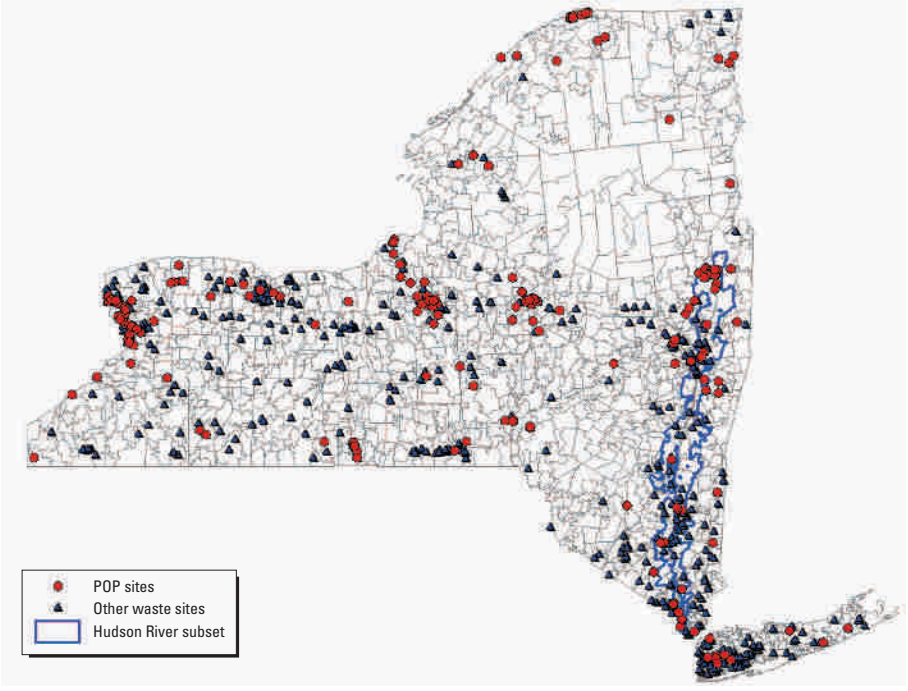

Figure 1. Map of distribution of the waste sites in NYS by ZIP code.

We restricted our regression analysis to the two middle quartile income groups (second and third quartiles), with the median household incomes ranging from $31,107.00 to $51,482.00. Our previous studies have shown that the extremes of SES show different health impacts (Huang et al. 2006; Shcherbatykh et al. 2005). Table 1 shows the characteristics of the study population. Some epidemiologic studies have demonstrated differences in rates of diabetes in urban compared to rural residents (Al-Moosa et al. 2006; Illangasekera et al. 2004; Wild et al. 2004; Zimmet et al. 1983). Therefore, we controlled for population density. Using the Census Bureau classification (U.S. Census Bureau 2002), we considered ZIP codes with > 386 persons per square mile to be urban, and those with ≤ 386 to be rural.

Assessment of exposure. Hazardous waste sites in New York were identified as previously described (Huang et al. 2006; Sergeev and Carpenter 2005; Shcherbatykh et al. 2005). The NYS Department of Environmental Conservation has identified 818 sites (state Superfund sites) that pose a potential threat to human health. The list includes 89 National Priority Sites identified by the U.S. Environmental Protection Agency (EPA 2003). In addition there are six areas of concern, highly contaminated portions of the Great Lakes and St. Lawrence River, in NYS identified by the International Joint Commission (U.S. EPA 2004). We identified the ZIP code(s) in which these sites were located, or in the case of a contaminated body of water, the ZIP code(s) that abuts the site. We classified ZIP codes into three distinct groups. "POP" ZIP codes are 194 ZIP codes that contain or abut one or more hazardous waste sites contaminated with POPs (dioxins/furans, PCBs, persistent pesticides); these include all ZIP codes that abut the six areas of concern and the PCB-contaminated portion of the Hudson River from Hudson Falls to Manhattan (NYC). There were 213 "other" ZIP codes that contain a hazardous waste site containing, for example, volatile organics and metals, but no POPs. The 995 ZIP codes that do not contain or abut any identified hazardous waste site were categorized as "clean" sites. We separately analyzed a subset of the POP sites consisting of the 78 ZIP codes in the PCB-contaminated portion of the Hudson River from Hudson Falls to NYC (30% of all people living in POP-contaminated ZIP codes). Figure 1 shows the location of the waste sites in NYS.

Statistical analysis. We calculated diabetes hospitalization rates per 100,000 as the number of discharge diagnoses of diabetes divided by the total population residing in the ZIP codes of each category. All statistical analyses were performed with SAS software (version 8.2; SAS Institute Inc., Cary, NC). We modeled the

rates of diabetes hospitalization in the different categories of ZIP code as a Poisson process. However, when Poisson regression, a log-linear model, was applied using PROC GENMOD (SAS Institute), the deviance test for the quality of fit of model and the residual plot indicated extra Poisson variation (Woodward 1999). Consequently, we used the negative binomial regression model (Cameron and Trivedi 1998). This model is a log-linear model (i.e., the mean number of discharges is determined by the linear combination of covariates):

$$\begin{aligned}\log(&\text{expected number of type 2 diabetes discharges})\\ &= \log(\text{total person-time}) + \text{intercept}\\ &\quad + b1*POP + b2*OTHER + b3*AGE6\\ &\quad + b4*AGE5 + b5*AGE4 + b6*AGE3\\ &\quad + b7*AFRICAN\text{-}AMERICAN + b8*MALE\\ &\quad + b9*INCOME1 + b10*INCOME2\\ &\quad + b11*INCOME3 + b12*URBAN, \qquad [1]\end{aligned}$$

where AGE3 is ages 35–44, AGE4 is 45–54, AGE5 is 55–65, and AGE6 is 65–74; INCOME1 is an annual median household income of $31,107.00–33,708.50, INCOME2 is $33,708.50–37,687.50, and INCOME3 is $37,687.50–42,500.00; URBAN is the ZIP codes with ≥ 386 persons/k^2; and POP and OTHER are the covariates with the value of zero or 1.

Results

Crude analysis showed an increased rate of inpatient hospital diagnosis of diabetes in individuals residing in POP sites compared with "other" waste or clean sites (Figure 2A). The rate of diabetes increased with age and was significantly higher among subjects residing in both POP and other sites compared with clean sites (Figure 2B). The relative increase, especially for POP sites, was greatest at younger ages, suggesting earlier age of onset of diabetes. These data include the full NYS population (except NYC) and not only the two middle quartiles of income that are used below for the regression analysis.

In our analysis of only the two middle quartiles of income (Table 2), after adjusting for the potential confounders (age, race, sex, income, urban/rural population density), the rate ratio (RR) was significantly elevated (23%) among residents of POP sites compared to clean sites. We also found a 25% increase in discharge rates for diabetes in the "other" sites compared with clean sites. The difference between rates in POP sites and "other" sites was not significant. As expected, the overall RRs gradually increased along with age. These results are consistent with previous studies and the national trends (National Center for Health Statistics 2003). African-Americans were 2.6 times more likely to be diagnosed with diabetes than Caucasians. We found no significant difference between the sexes in our sample. As expected, hospitalization rates varied with income, being higher in individuals with lower income. Urban/rural population density was also a significant risk factor, with rates elevated in the urban population (RR = 1.09).

We tested the quality of fit of our negative binomial model. The value of the Pearson chi-square and deviance divided by the number of degrees of freedom was close to 1, which indicates that the fit of the model was adequate.

There are other important confounders for diabetes for which information is not available in the SPARCS dataset, including rates of smoking and obesity and frequency of exercise. We previously reported a comparison of some behavioral factors in counties that abut the contaminated portion of the Hudson River compared with the rest of NYS using data obtained from the BRFSS data set (Huang et al. 2006). The results showed that Hudson River residents got more exercise and ate more fruits and vegetables, both of which are indirect measures of the incidence of obesity. The current smoking rates along the Hudson River are less than in the rest of the state. The residents of the ZIP codes along the contaminated portion of the Hudson River have higher average incomes (Huang et al. 2006); there are fewer families with incomes < $24,999, and more families with incomes > $50,000. The important conclusion is that Hudson River residents have higher incomes, live a healthier lifestyle, and smoke less than other New Yorkers.

We determined the rates of diabetes diagnosis among hospitalized residents in the 78 ZIP codes along the PCB-contaminated portion of the Hudson River compared with the "other" (non-POP) sites and the clean sites. The results of the negative binomial model for this population are shown in Table 3. The rates of diabetes diagnosis were 36% higher among Hudson River residents than those of clean sites, in spite of the fact that they have a healthier lifestyle.

Discussion

Diabetes is not one of the diseases usually thought to be secondary to environmental

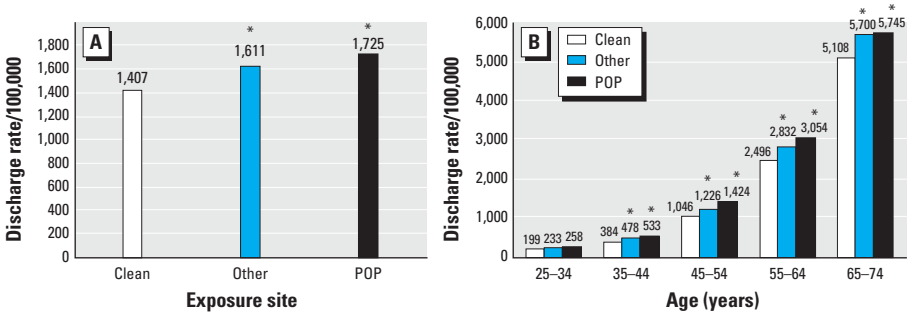

Figure 2. Crude (unadjusted) hospitalization rates for diabetes before modeling for all of the NYS population (except NYC) for ages 25–74 years in clean, "other," and POP sites (*A*) and broken down by age (*B*). The numbers above the bars indicate the rates per 100,000.
*Statistically significant compared to clean sites ($p < 0.05$).

Table 2. Results of regression analysis for diabetes discharges (POP sites).

	Coefficient	SE	RR (95% CI)	*p*-Value
Site				
POP	0.208	1.04	1.23 (1.15–1.32)	< 0.0001
Other	0.222	1.04	1.25 (1.16–1.34)	< 0.0001
Clean (reference)	0.000	1.00	1.00	
Age (years)				
65–74	3.151	1.05	23.36 (21.29–25.63)	< 0.0001
55–64	2.596	1.05	13.41 (12.22–14.71)	< 0.0001
45–54	1.774	1.05	5.89 (5.37–6.47)	< 0.0001
35–44	0.769	1.05	2.16 (1.96–2.37)	< 0.0001
25–34 (ref)	0.000	1.00	1.00	
Race				
African American	0.953	1.03	2.59 (2.45–2.75)	< 0.0001
Caucasian (reference)	0.000	1.00	1.00	
Sex				
Male	–0.023	1.03	0.98 (0.92–1.04)	< 0.4445
Female	0.000	1.00	1.00	
Income (US$)				
31,107.00–33,708.50	0.318	1.04	1.37 (1.27–1.49)	< 0.0001
33,708.50–37,687.50	0.308	1.04	1.36 (1.25–1.48)	< 0.0001
37,687.50–42,500.00	0.068	1.04	1.06 (0.99–1.16)	0.1033
42,500.00–51,482.00 (reference)	0.000	1.00	1.00	
Urban	0.090	1.03	1.09 (1.03–1.16)	0.0023
Rural (reference)	0.000	1.00	1.00	

contaminants. However, although chemical contaminants are certainly not the only, or perhaps even the major, risk factor for diabetes, they are a factor that must be considered. In a study of U.S. Air Force personnel who dropped Agent Orange in Vietnam, Henriksen et al. (1997) found a highly significant relationship between exposure to dioxin and onset and severity of diabetes in individuals with the highest exposure. This resulted in a committee of the Institute of Medicine (2001) concluding that there was suggestive evidence of an association between dioxin exposure and diabetes. Pesatori et al. (1998) and Bertazzi et al. (1998) found elevated rates of diabetes in individuals exposed to dioxin in Seveso, Italy, after an explosion of a pesticide plant in which dioxin was an unwanted by-product. Vena et al. (1998) reached a similar conclusion in a WHO study of workers exposed to dioxins during production of phenoxyacid herbicides and chlorophenol. Cranmer et al. (2000) studied individuals exposed to dioxin from the site of a former pesticide manufacturing plant in Arkansas; they found that plasma insulin concentrations were significantly higher in individuals with elevated dioxin levels, and they concluded that elevated serum dioxin levels cause insulin resistance.

Longnecker et al. (2001) studied 2,245 pregnant women, 44 of whom had diabetes. The mean serum PCB level in the women with diabetes (3.77 ppb) was 30% higher than that in the controls (2.79 ppb), and the relationship of PCB level to adjusted OR for diabetes was linear. Taking PCB levels < 2.50 ppb to have an OR of 1.0, the OR was 2.9 for PCB levels of 2.50–3.75 ppb, 4.4 for PCB levels of 3.75–5.00 ppb, and 5.1 for PCB levels of > 5.0 ppb. All values were statistically significant. Thus, this study shows a dose–response relationship. In a population-based study, Fierens et al. (2003) found, after adjustment for age and other covariates, that total toxic equivalence and concentrations of the sum of 12 marker PCBs were 62% and 39% higher, respectively, than in controls. The ORs were 5.1 (95% CI, 1.18–21.7) for dioxins, 13.3 (95% CI, 3.31–53.2) for coplanar PCBs, and 7.6 (95% CI, 1.58–36.3) for 12 marker PCBs in the upper decile of exposure. Radikova et al. (2004) reported an elevated incidence of impaired fasting glucose and incidence of diabetes in an exposed human population with serum measurements of PCBs and chlorinated pesticides.

Animal studies also show that PCB and dioxin exposure increases risk of diabetes. Nishizume et al. (1995) showed that rats given Kanechlor 400 showed depressed insulin sensitivity, which increased with the duration of PCB exposure; Kanechlor 400 also disturbed glucose and lipid metabolism and elevated serum lipids. Stahl (1995) reported that dioxin alters enzyme activity related to glucose metabolism in rat liver cells. Others have demonstrated morphologic changes of the beta cells in the pancreas after PCB exposure (Kimbrough et al. 1972; Wassermann et al. 1975). Boll et al. (1998) demonstrated that gluconeogenic enzymes in rat liver are altered after PCB exposure.

Although we are not aware of previous studies on diabetes in relation to site of residence, others have reported elevated disease in individuals living near hazardous waste sites, including rates of congenital anomalies (Dolk et al. 1998; Geschwind et al. 1992; Malik et al. 2004), low birth weight (Elliott et al. 2001), and end-stage renal disease (Hall et al. 1996). Gaffney et al. (2005) demonstrated that human serum levels of dieldrin, one of the chlorinated pesticides, decreased significantly in an inverse relation to residential distance from a contaminated site. However others (Pless-Mulloli et al. 2005) have not demonstrated any elevation in serum levels of dioxins or PCBs among individuals living near a chemical complex.

Results of the present study demonstrate a statistically significant increase in the rate of hospitalization for diabetes after controlling for major potential confounders among the adult population residing in the ZIP codes containing toxic waste sites, particularly waste sites containing POPs. However, our results do not constitute proof of cause and effect for a variety of reasons. Residence near a hazardous waste site was our only measure of exposure, and it is a very crude measure. We are aware of the methodical limitations in this study. The exposure and response are measured only at an aggregated level rather than for individuals, which introduces a possible aggregation bias. Although there are several individual risk factors for diabetes that we did not control for (e.g., diet, exercise, and smoking), they are only confounders when their frequency in the subpopulation is associated with exposure.

We do not have personal behavioral information on individuals, and there are many known risk factors for diabetes. The BRFSS data from the counties near the contaminated portion of the Hudson River indicate that, on average, individuals living there get more exercise and eat more fruits and vegetables (a surrogate measure of obesity) than other residents; but again, these are aggregated data and may not apply to the specific individuals with diabetes. The same applies to the SES data, which are based on ZIP code; the data represent the average family income in that ZIP code, and not information on the patients diagnosed with diabetes. We have no information on duration of residence in the current ZIP code, which could lead to a migration bias that can affect the validity of ecologic studies, particularly for long-latency, chronic diseases (Tong 2000). We have no control for past occupational or residential exposures that are not correlated with an existing and identified hazardous waste site. Ashton et al. (1999) demonstrated geographic variation in utilization of Veterans Affairs hospitals. This is not a factor for which we have control in this study; also, because our population consists of inpatients, this is a potential source of bias and measurement error. However, Twigger and Jessop (2000) did not find any relationship between travel time to a hospital and rates of admission for diabetes.

Despite the limitations, one might argue that if we find such clear elevations in rates of diabetes when our exposure assessment is so crude, the real relationship between disease and

Table 3. Results of regression analysis for diabetes discharges (Hudson River POP subset).

	Coefficient	SE	RR (95% CI)	*p*-Value
Site				
Hudson River POP subset	0.311	1.04	1.36 (1.26–1.47)	< 0.0001
Other	0.222	1.04	1.25 (1.16–1.35)	< 0.0001
Clean (reference)	0.000	1.00	1.00	
Age (years)				
65–74	3.158	1.05	23.53 (21.27–26.03)	< 0.0001
55–64	2.618	1.05	13.71 (12.39–15.17)	< 0.0001
45–54	1.787	1.05	5.97 (5.39–6.61)	< 0.0001
35–44	0.762	1.05	2.14 (1.93–2.38)	< 0.0001
25–34 (reference)	0.000	1.00	1.00	
Race				
African American	0.954	1.03	2.60 (2.44–2.77)	< 0.0001
Caucasian (reference)	0.000	1.00	1.00	
Sex				
Male	0.007	1.03	1.01 (0.95–1.07)	0.8271
Female	0.000	1.00	1.00	
Income (US$)				
31,107.00–33,708.50	0.332	1.05	1.39 (1.28–1.52)	< 0.0001
33,708.50–37,687.50	0.311	1.05	1.36 (1.25–1.49)	< 0.0001
37,687.50–42,500.00	0.082	1.05	1.08 (0.99–1.18)	0.0700
42,500.00–51,482.00 (reference)	0.000	1.00	1.00	
Urban	0.108	1.03	1.11 (1.04–1.19)	0.0008
Rural (reference)	0.000	1.00	1.00	

exposure is likely much stronger. Our observations suggest that residence near a hazardous waste site constituted a risk of exposure to these individuals at some time in the past, and this has led to an increased risk of developing diabetes. The risk may still exist. The most likely pathway of exposure is air transport of contaminants; contaminated particulates may be ingested, and both vapor-phase and particulate-bound contaminants may be inhaled. It is unlikely that there are different ingestion patterns of contaminated fish or other food products within specific ZIP codes of residence. Although our observations must be viewed as being hypothesis generating, they provide additional support for a relationship between exposure to environmental contaminants, especially POPs, and risk of diabetes. Further study is necessary to determine whether this is a causative relationship; if so, we need to determine the relative contribution of POPs.

Correction

In the original manuscript published online, RRs and 95% CIs in the Abstract, all values in Tables 2 and 3, and values in the text referring to these tables were underestimated. Also, the numbers and percentages for the the clean group were incorrect in Table 1. All of these have been corrected here.

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Attachment Y

Hudson River Fish Advisory Outreach Project Update

Hudson River Fish Advisory Outreach Project Update

PCBs Superfund Site
Community Advisory Group

September 19, 2013

Hudson River Fish Advisory Outreach Project Update

- Funded partners
- Consumption surveys
- Signs and materials
- 2013 outreach
- Moving forward

Funded Partners

- "River Haggie Outdoors" environmental educator

  School, library, camp and environmental education programs

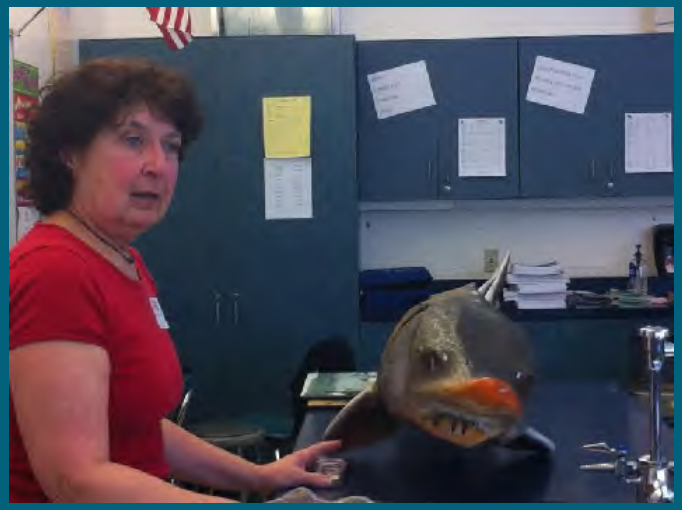
- Cornell Cooperative Extension (CCE) of Dutchess County

  In-home nutrition education program

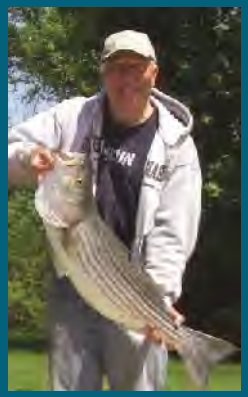
- Cornell Cooperative Extension of Rockland County

  Work with Americorps and local DOH

Funded Partners



- River Haggie Outdoors, environmental educator
- Over 4,000 schoolchildren and 2,000 adults
- Exercise with fish pictures to learn the advice



Nutrition Program Consumption Surveys

2012- CCE Dutchess County compiled surveys from Dutchess, Ulster, Greene and Columbia counties (327)

- nutrition classes and at community settings

18% ate fish or crabs they or someone they knew caught (60)

- Of local fishers, 35% checked they ate Hudson fish
- 61% including “bass/striper” eaters

Nutrition Program Consumption Survey

- Very small numbers!! – beginning data collection
- Hudson consumers - 11% of total surveys
- 62% women and 48% women under 50
- 51% ate annually
- 32% ate crabs

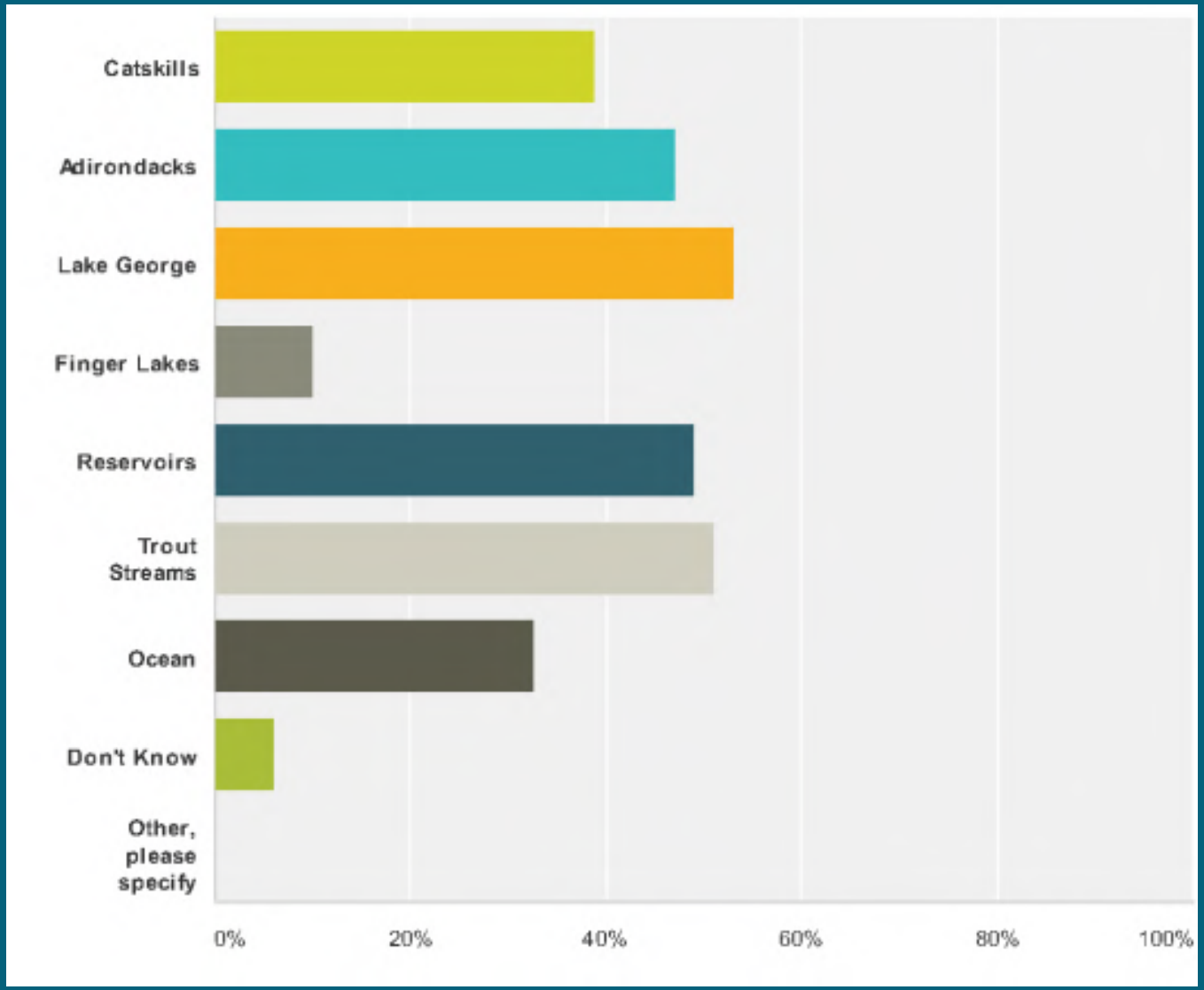
Nutrition Program Consumption Survey

- 62% were in zip code 12401 - Kingston
- 26% of clients in 12401 ate Hudson fish
- 74% aware of advice vs 52% of people eating any local fish

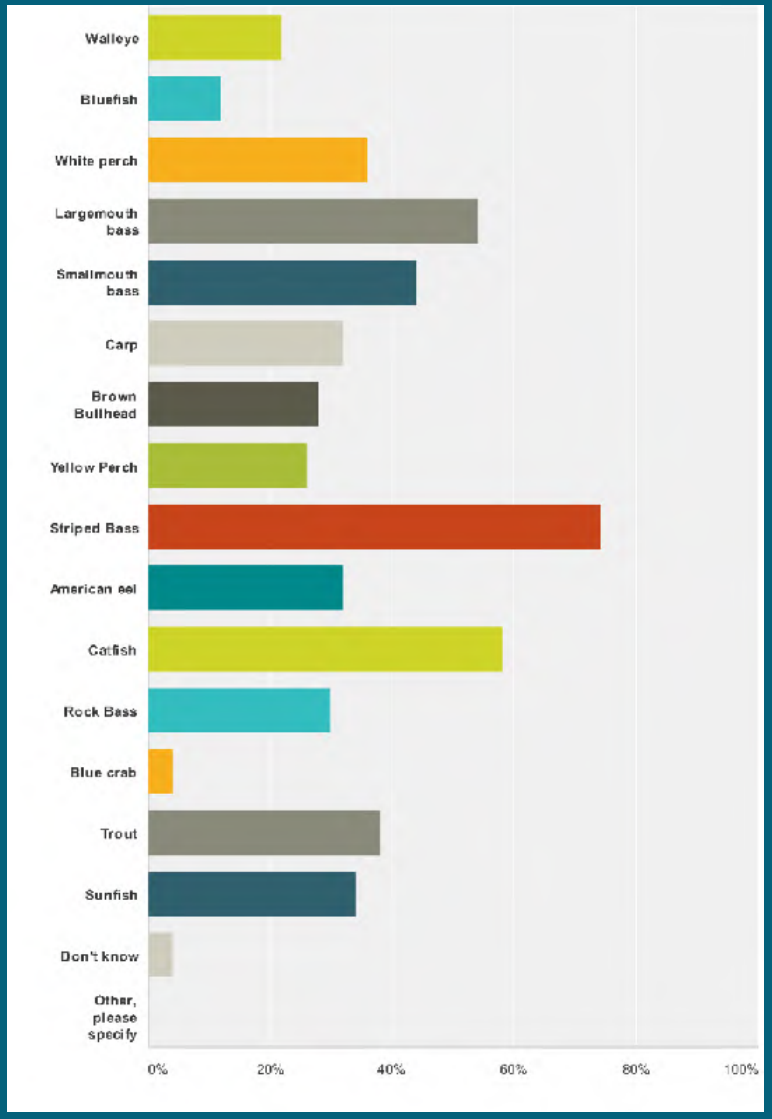
DOH Hudson Fish Consumption Surveys

- Short convenience samples at 2013 outreach events from Saratoga to Rockland County
- To develop some baselines, see patterns

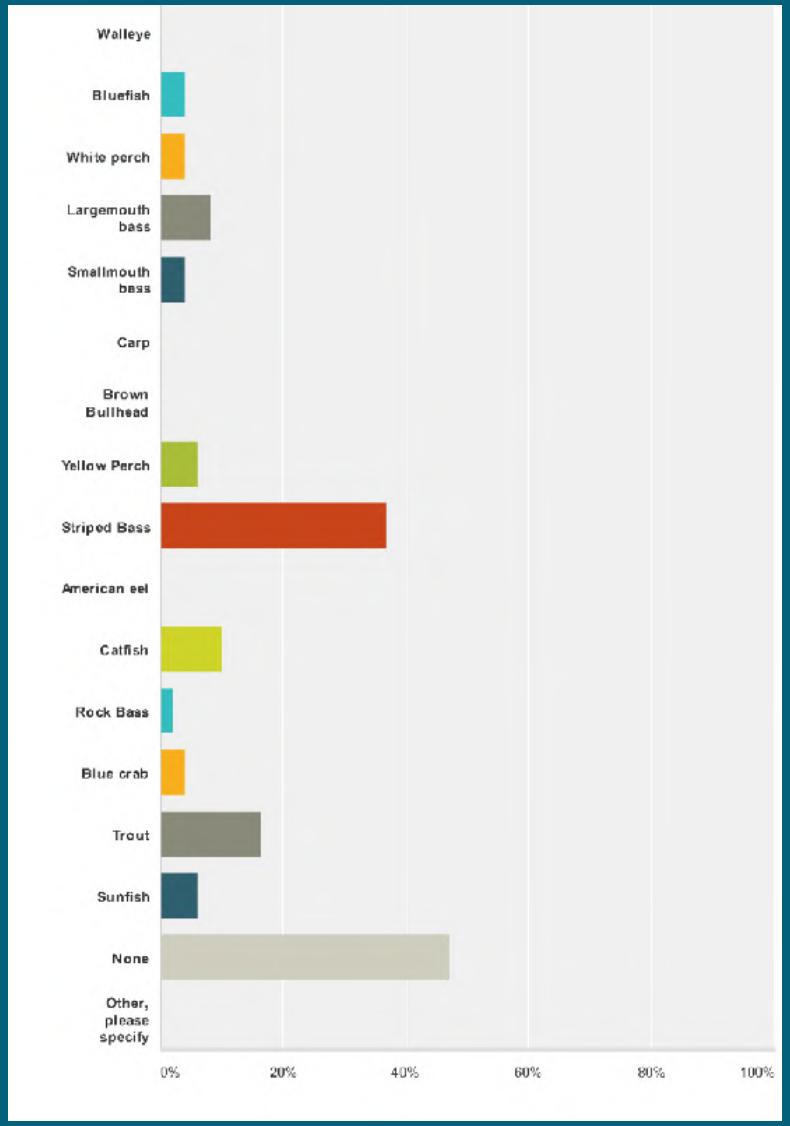
Where else do you fish?



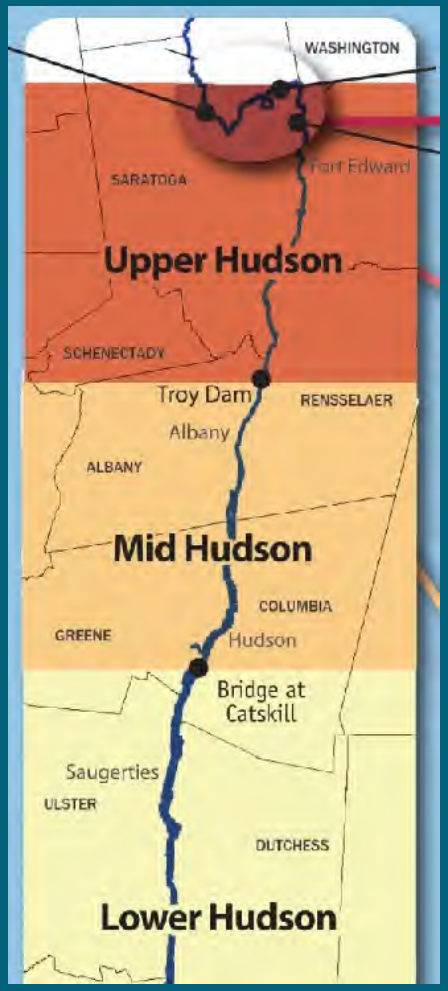
What do you catch?



What do you eat?



Hudson River: Where You Fish



Upper Hudson

From the Rt. 9 Bridge to Troy Dam

Do not eat fish from the Route 9 Bridge to the Troy Dam.

From Baker's Falls to the Troy Dam, New York's State Department of Environmental Conservation's "catch and release" regulations apply.

Take No Fish. Eat No Fish.

Mid Hudson

From Troy Dam to Bridge at Catskill

Eat up to one meal a month:

Alewife

Rock bass

Blueback herring

Yellow perch

Do not eat other fish from the Mid Hudson including striped bass

Hudson River: What You Catch (Men over 15 and Women over 50)

Lower Hudson

From Bridge at Catskill to the NYC Battery

Don't eat:


White catfish


Channel catfish


American eel*


Gizzard shad

Eat up to one meal a month:


Striped bass


White perch


Carp


Walleye


Bluefish


Brown bullhead


Smallmouth bass


Largemouth bass


Rainbow smelt


Goldfish


Atlantic needlefish

Eat up to six crabs a week:

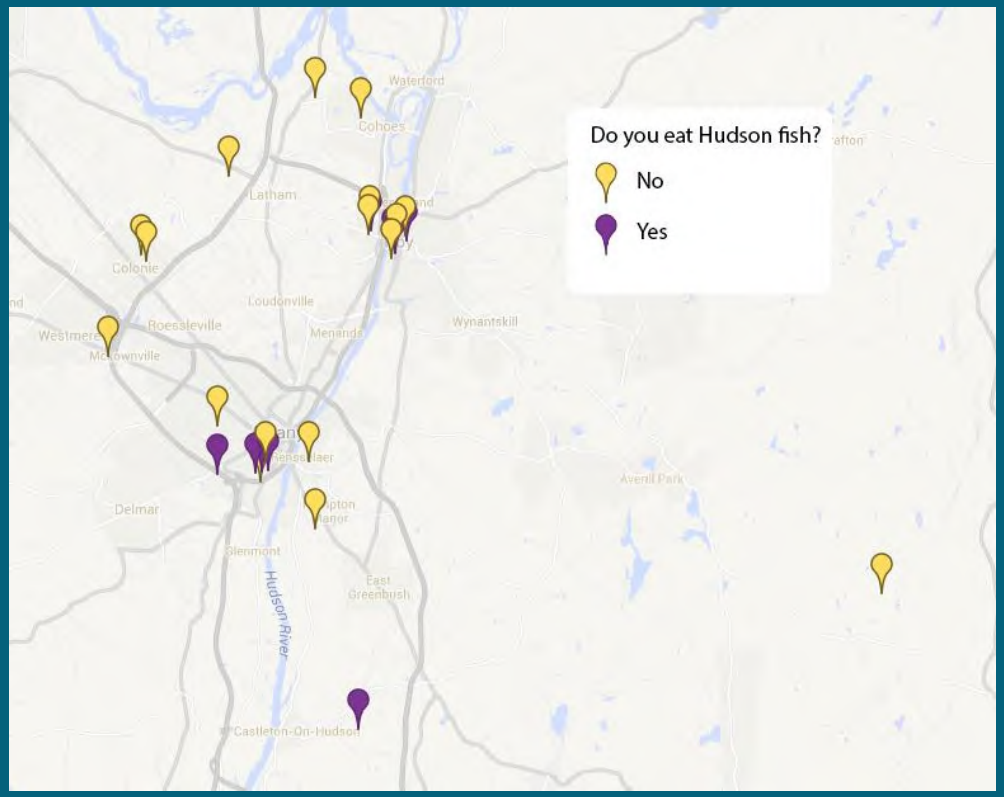

Blue crab
Do not eat the tomalley (green stuff, mustard) or reuse cooking water

Eat up to four meals a month:

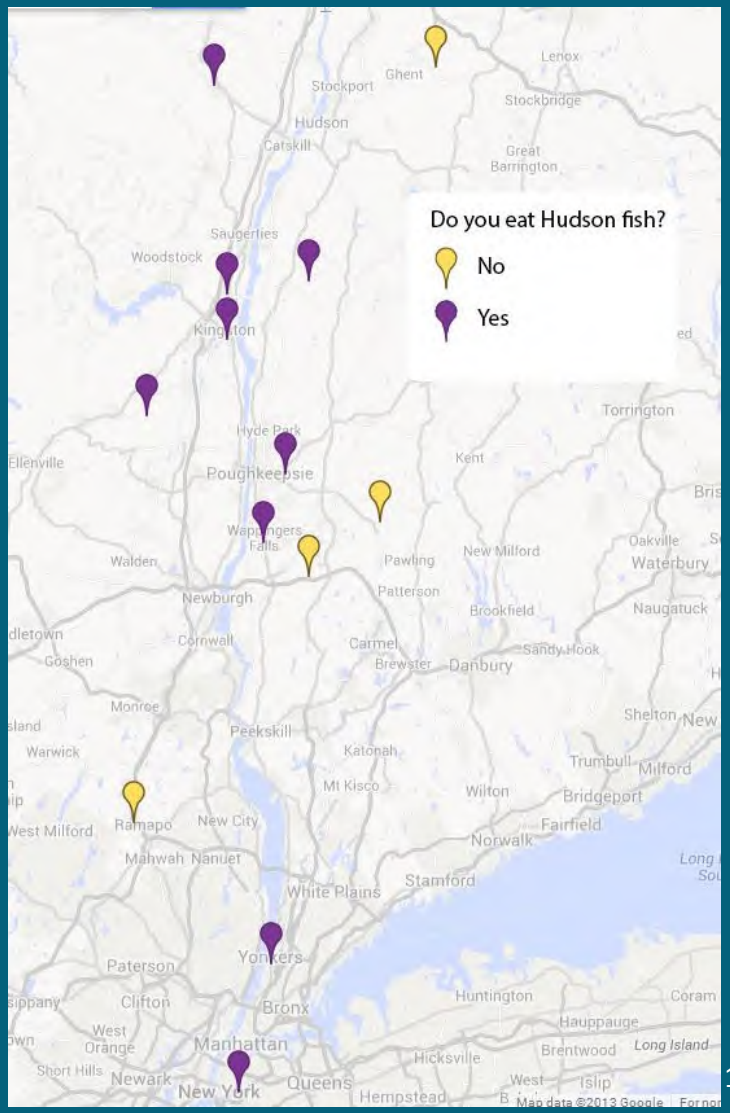
All other species

**DEC regulations prohibit taking American eel for food from the Hudson River*

Eating Hudson fish



Capital District



Lower Hudson

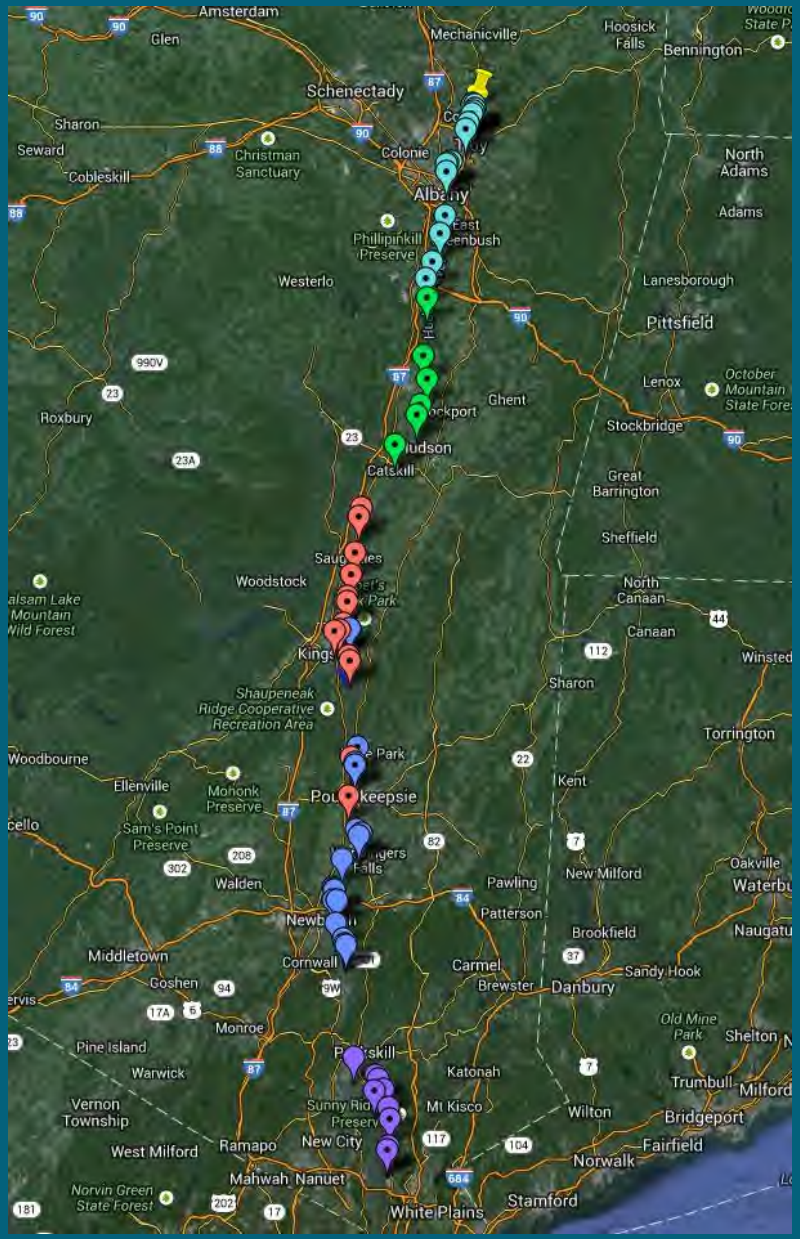
Signs South of Troy

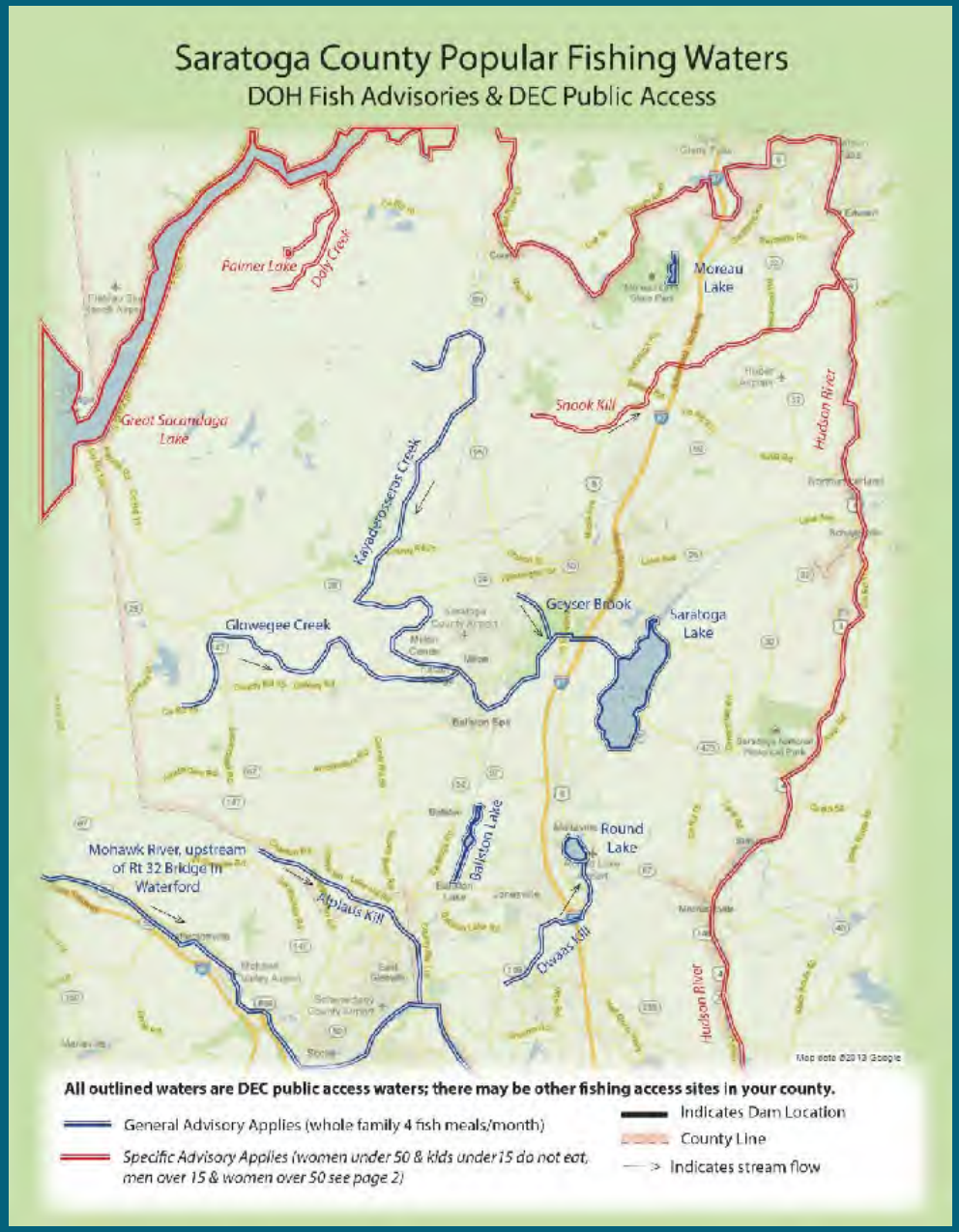
north of the Rip Van Winkle Bridge at Catskill

south of the Rip Van Winkle Bridge at Catskill

Advisory Sign Reconnaissance

Advisory Sign Reconnaissance




Saratoga County Popular Fishing Waters
DOH Fish Advisories & DEC Public Access
Palmer Lake
Dolly Creek
Moreau Lake
Great Sacandaga Lake
Snook Kill
Hudson River
Kayaderosseras Creek
Geyser Brook
Saratoga Lake
Glowegee Creek
Ballston Spa
Ballston Lake
Round Lake
Mohawk River, upstream of Rt 32 Bridge in Waterford
Alplaus Kill
Dwaas Kill
Hudson River
All outlined waters are DEC public access waters; there may be other fishing access sites in your county.
General Advisory Applies (whole family 4 fish meals/month)
Specific Advisory Applies (women under 50 & kids under 15 do not eat, men over 15 & women over 50 see page 2)
Indicates Dam Location
County Line
Indicates stream flow

County Maps

Material Distribution

- DEC includes order form with licensing information

January - August

12,000 coloring books

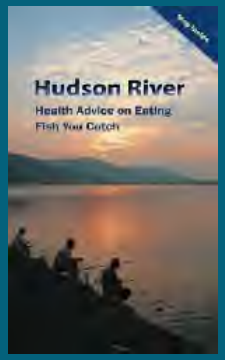
5,000 angler cards

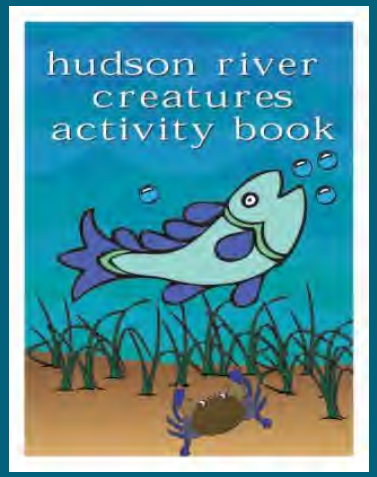
500 posters

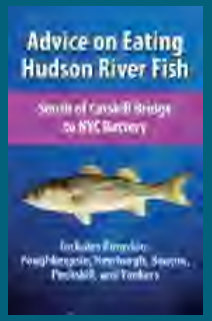
6,000 brochures

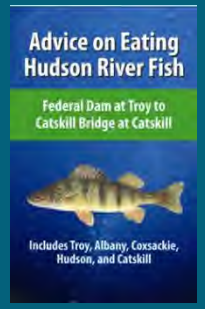
5,500 magnets

2,000 Northern Hudson





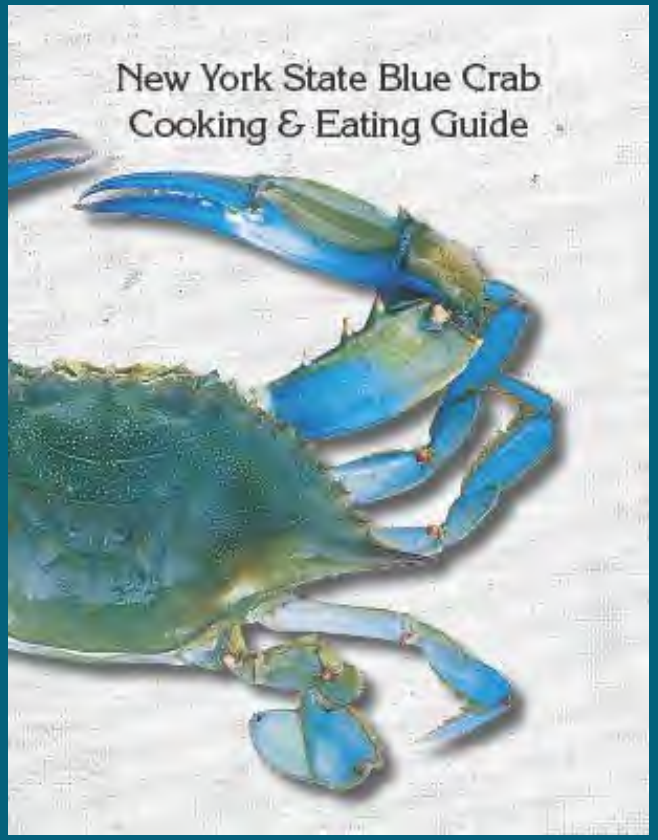




New Materials: Crab Card

- No license required to harvest crabs

- In the nutrition survey, 32% of the Hudson fish eaters ate crabs
- 25% ate the tomalley



Downstate Less Aware

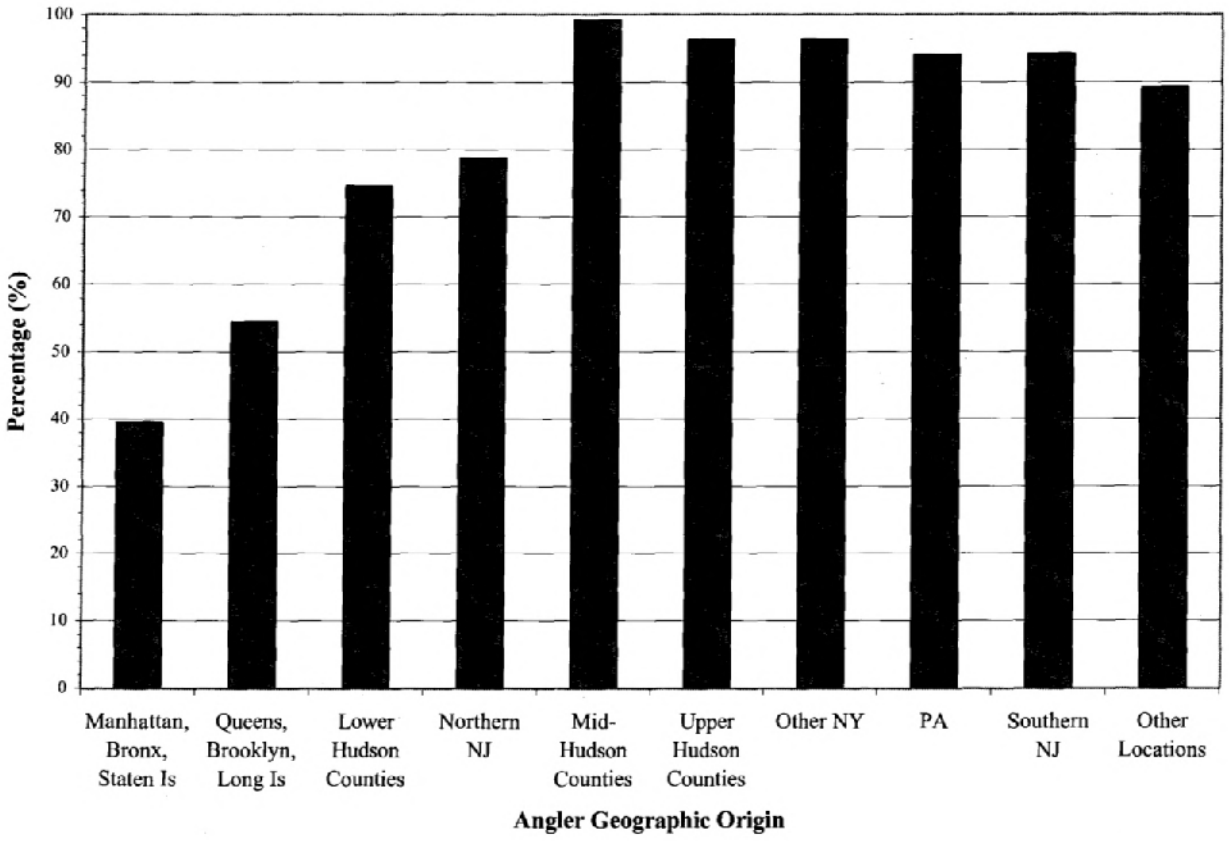


Figure 3.7-7

Percentage of anglers by residence aware of health warnings about eating fish from the Hudson River in 2001.

NYSDEC Normandeau Creel Survey

“Hooked on our Waters”

Forum in Manhattan
Saturday October 19th

Cosponsor - NY-NJ Harbor & Estuary Program

- NYS Department of Environmental Conservation - I FISH NY & Hudson River Estuary Program
- New York City Health Department
- Hudson River Fishermen’s Association
- New York Harbor School & Harbor Foundation
- NY/NJ Baykeeper
- NYC Watertrail Association
- Mount Sinai Medical Center
- NYC Parks
- Hudson River Park Trust

2013 Outreach Venues

- Tech Valley High School, Albany
- PCB Forum, Poughkeepsie
- Hudson River Boat and Yacht Club Association
- Hudson River Fishermen's Assn. Family Fishing Derby
- Saint Peter's Church, Yonkers
- Troy River Fest
- Clearwater festival
- GE Kids Day, Albany

2013 Outreach Venues

- Saratoga County Fair
- African American Family Day, Albany
- Cardboard Boat Race, Schuylerville
- Columbia, Dutchess, and Ulster County Fairs
- Mississippi Day in Albany
- Hudson Health Plan
- Clarkstown Hunting and Fishing Day

Moving forward

- Request for Application for funded partners
- Discussion with DEC on supporting Catch and Release signage
- Brochure and signs in Chinese
- Crab card and Hudson Valley brochure
- Continue Hudson fish consumption surveys
- County maps
- Catfish
- Boat and outdoor shows

Hudson River Waterfowl Consumption Advisory

- Waterfowl between Hudson Falls and Troy have higher PCB levels than from other portions of the Hudson River
- Likely to have higher PCB levels than waterfowl from other areas of the state.
- Advise to harvest waterfowl from other locations on the Hudson River or in other areas of New York State
  - Particularly in the early season when many of the available birds are likely to be resident waterfowl (i.e., non-migratory).
- Advice for the state: Eat up to two meals per month of wild waterfowl, remove skin and fat. Do not eat mergansers.

Support the Project

- Distribute materials
- Link to our website
- Invite us to events, or to talk to staff or membership

Regina Keenan 518-402-7530

rmk05@health.state.ny.us

Audrey Van Genechten

agl02@health.state.ny.us

hrfa@health.state.ny.us

Attachment Z

Community Advisory Group
(CAG) Meeting

Sept 19, 2013

Community Advisory Group (CAG) Meeting
Hudson River PCBs Superfund Site
Meeting Summary
Thursday September 19
1:00 PM - 3:30 PM

Fort Edward Fire Hall, Fort Edward, NY

CAG Members and Alternates Attending: Manna Jo Greene, Abigail Jones, Richard Kidwell, Bill Koebbman, Roland Mann, Althea Mullarkey, Merrilyn Pulver-Moulthrop, Andrew Squire, Lois Squire, Julie Stokes.

CAG Liaisons Attending: Danielle Adams (Ecology & Environment), John Callaghan (NYS Canal Corporation), John Davis (NYSOAG), Kevin Farrar (NYSDEC), David King (USEPA), Gary Klawinski (USEPA), David Kluesner (EPA), Deanna Ripstein (NYSDOH), Larisa Romanowski (USEPA).

Others Attending: Kathleen Bartholomay (Thomson-Clarks Mills Residents Committee for a Heritage Corridor Park), Jim Caird (Cashman Dredging), Michael Cheplowitz (Ecology and Environment), William Cook (Washington County Public Safety), Peter deFur (Environmental Stewardship Concepts), Johanna Dyer (Natural Resources Defense Council), Joe Finan (Saratoga NHP), Tom Gentile (NYSDEC), Kathryn Jahn (Hudson River Natural Resource Trustees), Regina Keenan (NYSDOH), Joe Moloughney (NYS Canal Corporation), Jamie Munks (The Post-Star), Jonathan Pease (Washington County Public Safety), Bill Richmond (Behan Communications), Lewis Steele (Thomson-Clarks Mills Residents Committee for a Heritage Corridor Park), Audrey Van Genechten (NYSDOH), John Vetter (Ecology and Environment), Randi Walker (NYSDEC).

Facilitators: Patrick Field, Eric Roberts

Members Absent: David Adams, Cecil Corbin-Mark, Darlene DeVoe, Rich Elder, Mark Fitzsimmons, Richard Fuller, Brian Gilchrist, Robert Goldman, Robert Goldstein, Gil Hawkins, Christine Hoffer, Jeffrey Kellog, Edward Kinowski, Aaron Mair, David Mathis, Thomas Richardson, Sharon Ruggi.

Next Meeting: The next meeting is scheduled for December 5, 2013.

Action Items

EPA

- Notify the CAG and provide clarification of safety issues and plans for addressing work in CU 60 at next meeting.

Dr. deFur

- Provide the Hudson CAG with the toxicology literature review report.

CAG Administrative Committee

- Plan the next CAG meeting

CBI

- Create a CAG member contact sheet for distribution to the CAG members.
- Obtain information from Mr. Kluesner about using Google groups as a method to contact the CAG.

Welcome, Introductions, Review June 2013 Meeting Summary

The facilitators welcomed everyone to the meeting and reviewed the agenda. The CAG approved the draft June meeting summary without any revisions. All CAG handouts and presentation slides are available within one week of CAG meetings on the project website: http://www.hudsoncag.ene.com/documents.htm.

The CAG briefly discussed a mapping project led by the Historic Hudson Hoosic Rivers Partnership, a group of town supervisors, mayors, and representatives from other organizations who meet once a month to discuss and prioritize project development opportunities. The map shows the locations of potential development projects over the next 10-15 years within the Hudson River floodplain. Kevin Ferrar, NYSDEC, offered to digitize the map using DEC's large scanner. Manna Jo Greene also offered the TAG grant for help with the map project.

Project Update on 2013 Dredging Season

David King, EPA, presented an update on the 2013 Dredging Season. Key points from his presentation include:

More than 466,000 cubic yards (>110 acres) were dredged as of September 14, 2013, surpassing the 2013 dredge season goal of 350,000 cubic yards and increasing the total cubic yards dredged to date to 1.8 million. The 2013 dredging season targeted 23 CUs (CUs 49-60 and CUs 67-78); 22 CUs were either completed or active. Dredging was in progress in CU 57 to CU 59. Capping/Backfilling was complete or underway in 17 CUs. At 5.7 percent, the capping percentage remains below the maximum limit of 11 percent, not including areas where capping was unavoidable. In response to a CAG member question about the distinction between avoidable and unavoidable capping areas, Mr. King said the unavoidable percentage was 4.27 percent.

Work will continue 24 hours per day six days per week until November, provided optimal weather and river flow conditions persist. The Moreau backfill area, the Route 4 support property, and the Route 4 crew change area will move downstream as work is completed in the current CUs. Equipment demobilization and planning for 2014 will begin in November. GE will begin to submit plans for the remaining portions of the project.

Safety concerns preempted the start of dredging in CU 60, which is near the Thompson Island Dam on the east side of the river. Last winter EPA requested that GE complete a detailed safety assessment of the dredging options for this area before deciding on an approach. Dredging may be completed from the land; but if the investigation concludes that the material cannot be dredged safely, then the EPA will check to see if additional dredging can be done elsewhere.

No exceedances of the total PCB standard in water had been detected to date during the 2013 season; the PCB load at Stillwater and Waterford remained below the in-season criteria used to assess compliance. Three percent of the collected air quality samples were above the air PCB standard at the facility and river corridor. Some of these increases were near Hotspot 28. Best management practices were implemented to minimize exposure upon detection of the exceedance. In response to a member question, Mr. King clarified that a series of consecutive measurements at the same location exceeding the standard could lead to a temporary shutdown and relocation of dredging activities.

More than 800 barges have been unloaded to date. The facility was generally not operated on Sundays as unloading and processing kept pace with dredging, which resulted in very low staging piles at the facility. Between May 18 and September 16, 2013, 46 TSCA unit trains and 6 non-TSCA unit trains containing a total of approximately 442,000 tons of material were sent to disposal facilities in Oklahoma and Ohio.

GE submitted design and work plan documents to request permission to begin dredging in CUs 79-84. Although they anticipate completing CU 84 by the end of the season, they must first address the wetland habitat in the area in cooperation with the NYSDEC. GE also submitted a design and work plan for CU 97-100. It is anticipated that the most northern subunit of CU 99 and CU 100 will be completed this year. These two areas are near eagle nests and require work to be completed later in the year, after the breeding season is over and the eagles leave the nests. GE has not yet submitted a design for the land locked area.

Cultural resource investigations, habitat reconstruction, and outreach activity continue. Upcoming outreach activities will focus on the land locked area. GE was preparing to send information to residents between CU 59 to 100 to notify them of dredging below Lock 5.

CAG members had the following questions and comments after Mr. King s update. Responses from Mr. King or other EPA colleagues are *italicized*:

- A member commented on the disturbance caused by fast moving trucks on Route 4. The member suggested the public should be permitted to provide input on truck access south of Lock 5 and that citizens in Saratoga should receive information about truck traffic caused by the project. The member also noted that the GE representatives were absent at the meeting.
- A CAG member said she is receiving complaints about sediment suspended between 4 to 7 days. *Mr. King said the backfill can remain suspended for a while and that the carbon in particular may stay suspended for a longer time. He noted that attempts were made to find methods that reduce sediment suspension, albeit unsuccessfully.*
- A CAG member asked if resuspension was due to the pace of backfilling and if any phasing of the work could be done to reduce resuspension. *Mr. King said it was not due to the pace and that GE is required to backfill within 10 days of dredging. In some spots nearly 10-12 feet of backfill is required. Although they try to accommodate river activities, the amount of backfill required causes a lot of sediment resuspension.*
- A member commented that GE seems to be dredging much more rapidly, that they are submitting dredge plans for new CUs faster than before, and that GE previously stated they were maxed out and unable to do extra work in a season. The member questioned why GE could not do extra work in upstream areas that are contaminated but not included in the ROD before moving so quickly downstream. *Mr. King said GE still must submit plans to start a new CU and that they are working more quickly within the delineated dredge areas due to experience from the past years and conditions in the CUs in the run of the river*
- The safety concern at CU 60 was discussed. One member said it seemed as if GE was attempting to receive permission to not dredge in an area that is required to be dredged and asked for clarification. *Mr. King explained that the EPA requested that GE document safety issues and propose alternative methods such as dredging from the land to conduct the work safely. If dredging cannot occur here safely, then the EPA will check to see if GE can clean up a similar amount elsewhere.*
- Peter deFur asked if documents pertaining to GE s investigation of health and safety at CU 60 will be public before it is finalized. *Mr. King said if GE says they can dredge safely, then the EPA will not stop them. But, Mr. King speculated that GE would not complete the study in time for this season and it would be completed next year. The EPA can provide the safety plan to the CAG for review before EPA approves the dredging design for this area.* Another member suggested that if GE cannot complete dredging at CU 60, then the ratio of dredging completed elsewhere should be greater than 1:1.

Overview of Environmental Monitoring Results and Toxicology Literature Review Findings

Dr. Peter deFur, TAG Advisor, presented an overview of environmental monitoring results and a toxicology literature review. Key points from his presentation include:

Dr. deFur reviewed the air quality; odor, noise, light, and navigation; PCB concentrations in water; and, water discharge reports on the Hudson Dredging Data website and compared them to reports from years past. No particular trends were identified, but he noted changes in dredging practices have reduced the number and frequency of air quality exceedances over the years. No reports of odor, noise, light, or navigation exceedances were reported on the website.

No water quality standard exceedances were reported; however, measurements were recorded above zero. Neither of the concentration trends shown on the slides are statistically significant, but there appears to be an increase in the trend in the early part of the season. This increase is likely due to increased flow conditions, which increase concentration levels. In response to a question about the apparent early pattern of PCB material settling out before traveling down the river early, Dr. deFur said high flow events do not permit the PCB material to settle out. Kevin Ferrar noted that transport of PCBs downstream is not related to solids transport. Rather, PCBs in the dissolved phase more readily move down stream. Dr. deFur noted that several tributaries are also under PCB impairment and probably contribute a low level of PCBs to the Hudson River.

CAG members had the following questions and comments after Dr. deFur s update on the environmental monitoring results. Responses from Dr. deFur or EPA representatives are *italicized*:

- A member asked if noise complaints are only for dredging or if they would also include noise from trucks. *Mr. King responded that the EPA would ask GE to set up sound monitors if complaints about road noise were received and that there are noise monitoring requirements for new equipment.*
- Another member commented that truck and heavy industrial traffic on Route 4 increased dramatically due to the facility north of Schuylerville. The member noted that noise complaints from large trucks may be more likely in the land locked portion since it is mostly agricultural.
- A member suggested Dr. deFur identify which tributaries may be contributing PCBs to the Hudson River.
- A member asked where the New York state water quality standard is applied. *Kevin Farrar said the standard is applied to all waters of New York State. However, the standard was waived by EPA as part of the ROD due to lack of technology to meet the standard.*

Dr. deFur next presented the findings of the toxicology literature review. Dr. deFur noted that the literature review process involved review of 390 toxicology and health studies from the past 10 years. The review supports previous findings that PCBs are likely to be carcinogenic and cause reproductive and neurological health effects. Exposure to PCBs can threaten immune systems and developing fetuses and children are particularly sensitive to PCB exposure. PCBs in breast milk and adipose tissue are more widespread than 10 years ago. New findings suggest PCB exposure may be associated with Parkinson s disease, contribute to low IQ and increased likelihood of ADHD in young boys, and potentially alter bird song. A study of mink that consumed PCB contaminated fish from the Housatonic (in Massachusetts) and Hudson Rivers showed that PCB exposure reduced litter size and altered development of reproductive tracts in male and female mink. No new studies of PCB exposure in amphibians or turtles were located.

CAG members had the following questions and comments after Dr. deFur s update on the toxicology literature review. Responses from Dr. deFur are *italicized*:

- A member commented on the bird song study. She said this was a long-term study completed by Cornell University and the findings have major implications because bird song is required for

mating. This issue is of large concern for The Nature Conservancy, the Audubon Society, and other conservation organizations and could be a big issue economically for bird watchers.

- A member asked for clarification that there have been no new conclusions on PCB and carcinogenicity. *Dr. deFur said the new results support what the group knew in 2002—that PCBs are probably a human carcinogen—and that the classification of PCBs has not changed by the EPA or other agencies. Dianna Ripstein, NYSDOH, commented that the International Agency for Research on Cancer (IARC) recently published a report stating that PCBs are carcinogenic. The NYSDOH is reviewing the report.*

Fish Consumption Advisory Outreach Activities Update

Regina Keenan, NYSDOH, updated the CAG members on the fish consumption advisory. Main points from her presentation follow.

The NYSDOH continued working closely with their funded partners. Since 2009, the NYSDOH has reached approximately 4,000 students and 2,000 adults through a partnership with River Haggie Outdoors. A fish consumption survey conducted by another funded partner, the Cornell Cooperative Extension of Dutchess County, elucidated the consumption patterns of some low-income families. Of 327 respondents, 18 percent said they ate fish, which they or someone they know caught. Of the 18 percent who ate locally caught fish, 35% said they ate fish from the Hudson River but this percentage was 61% when it included fishermen/women who reported eating striped bass. In the survey, many respondents wrote that striped bass, which migrate to the ocean each year, are an ocean fish and not a Hudson fish.

DOH staff have begun a Hudson fishing consumption survey, which is a convenience sample conducted at outreach events they attend. This project is still in the early phase of the data collection process and about 50 have completed the survey. Data from this survey will indicate where people tend to fish, the type of fish they catch, and the fish they eat from the Hudson. Preliminary data, which Ms. Keenan stressed is from a very small sample, indicates that people are catching and eating striped bass more than other fish. The survey data also may suggest that people in the upper Hudson near the Capital District are aware they should not eat fish from the Hudson and people in the Lower Hudson near Kingston consume Hudson River fish more frequently. The NYSDOH will partner with the New York/New Jersey estuary program to raise awareness of the fish consumption advisory at an upcoming event in New York City in October.

The NYSDOH continues to work with property owners to post signs to inform anglers of the potential harm posed by eating fish from the Hudson River. There are two types of signs, one for the river north of the Rip Van Winkle Bridge and another for South of the Rip Van Winkle bridge, and both are in Spanish and English. The difference in the signs above and below the bridge is intended to reflect the different fish consumption advisories for the two river segments. Ms. Keenan said the NYSDOH is also conducting sign reconnaissance and noted that it is challenging to get municipalities and private organizations to erect the signs. To overcome the challenge and persuade municipalities to post the signs in specific locations, the NYSDOH visits the riverfront to provide municipalities with specific locations and GPS coordinates where they would suggest sign placement. This approach has been more successful than past efforts.

The NYSDOH also created new materials for distribution. County maps for the Hudson corridor counties illustrate the locations of consumption advisories on DEC public access waters, and also show DEC access waters without advisories where a family can eat the fish; these are currently in draft form. The Northern Hudson Brochure provides readers with consumption advisory information specific to Saratoga, Warren, and Washington Counties. Through an agreement with the DEC, order forms for the advisory materials are distributed with licensing information. Some bait and tackle shops have ordered the materials and are distributing them in their shops. A new Crab Card informs readers of the risks

associated with eating crabs. This information is particularly useful because no license is required to harvest crabs and 32 percent of the Hudson fishers are eating them. Furthermore, 25 percent of those who eat the crabs also eat the tomalley, the organ where PCBs tend to accumulate in greater concentrations.

Outreach activities occur at a variety of public venues. In 2013, the NYSDOH conducted outreach at county fairs, fishing and yachting association meetings, high schools, and other public festivals. The NYSDOH plans to work with Hudson Health Plan, a health provider which accepts migrant worker vouchers, to reach an audience that may potentially fish in the Hudson. While working with another migrant program through the Columbia County Health Department in 2011, the NYSDOH discovered that migrant workers in the Columbia County area are fishing on farms since they do not have time or transportation to access the Hudson River.

Moving forward, the NYSDOH will launch a request for application for funded partners. Selected partners will receive grants to conduct outreach in partnership with the NYSDOH. Brochures and signs will be produced in Chinese. The DOH will support from the DEC on Catch and Release signage from Troy to Hudson Falls. The DOH may also start conducting outreach at boat and outdoor shows in the fall, winter, and spring.

In conclusion, Ms. Keenan briefed the CAG on the DEC announcement of PCB concentrations found in waterfowl. The DEC collected approximately 200 birds from along the Hudson River and a location upstream of Hudson Falls and tested the tissue for PCBs. The findings indicate that waterfowl in and around the Hudson River from Hudson Falls to the Troy dam are likely to have greater PCB concentration levels than waterfowl from other areas of the state. The public is advised to not eat waterfowl from along the Hudson River from Hudson Falls to the Troy dam.

CAG members had the following comments and questions about the consumption advisory outreach update. Ms. Keenan s responses are *italicized*.

- In relation to the consumption surveys, Mr. King asked about data from the people in the 40 miles of river around Ft. Edward. *Ms. Keenan said they have some information and a little data from the Saratoga County fair, but the entire population in this area has yet to be surveyed. Technically it is illegal to harvest fish from there since it is designated as a catch and release area; but anecdotal information indicates that some people are taking fish from near the Peeble's Island area.*
- A CAG member applauded the effort of the DOH and asked about the grant process for funded partners. *Ms. Keenan indicated the DOH has the funds and will again request applications for local organizations to partner with them. The DOH usually does not fund one-time projects. Instead, they prefer longer commitments but may consider one-time projects this time. Ms. Keenan indicated they would like to find community partners in places they do not have current partners, such as in the dredging area, in New York City and Newburgh, or who can address consumption of specific fish species such as catfish.*

Brief Updates and CAG Business

The CAG members received a brief update from Kathryn Jahn of the Hudson River Natural Resource Trustees. Main points from her update include the following.

Four new items were posted recently on the Natural Resource Trustees website. The Hudson River Status Report from January 2013, which is based primarily on data collected between 2002 and 2008, provides an overview of the PCB contamination in the Hudson River. The Mink Modification Report outlines proposed changes to the study plan based on a 2012 pilot study. The List of Restoration Project Proposals Submitted by the Public (September 2013) includes all of the projects proposed through the restoration project proposal form and projects suggested at public meetings. The freshwater mussel restoration

planning pilot study fact sheet describes the study goals and the anticipated outputs from the investigation. Ms. Jahn welcomed feedback on the four items.

CAG members made the following comments. Ms. Jahn s responses are *italicized*.

- A CAG member said they would like to see a detailed presentation on the impacts to wildlife as well as a presentation about how the Hudson River Trustees conduct Natural Resource Damage Assessments and how they contract and work with project partners on restoration projects.
- Another CAG member expressed concern that the NRDAs are not linked to economic impacts. She described how Global Foundries needed a piece of equipment and the best way to transport it was by river; but the lack of dredging in the navigation channel prevented the shipment. The member refuted GE claims that the river is not used for economic purposes and therefore there is no need to dredge the navigation channel. The member said the economic impact of river use must be addressed before the dredging project concludes. Another member agreed with these statements.
- A member asked if restoration projects can still be proposed. *Ms. Jahn said people can still submit restoration project proposals and they can update projects they have already proposed.*

Public Comment

Kathleen Bartholomay and Lewis Steele, members of the Thomson-Clarks Mills Residents Committee for a Heritage Corridor Park, commented on their attempts to obtain information from the EPA regarding cultural resources, provide input on the clean-up process, and contact CAG members.

They expressed frustration with the Section 106 process and their ability to participate in it. They said the rate at which the EPA responded to their email requests, the quality of the information provided to them, and the format in which it was provided was not acceptable to them. They indicated that emails to the EPA went unanswered for over a month and after meeting with the EPA, they received a black and white map that was illegible and outdated. However, a better map was recently provided. Additionally, other data was provided in electronic format; but they wanted hard copies. They said the Section 106 process is supposed to enable the public to participate in advance of the dredging operations, but they had not been able to participate. They commented that they wanted the CAG to hear about their challenges participating in the process and talk with the CAG and the public about how to interact and engage in the process; and suggested that the EPA provide the CAG with monthly community engagement reports to inform the CAG about who was contacted, why they were contacted, and the result of the engagement.

In response, a CAG member commented that longevity is a challenge of this project. She said the dredging locations were identified 6-8 years ago when the Section 106 process was started and that the CAG reviewed the cultural resources information and received presentations on the issue at that time. She noted that the consultation happened long before people realized the impact it would have on their daily lives. Mr. Steele replied that he thought the Section 106 determinations were still needed for the sampling in the floodplains and shorelines and that he hopes the program will work harmoniously with the public to complete it.

Ms. Bartholomay said she hopes the contaminated material, which lies outside of CU boundaries nearest to her home, will be cleaned up during the shoreline remediation process. In response to this comment, a CAG member said they too are aware of several locations outside of the CUs they hope will be cleaned up and noted that the CAG does receive community engagement reports. Regarding the CAG member contact information, Ms. Bartholomay and Mr. Steele indicated that they would like to directly contact the CAG and requested that either CAG member contact information or a public CAG address be made available on the website.

CAG members asked the following questions or made the following comments:

- A CAG member said she also receives electronic resources from the EPA and that she was unaware there were still determinations to make. She supported adding her name to a public contact list.
- Another member said he would rather the public relay messages to the CAG through CBI staff.
- Mr. David Kluesner, EPA, said other CAGs have created Google groups that are administered by the CAG. Mr. Kluesner will send CBI information about the Google groups.

Mr. deFur requested an update on the floodplain remediation planning. Mr. King said discussion on the draft work plan continues. Technical comments were provided to GE and GE will conduct limited sampling this fall in new places where people are using the river. The EPA hopes to share the next work plan they receive with the public.

The meeting adjourned at approximately 3:30 pm.

Attachment AA

Hudson River Angler Survey

Dec, 2016

HUDSON RIVER ANGLER STUDY

A Snapshot of Current Fish Consumption Trends on the Lower Hudson River

December 2016 • Author: Michael Garcia; contributor: Jeremiah Stone

Local surveying of anglers along the Lower Hudson River was conducted to assess the current level of consumption of fish as well as the demographics of consumers. Twenty-eight percent of those surveyed reported consuming Hudson River fish. Males of color with a reported annual household income of $25,000 to $50,000 were the highest represented consumers.

INTRODUCTION

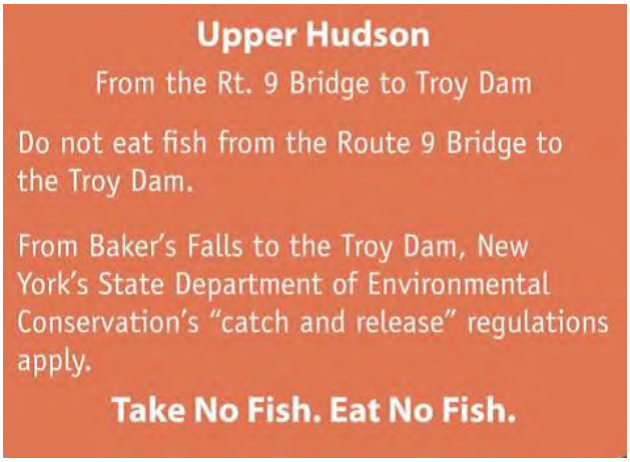
The continued presence of polychlorinated biphenyls (PCBs) in the Hudson River ecosystem has established it as the largest Superfund site in the country. PCBs contaminate every layer of the river's ecosystem—water, sediment and wildlife, including many species of edible fish that subsistence fishermen rely on for food. The New York State Department of Health (NYSDOH) is engaged in a multi-year initiative[1] to warn anglers of the dangers of eating contaminated fish from Hudson Falls to the Battery in New York City.

Fish consumption advisories includes the following:

- From Hudson Falls to Troy—New York State Department of Environmental Conservation (NYSDEC) regulations are in place—catch and release fishing only, no one can take fish home.
- In the entire Hudson River—No one should eat heavily contaminated "do not eat" species: catfish, eel, walleye and gizzard shad.
- In the entire Hudson River—Women under age 50 (childbearing years) and children under 15 should eat no fish.
- From Troy to Catskill—Women above age 50 and men can eat four species up to once a month.
- From Troy to New York City—Women above age 50 and men can eat most species up to once a month and some marine species up to once a week.

New York State fish consumption advisories are a key component of the Environmental Protection Agency's (EPA) Superfund effort to reduce the health risks to humans posed by PCB contamination[2].

NYSDOH's recommended restrictions on fish consumption are intended to ensure that the risks of cancer from eating PCB-contaminated fish do not exceed the EPA's acceptable range.



[1] Hudson River:Health Advice on Eating Fish You Catch.(Feb. 2016) http://www.health.ny.gov/publications/2794.pdf

[2] US Environmental Protection Agency: http://www.epa.gov/risk_assessment/health-risk.htm

METHODS

Over the course of 90 days during the summer of 2016 Scenic Hudson and the Sierra Club partnered to gather data on current fishing and fish consumption trends by surveying anglers along the Lower Hudson River. The surveys were meant to answer two main questions: 1) How common is fish consumption today?; and 2) What segments of the population consume the most fish from the Lower Hudson?

To ensure consistency with previous angler audits conducted on the Hudson River, Scenic Hudson and the Sierra Club utilized a pre-existing angler survey used in a similar project in 1993. The current survey was refined to ensure it effectively answered the main questions posed for this project. Specifically, survey questions were purposely narrowed to capture firsthand accounts of current fishing trends and practices during the fishing season, as well as current fish consumption among anglers and their families. A total of 150 surveys were conducted at 15 locations between Troy and Peekskill from June to August 2016. The survey targeted hook-and-line anglers, but also included anglers using trapping and netting practices. An analysis of survey responses was performed to identify the most at-risk ethnic and socioeconomic groups.

RESULTS

Among anglers surveyed, 28% reported consuming fish caught from the Lower Hudson River, with the most common species consumed being bass, catfish and blue crab. Of those respondents who consume fish, 32% reported eating them in amounts and portion size exceeding NYSDOH guidelines. (Figure 1) PCB advisories are defined in portion size and meal frequencies (i.e., one meal per month or four meals per year). One meal size is considered to be an uncooked 8-ounce fillet.

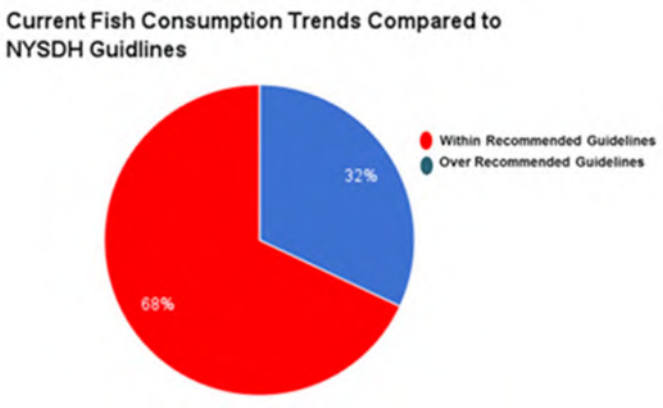


Figure 1: Current fish consumption exceeds the guidelines set by the NYSDOH

Study findings indicated that currently the typical consumer of fish from the Lower Hudson is a male of color. Of those surveyed, Latino anglers reported the highest rate of fish consumption (64%), followed by African-Americans (41%)[3]. (Figure 2)

The most affected socioeconomic group was that with annual income between $25,000 and $50,000; 53% of this group reported eating fish. Data for fish consumption on socioeconomic groups is shown in Figure 3.

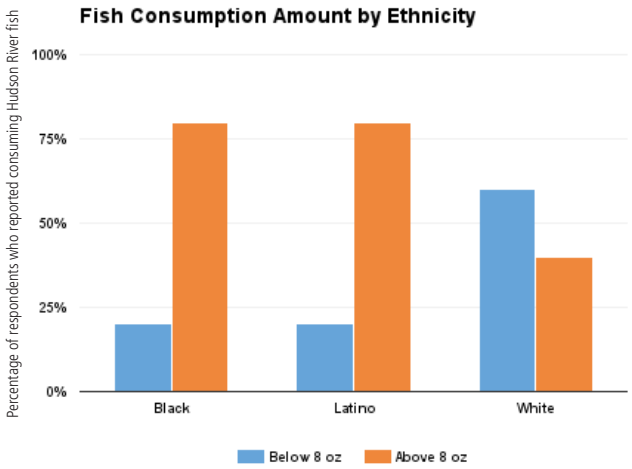


Figure 2: Current fish consumption trends by race and ethnicity when compared to recommended portion size of 8 ounces

[3] The survey size for the Asian population was too small to accurately gauge the level of consumption.

When compared to the findings of the 1993 angler report, the consumption of fish from the Hudson River may have decreased among Latino and African-American anglers. However, both remain the most at-risk ethnic groups. No findings were reported on the most affected socioeconomic groups in 1993.

The recommended fish consumption was compared among different gender and age groups (as shown in Figure 4) to actual fish consumption frequency. The NYSDOH recommends that consumption of catfish and eel be avoided, while consumption of bass, white perch and carp be limited to one meal a month for men over the age of 15 and women over the age of 50.

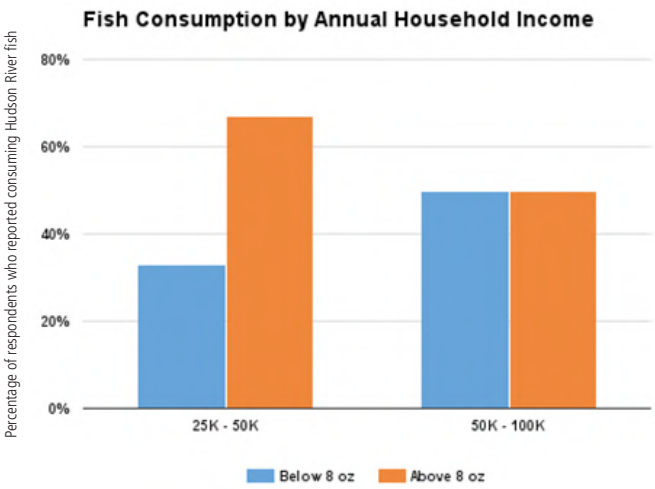


Figure 3: Over 60% of fish consumers with household incomes between 25K and 50K reported eating fish in quantities exceeding NYSDOH recommendations

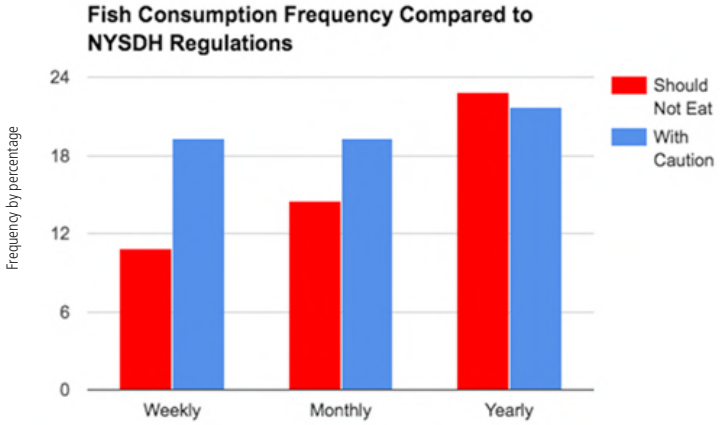


Figure 4: 32% of fish consumers are eating fish more frequently than NYSDOH recommends

DISCUSSION

Current fish consumption from the Lower Hudson River is significantly higher than recommended by the New York State Department of Health (see Figure 3), which recommends that catfish and eel be avoided and bass, white perch and carp be limited to once-a-month consumption and no greater than 8 ounces per serving. These findings are likely conservative. For instance, many Asian anglers were observed at certain locations, but language barriers limited their participation in the surveys.

Both surveyors and anglers noted difficulty in locating signage about fish consumption guidelines at many locations. At some regularly visited fishing spots, there was no signage at all warning anglers of the dangers of consuming fish from the Lower Hudson River. For this survey, there were insufficient data to correlate the rates of fish consumption to the lack of signage, but this should be considered in future angler survey efforts.

The survey also intended to characterize how many anglers shared their catch with children and women of childbearing age, but most adult anglers were reluctant to discuss and/or answer questions regarding this practice. At certain locations popular for sport fishing, surveyors observed women who were not fishing receive fish from anglers.

The commercial sale of Hudson River fish for public consumption also is an important issue that should be evaluated in a more appropriately scaled creel survey with a larger focus group.

NYS Department of Health

CONCLUSION

Similar to the results of the 1993 survey, ethnic groups such as Latino and African-American remain at relatively higher levels of risk due to higher-than-recommended levels of fish consumption by both amounts and species.

Future surveying efforts should include multilingual surveyors to increase the accuracy of current fish consumption among groups such as the Hudson Valley's growing Asian population, which was represented minimally in the 1993 survey. Surveyors in 2016 observed Asians catching (and very likely consuming) species, such as pumpkinseed, not currently on fish consumption advisories but that perhaps should be considered for inclusion.

SCENIC HUDSON
land ■ parks ■ advocacy

SIERRA CLUB
Atlantic Chapter

Re: Comments on Hudson River Draft Five Year Review

Hayley Carlock <hcarlock@scenichudson.org>

Fri 9/1/2017 4:09 PM

Inbox

To: epahrfo@outlook.com <epahrfo@outlook.com>;

1 attachments (5 MB)

FINAL_Hudson_River_FYR_Review_2017, 08-31.pdf;

As a follow up to the below comments submitted by Hudson River environmental groups, please find attached a report commissioned by Scenic Hudson and Riverkeeper that evaluates EPA's findings in the Proposed Five Year Review. This report is included as one of the attachments sent via first class mail, but we wanted to assure you were able to access this document concurrently with our comments.

Hayley Carlock

Sent from my iPhone
On Sep 1, 2017, at 3:23 PM, Hayley Carlock <hcarlock@scenichudson.org> wrote:

> Dear Director Klawinski,
>
> Please find attached a cover letter and comments on EPA's Proposed Second Five Year Review for the Hudson River Superfund Site on behalf of Riverkeeper, Scenic Hudson, Hudson Riverkeeper Fisherman's Association, Hudson River Sloop Clearwater, Natural Resources Defense Council, and Sierra Club Atlantic Chapter.
>
> Please note that due to the large file size, we are unable to send Attachments A-AA to our comments via email. We have sent the attachments and a hard copy of our comments via First Class mail.
>
> Best,
>
> **Hayley Carlock, Esq.**
> *Director of Environmental Advocacy*
> **Scenic Hudson, Inc.**
> Tel: 845 473 4440 Ext 210
> Fax: 845 473 2648
> hcarlock@scenichudson.org
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>
> **SEIZING THE MOMENT,**
> **FACING THE FUTURE:**
> Scenic Hudson's Annual Report
> highlights our recent successes
> and plans for the year ahead.
>
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>
> <9.1.17 FYR Cover Letter to EPA and Executive Summary FINAL.pdf>
>
> <2017_09_01 FINAL FYR Comments.pdf>

Hudson River PCBs Site Proposed Second Five Year Review – Technical Review

Prepared for:

Scenic Hudson, Inc.
Riverkeeper, Inc.

Prepared by:

S.S. PAPADOPULOS & ASSOCIATES, INC.
Environmental & Water-Resource Consultants

August 2017

7944 Wisconsin Avenue, Bethesda, Maryland 20814-3620 • (301) 718-8900

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REPORT

Summary Points:

- Data interpretation for fish tissue in EPA's proposed 2017 FYR ignores the very large degree of uncertainty introduced by the data transformation procedure applied to calculate fish recovery rates. Data interpretation to derive fish recovery rates must consider the very large uncertainty introduced by the data transformation procedure as well as the uncertainty inherent to fish tissue results to establish a degree of confidence in the calculated recovery rates. There is a strong likelihood that any recovery rate calculated based on the available data for fish tissue is so uncertain as to be meaningless for predicting fish recovery in the Hudson river.
- Transforming PCB concentration data from different laboratories, different analytical methods and different field studies into a PCB homologue equivalent database introduces a very large degree of uncertainty on the transformed data. EPA did not test the effect of that uncertainty on the confidence that can be attached to its fish tissue recovery rates.
- EPA's conclusion that the recovery rate for fish is on track to meet the goals of the ROD is not supported by the data with any reasonable degree of confidence or scientific certainty. EPA's procedure to calculate a recovery rate for fish in the Hudson river is too uncertain and is unreliable to support EPA's conclusion that the goals of the ROD will be achieved as previously predicted in the ROD.
- EPA's procedure to calculate recovery rates is at the upper end of the range of rates that the data could potentially support (were the transformed data not impaired by unaccounted for uncertainty).
- EPA's approach for calculating fish tissue trends included rib-out sample sets taken by GE in 2007 and 2008. Compared to the rib-in data (NYSDEC standard fillet samples), the rib-out measurements are consistently lower. Lower concentrations for these samples in 2007-2008 influence the trends calculated for the period 1995-2008 toward faster recovery rate predictions.

- Overall, fish tissue recovery rates are highly variable. The use of an arithmetic or weighted average rate is unrepresentative of this variation and deceptive when making conclusions about the protectiveness of the remedy.
- Using Aroclor-based data without transforming the data to homologue equivalents avoid the uncertainty inherent to the data transformation procedure. Calculating trends using the Aroclor data yields an average recovery rate that is different and substantially lower compared to the rate calculated using the homologue equivalent data. This demonstrates that the uncertainty introduced by the data transformation procedure is significant.
- The slowest fish tissue recovery rates hold more weight when considering the remedy effectiveness, since these species will continue to be a pathway to human exposure past the timeframe asserted by EPA. The use of an average recovery rate applied to all fish species conceals the variability in individual recovery rates by species.
- Using slightly different approaches to data interpretation results in consistently lower average recovery rates than the EPA reported 8% per year decline in fish tissue. These differently calculated average rates correspond to post dredging recovery times of about 20 years to reach 0.4 mg/kg in wet weight Tri+ PCB concentration, and 30-40 years to reach 0.2 mg/kg. This compares with the ROD predictions that the 0.4 mg/kg goal would be reached 5 years post dredging and the 0.2 mg/kg goal 16 years post dredging.

1. Introduction

This report concerns the United States Environmental Protection Agency Hudson River PCBs Superfund Site. In June 2017, EPA issued its Proposed Second Five Year Review of the Site regarding Operable Units (OUs) 1 and 2. In this Review, EPA included analyses of certain selected water column, sediment, and fish tissue PCB measurement datasets. Based on these analyses and comparison with output from models used in the Risk Assessment and Feasibility Study, EPA concluded that the selected remedy for the site, REM-3/10/Select, will be protective, and that no further remedial action is required.

The Site is formally defined as the 200-mile stretch of the Hudson River located in New York State from the Village of Hudson Falls to Battery Park in New York City. The Upper Hudson consists of the upper 40-mile stretch in between Hudson Falls and the Federal Dam at Troy, while the Lower Hudson consists of the remainder of the river. The Upper Hudson is subdivided into River Sections (RSs) 1, 2, and 3.

The original sources of PCB contamination were two General Electric facilities, located at Fort Edward and Hudson Falls. Between 1947 and 1977, these plants discharged an unknown quantity of PCBs into the river. Beginning in 1976, the New York State Department of Environmental Conservation closed fisheries and issued fish consumption advisories due to high levels of PCBs found in Hudson River fish.

River Section 1 includes Remnant Deposits that were capped following the 1984 Record of Decision for OU1. The ROD also included an interim no-action decision regarding the contaminated sediments of the Upper Hudson. Between 1989 and 2000, a multi-phase Reassessment RI/FS was conducted to reevaluate the decision concerning the sediments. This study included the development of several models to predict the transport, fate, and bioaccumulation of PCBs in sediment, water column, and fish. These models were used to forecast the recovery times for several remedial alternative plans, with active alternatives each defined by different RS-specific dredging criteria. These criteria were based upon the metric of Tri+ PCB mass per unit area (MPA) in grams per square meter, which is calculated by multiplying PCB concentration by core length and

solid specific weight (sediment density). MPA was used because it provides a more useful and appropriate measure for spatial delineation purposes than concentration, which is highly variable with sediment depth. Tri+ PCBs are defined by the EPA as the sum of all congeners in a sample containing 3 or more chlorine atoms. Tri+ PCBs are considered to pose more of a risk to human health and the environment, and are considered to more readily bioaccumulate than monochlorobiphenyls or dichlorobiphenyls. For this reason Tri+ PCBs and TPCBs were assumed to be approximately equal or interchangeable in the interpretation of fish tissue data conducted by EPA. Based on congener data obtained by EPA in the 1990s, Tri+ PCBs were found to represent 90% or more of total PCB burden in fish samples.

As part of the RI/FS, a system was developed to calculate a metric for fish tissue concentrations weighted by species and River Section length to represent a typical angler's fish diet. This weighted average metric is based upon three fish species (with weights of 0.47 for largemouth bass, 0.44 for brown bullhead, and 0.09 for yellow perch). River Sections are weighted by their proportional length in miles (0.154 for RS1, 0.125 for RS2, and 0.721 for RS3). This set of weighting factors puts emphasis on largemouth bass and brown bullhead in River Section 3, causing these fish to dominate the calculation of "average" recovery rates in the proposed FYR.

The Reassessment RI/FS was the basis for EPA's 2002 decision that the sediments posed an unacceptable risk to human health and the environment. The 2002 ROD set RS-specific criteria to be used to dredge contaminated sediments in the Upper Hudson, as well as ultimate and interim goals for fish tissue PCB concentrations. The ultimate goal of 0.05 mg/kg Tri+PCBs in wet-weight fish fillet was not attained within the 70 years period modeled in the RI/FS and was not a basis for selecting the remedy. The predicted interim goals included 0.4 mg/kg within 5 years after the completion of dredging and 0.2 mg/kg within 16 years. The Lower Hudson was not targeted for dredging, and no specific goals were set for fish tissue concentrations in that portion of the river. However, the ROD assumed that fish tissue concentrations in the Lower Hudson would decline following remediation of the Upper Hudson, and the active remedy in the Upper Hudson was intended to be protective of both the Upper and Lower

River. The Lower Hudson was predicted to recover much more quickly than the Upper Hudson since PCB concentrations in fish were lower than those in the Upper Hudson.

The 2002 ROD and 2004 Final Decision defined the dredging target areas as any areas having the following contamination levels or greater. EPA defined surface sediment as the top 12 inches of river bottom sediment.

- RS1: An MPA of 3 g/m^2 and a surface concentration of 10 mg/kg Tri+ PCBs
- RS2: An MPA of 10 g/m^2 and a surface concentration of 30 mg/kg Tri+ PCBs
- RS3: An MPA of 10 g/m^2 and a surface concentration of 30 mg/kg Tri+ PCBs (Hot Spots 36, 37, and the southern portion of 39)

The values of 3 and 10 g/m^2 were determined to be "breakpoints where a small change in MPA would mean a large increase in sediment area or mass to be remediated" (USEPA 2002, p. 64). In this way, the 3 and 10 MPA values were intended to maximize the efficiency of the remediation criteria.

Dredging was planned in two phases. These phases occurred later than the original ROD plan, which had called for dredging between 2005 and 2010.

- Phase 1, RS1: Delineated in 2005, dredged in 2009
- Phase 2, RS2 and RS3 (with a return to RS1): Delineated in 2007, dredged in 2011-2015

To delineate the areas to be dredged for both phases, sediment data were collected as part of the Sediment Sampling and Analysis Program (SSAP) between 2002 and 2005. The data were used in a kriging interpolation procedure to delineate dredge areas both horizontally and vertically. The design of this sampling program for RS2 and RS3 was intended to identify contaminated areas, not to characterize the distribution of PCBs across entire areas of the river in an unbiased manner. This has important consequences when comparing the SSAP data to 2016 OM&M post-dredging data, as discussed further below. However, EPA used these data as the primary source of pre-dredging data to assess sediment recovery, while acknowledging the bias present in the RS2 and RS3 sample design (USEPA 2017, Appendix 4). During the SSAP period, it was found that the RI/FS methods had consistently underestimated the depth of contamination in certain

areas. This led a larger volume of sediment over a smaller spatial area to be included in the dredging delineation than had been estimated in the ROD.

In total, an estimated 2.75 million cubic yards of sediment and 155,760 kg total PCBs were removed from the Upper Hudson during dredging implementation, compared to the original estimates of 2.65 million cubic yards containing 70,000 kg of total PCBs.

2. About the Data: Transformation from Aroclor to Homologue Equivalent Estimates

- Prior to the analysis of fish tissue concentration trends, EPA developed regression equations to convert Aroclor based concentration data into "homologue equivalent" data.
- A considerable amount of these data were transformed using an equation from paired samples that is a decade older than the transformed data (extrapolation).
- The process of transforming PCB concentration data introduced considerable uncertainty and systematic bias into the overall analyses, and that uncertainty is unaccounted for in EPA's estimates of fish tissue recovery rates.

Several analytical methods and laboratories were used over a period of more than two decades to generate the PCB concentration data for the Hudson river sediment. The analytical methods have included M8082, for Aroclor measurement; M1668, mGBM and NYSDEC M91-11 for specific congener measurements; and M680, for homologue measurement. M8082 was the method used to analyze the bulk of the data used by EPA in the 2017 Proposed FYR. This method is known to result in inaccuracy, due to its neglect of overlap in congener content among Aroclor mixtures ("double counting"). Different labs have different ways of reducing double-counting, leading to increased uncertainty. In addition, a proportion of the constituents of Aroclor mixtures will change over time in response to environmental exposure. Compositional changes for PCBs in sediment and fish tissue occur by dechlorination, which leaves behind lighter congeners than were originally present, and/or volatilization and dissolution, which leaves behind a mixture enriched with heavier congeners. M8082 assumes that the Aroclor mixtures in environmental samples remain as for the original PCB product, leading to inaccuracy in analytical results and adding uncertainty to data interpretation.

EPA used a regression procedure to convert all fish tissue $TPCB_{Aroclor}$ results into TPCB "Homologue Equivalent," or $TPCB_{HE}$, values (this is illustrated in Table A5-20 in the 2017 FYR). It was these transformed data that were plotted in the 2017 Proposed FYR fish tissue trend analyses to support a weighted average 8% recovery rate for fish tissue Tri+ PCB concentrations. For each subset of data (by time period and laboratory)

between 1990 and 2013, EPA utilized the existence of paired samples that had been measured with both M8082 and a homologue or congener method. A separate regression was performed on each of these matched pair sets, and the geometric mean was used as the estimated proportionality factor to transform the broader subset of data from $TPCB_{Aroclor}$ to $TPCB_{HE}$. Regression equations that had been calculated for use in the ROD for data prior to 1998 were re-used without modification for the 2017 Proposed FYR. Uncertainty in the geometric mean was estimated using a bootstrap analysis, and the use of an adjustment factor was found to be statistically justified via a Wilcoxon signed rank test. However, the uncertainty in the geometric mean was not carried over to the next step in the analyses, namely the calculation of fish tissue trends. Rather, the analyses of fish tissue trends assumed that $TPCB_{HE}$ transformed values were measured data, and uncertainty in the percent rate of decline was calculated by measuring the standard error of the coefficient. The data transformation procedure carries a very large degree of uncertainty, and this uncertainty is unaccounted for in EPA's estimates of recovery rates.

Uncertainty due to extrapolation is also an issue for the 2017 interpretation, particularly regarding data collected by NYSDEC and analyzed by Mississippi State Chemical Laboratories. For this data subset, paired samples were only available from 1999-2000; the regression factor resulting from these paired samples was extrapolated onto data collected a decade into the future, from 2001-2011 (EPA 2017, Table A5-20). The extrapolated data included in the 2017 fish tissue analyses (n=3412) represent about 36% of all data included in the 2017 fish tissue analyses (n=9387). This percentage is higher for those species and River Section combinations with data limited to a timeframe within 2000-2011.

3. 2017 Proposed Five Year Review Fish Tissue Trend Analysis

- EPA used specific criteria for data to be included in the fish tissue trend analyses. This included the use of inconsistent rib-out data collected by GE in 2007 and 2008.
- EPA chose a lipid normalized approach as the most conservative method for examining fish tissue trends.
- Based on an analysis that excluded about 50% of the total data, EPA asserted transforming the data from Aroclor based to homologue equivalent measurements had virtually no effect on fish tissue trends.

The goal of the 2017 analysis was to estimate a recovery rate for Tri+ PCB concentrations in fish tissue over time. EPA considered data from periods of disturbance to Monitored Natural Attenuation unusable for the assessment. For this reason, EPA excluded data prior to 1995 (to avoid the effects of the Allen Mill event and to avoid uncertainty due to noncomparable historical analytical methods), as well as data collected after 2008 (to exclude the effects of dredging, which began in 2009). EPA did not include 2016 data because concentrations had not had enough time to reach equilibrium following disturbance and resuspension due to dredging.

EPA used three techniques to examine fish tissue concentrations: a wet weight basis, a lipid normalized basis, and a lipid restricted basis. Fish tissue concentrations on a wet weight basis are reported in mg PCBs/kg fish tissue. Lipid normalized concentrations are wet weight concentrations that have been divided by the lipid content of the fish and are reported in mg PCBs per kg of lipid. This method controls for the effect of changes in lipid content on PCB concentration, but assumes that PCB concentration and lipid content are perfectly correlated, which is never the case. This method makes datasets to appear comparable across time. Since a general decrease in the lipid content of fish was observed across the time period represented by the data, lipid normalized trends show slower rates of decline than wet weight concentrations. The lipid restricted analysis attempted to control for the non-linear relationship between lipid content and PCB concentration by analyzing fish in groups of similar lipid content. This method, while sound in theory, reduced sample sizes to unprofitably small numbers in practice. EPA

therefore chose the lipid normalized method as the means of estimating trends in fish tissue concentrations.

EPA examined two groups encompassing eight species of fish: sport fish, including brown bullhead, largemouth bass, smallmouth bass, striped bass, white perch, and yellow perch; and forage fish, including pumpkinseed and spottail shiner. Fish tissue data used in the 2017 analysis included NYSDEC data from 1995 to 2006 and GE data from 2004 to 2008. Data through 2016 were plotted, but not included in the trendlines.

Throughout the period, NYSDEC prepared standard fillet samples for sport fish and whole body composite samples for forage fish. GE generally followed the same procedures, but beginning in 2007, GE prepared fillet samples by removing the rib from the fillet, creating a dataset of "rib out" samples. This sampling method is inconsistent with that used by NYSDEC. In 2014 a special study was conducted to test the usability of the rib out data. The test used was: "If the margin of error between rib-on and rib-off measurements is less than 20% of the average of lipid normalized PCB concentrations with a 95% level of confidence, then the measurements are considered interchangeable" (USEPA 2015). The study used paired samples (two samples from the same fish) from largemouth and smallmouth bass ("black bass") collected specifically for the study. Wet weight rib out measurements were found to be different by a factor of two or more from rib in measurements and were deemed not usable; lipid normalized paired samples were found to have an average difference of less than 20%, but the difference for individual paired samples could be up to 75%. Importantly, the NYSDEC standard fillet results were found to be consistently greater than for the rib out fillet measurements. The measurements differed by a factor of two in a quarter of the cases. Despite this, EPA concluded that the 2007-2008 GE lipid normalized data are comparable to prior standard samples. These two years of data were included in the lipid normalized trends presented by EPA that were averaged to yield an 8% recovery rate for Tri+PCBs in fish. Yet again, the procedure to justify the use of the rib out dataset carries a large uncertainty and the effect of this uncertainty on trends was not evaluated.

EPA plotted $TPCB_{HE}$ transformed values vs year for each of the eight species in RSs 1-7 (with RSs 1-3 representing the Upper Hudson and RSs 4-7 representing the

Lower Hudson). For species/RS combinations where records were insufficient, a trendline was not calculated. This applied to spottail shiner, striped bass, and white perch in the Upper Hudson sections, spottail shiner in RSs 5-7, and smallmouth bass, largemouth bass, brown bullhead, and yellow perch in RS 7.

EPA then took the weighted average of the recovery rates calculated for sport fish species in the Upper Hudson; three species are represented in this weighted average 8% rate, which EPA found to be consistent with a first order half-life value of 8 years and consistent with model output for rates of decline in PCB concentration. EPA chose to include three sport fish species since these proportions of species are considered to represent the typical anger's fish diet, and therefore the pathway for human consumption and exposure to the contaminants. The so-called "Frankenfish" approach in weighting the average by species and River Section length was developed in the mid-1990s and it is unclear whether these proportions are still representative of the population's diet, especially in light of demographic changes. Different groups within the population may consume different species or use different preparation techniques than the EPA analyses assume.

In an effort to test the effect of data transformation into homologue equivalent measurements on the estimated decay rate, EPA calculated average decay rates by species and river section and plotted these against River Mile for both $TPCB_{HE}$ and $TPCB_{Aroclor}$ measurements. However, about 50% of the samples used in the $TPCB_{HE}$ trend analyses were eliminated for this step by selecting only those species-River Section combinations with at least 100 samples and 8+ years of data. This elimination procedure censors out a large portion of the data and the effect of this has not been statistically evaluated.

4. Replication and Variation of Fish Tissue Concentration Trends

- EPA's technique (i.e., decisions as to which data were included in the trend analyses) was replicated.
- Variations on EPA's technique were plotted to investigate the effects of data inclusion on the fish tissue recovery rates.
- Overall, fish tissue rates are highly variable, and the use of an average rate is unrepresentative of this variation and deceptive when making conclusions about the protectiveness of the remedy.
- Average rates calculated for each variation on the EPA technique were consistently lower than that calculated for the replication of the EPA technique.

The analyses presented here were conducted as a basic and preliminary means of showing the uncertainty in EPA's predictions, specifically regarding the 8% per year recovery rate for Upper Hudson sport fish species on a lipid normalized basis. The development of a method for reducing that uncertainty to reasonable levels would require more rigorous statistics in handling the data. The methods employed by EPA and followed here are inadequate to make confident estimates about how long fish in the Hudson will truly take to recover and meet the goals of the ROD.

Depending on which data are included or excluded (i.e. rib out data or Aroclor-based measurement data), the average recovery rate differs substantially. These variations in data inclusion, along with a replication run using the same parameters reported by EPA, were plotted using R statistical software, and an exponential curve was fit to the data (Figure 2, Figures 2A – 3O, attached at the end of this report). The results were coefficients taken as the percent per year declines in TPCB concentrations. The average 8% rate is shown to be uncertain when it is not reproducible with the slight variations in data inclusion. In fact, the variations consistently produced average rates of recovery lower than the rate calculated using EPA's approach. EPA's approach therefore results in recovery rates that are systematically biased high; the EPA rate is at the fastest end of the range of recovery rates found by applying slight changes (within the reasonable range) to the EPA procedure.

Furthermore, in EPA's reported rates (Table A3-3) and in all the subset variations used here for comparison, the individual rates of recovery vary drastically by species and river section, with the fastest rate of recovery at -18% per year (a negative rate indicates a decrease in concentration) and the least promising rate an increasing trend of +4% per year (Tables 1 and 2). Importantly, the use of an average rate, while useful in representing the central tendency of recovery rates, is deceptive in determining EPA's protectiveness statement for the Site, because those fish populations with slow recovery rates or slightly increasing trends have half-lives several decades longer than the 8 years suggested by the 8% rate. These populations will continue to be an exposure risk for human health beyond the timeframe suggested by the 2017 Proposed FYR.

Table 1 – Lipid Normalized $TPCB_{HE}$ vs. SDATE Recovery Rates (%/year) and Corresponding Half Lives (years); Negative Half-life Indicates Increasing Trend

		Trend A (Exclude Rib Out)		Trend B (Include rib out; EPA method)	
		Recovery Rate	Half-life	Recovery Rate	Half-life
RS 1	Largemouth Bass	-3.97	17.48	-9.29	7.46
	Brown Bullhead	-6.53	10.61	-8.16	8.50
	Yellow Perch	-10.62	6.52	-15.00	4.62
	Smallmouth Bass	-17.50	3.96	-12.76	5.43
	Pumpkinseed	-5.00	13.87		
RS 2	Largemouth Bass	-3.20	21.64	-7.63	9.09
	Brown Bullhead	1.81	-38.28	-2.27	30.59
	Yellow Perch	-15.55	4.46	-20.18	3.43
	Smallmouth Bass	-13.99	5.00	-15.24	4.55
	Pumpkinseed	-5.28	13.13		
RS 3	Largemouth Bass	-6.37	10.89	-10.75	6.45
	Brown Bullhead	-2.53	27.37	-3.13	22.14
	Yellow Perch	-18.56	3.73	-16.76	4.14
	Smallmouth Bass	-0.36	191.10	-3.87	17.94
	Pumpkinseed	-9.78	7.09		
	Arithmetic Mean Recovery Rate for Sport Fish	-8.12	~ 8	-10.42	~ 6
	Arithmetic Mean Recovery Rate Including Pumpkinseed	-7.83	~ 8		
	Species and Length Weighted Average Recovery Rate ("Frankenfish")	**-5.31**	**~ 13**	**-7.96**	**~ 8**
	Max Rate (Slowest recovery)	1.81	-38.28	-2.27	30.86
	Min Rate (Fastest recovery)	-18.56	3.73	-20.18	3.43
	Standard Deviation of Rates	6.26		5.74	
	Standard Error of the Mean	1.62		1.66	

Table 2 – Lipid Normalized $TPCB_{Aroclor}$ vs. SDATE Recovery Rates (%/year) and Corresponding Half-Lives (Years); Negative Half-life Indicates Increasing Trend

		Trend A (Exclude Rib Out, Include 2016 data for RS1)		Trend B (Include Rib Out)	
		Recovery Rate	Half-life	Recovery Rate	Half-life
RS 1	Largemouth Bass	-6.19	11.20	-10.12	6.85
	Brown Bullhead	-4.37	14.95	-6.05	11.46
	Yellow Perch	-8.36	8.29	-12.82	5.41
	Smallmouth Bass	-9.69	7.15	-7.29	9.51
	Pumpkinseed	-5.05	30.93		
RS 2	Largemouth Bass	-2.44	12.59	-10.78	6.43
	Brown Bullhead	3.34	-20.76	-3.34	20.73
	Yellow Perch	-10.06	6.89	-16.20	4.28
	Smallmouth Bass	-7.42	9.34	-10.81	6.41
	Pumpkinseed	-17.97	3.86		
RS 3	Largemouth Bass	-6.28	11.05	-9.54	7.27
	Brown Bullhead	-1.54	45.04	-1.79	38.72
	Yellow Perch	-13.03	5.32	-12.34	5.62
	Smallmouth Bass	4.16	-16.66	0.38	-184.47
	Pumpkinseed	-9.76	7.10		
	Arithmetic Mean Recovery Rate for Sport Fish	-5.16	~ 13	-8.39	~ 8
	Arithmetic Mean Recovery Rate Including Pumpkinseed	-6.31	~ 11		
	Species and Length Weighted Average Recovery Rate ("Frankenfish")	**-4.39**	**~ 15**	**-6.92**	**~ 9**
	Max Rate (Slowest Recovery)	4.16	-16.66	0.38	-184.47
	Min Rate (Fastest Recovery)	-17.97	3.86	-16.20	4.28
	Standard Deviation of Rates	5.79		4.91	
	Standard Error of the Mean	1.58		1.42	

Several data criteria were chosen to create variations on the data subset used by EPA. EPA's lipid normalized analysis generated a trendline for $TPCB_{HE}$ data between 1995 and 2008 for all three Upper Hudson River Sections, and included the GE rib out samples taken in 2007 and 2008. To address changes in the average decay rate caused by uncertainty in the transformation of $TPCB_{Aroclor}$ to $TPCB_{HE}$, two separate trendlines were calculated for each of these datasets. Trend A excludes rib out data taken by GE and Trend B includes the rib out data. The species-weighted average and arithmetic average decay rates were calculated for sport fish species (largemouth bass, brown bullhead, and yellow perch included in the weighted average, with smallmouth bass included in the arithmetic mean). Both Trend A and Trend B apply to the period between 1995 and 2008.

Trend A was also applied to records for pumpkinseed, for which the majority of samples were whole-body. Rib out fillet samples were therefore not an issue for this species and there is no Trend B trendline for pumpkinseed rates.

Trend B represents the same data subset used by EPA. When applied to the original $TPCB_{Aroclor}$ data, this trend represents a variation on EPA's method; when applied to the transformed $TPCB_{HE}$ data, this trend represents the replication of EPA's method. A diagram of the comparative variations of EPA's method is shown in Figure 1.

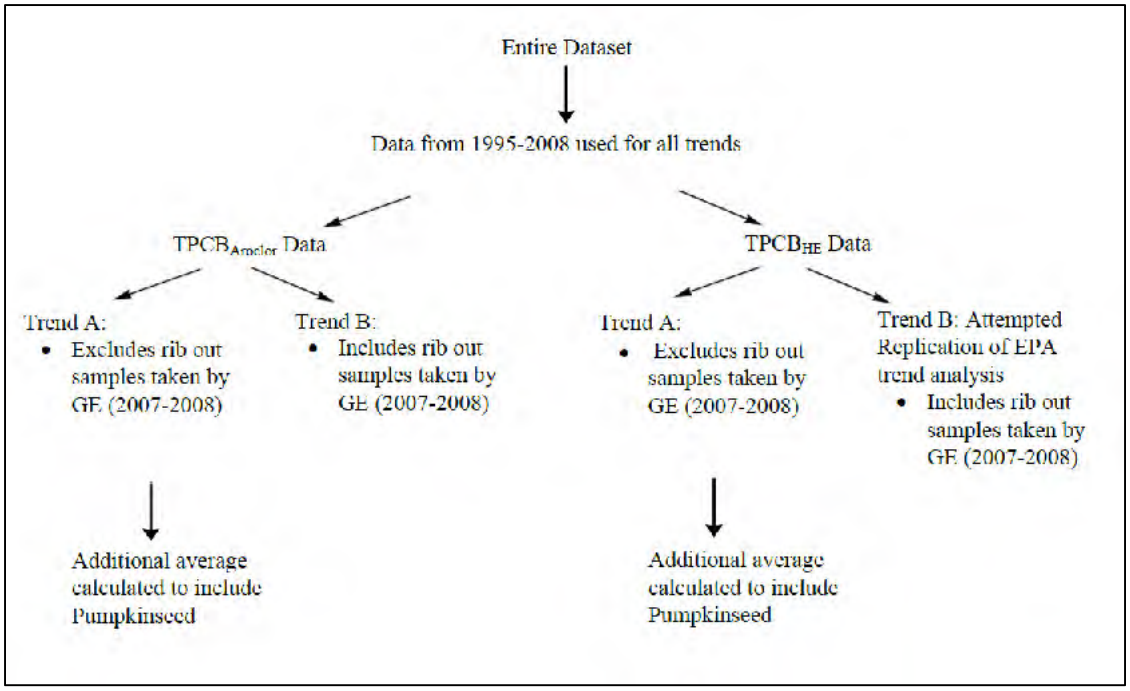

Figure 1 – Variations of EPA Data Criteria for Fish Samples Through Time.

The replication of EPA's analysis reproduced the weighted average 8% rate reported by EPA. Individual rates by species and RS for $TPCB_{HE}$ Trend B are different than those reported in Table A3-3, indicating that $TPCB_{HE}$ Trend B is not a true replication of the EPA process and likely includes differences in data criteria. In this analysis, it was assumed that the sampling design, which was to target fish of legal length (defined for each species in the Baseline Monitoring Program Quality Assurance Project Plan, QEA 2003), resulted in the entire database consisting of adult fish samples. Upon further query of the database, it is now known that no more than 10% of the data used for trends here were samples under the minimum legal length. Despite this possible discrepancy between the EPA method and the replication method, the replication achieved similar variation between rates and an 8% weighted average. Consistency between subsets used in this report allows a general and relative comparison of the EPA results with results from potential changes to that approach.

Table 1 shows the results of $TPCB_{HE}$ trends, while Table 2 shows the results of $TPCB_{Aroclor}$ trends. $TPCB_{HE}$ results show average recovery rates higher by 2-3% per year (arithmetic average) or 1% per year (weighted average) than the $TPCB_{Aroclor}$ average

recovery rates for both Trends A and B. In both Tables, Trend A shows average recovery rates lower than Trend B rates by about 2-3% per year. Inclusion of pumpkinseed recovery rates did not notably change the average recovery rates for Trend A in either Table. Out of all four different approaches, the species weighted average rate for $TPCB_{HE}$ Trend B is the fastest and differs from other rates by 25-40%.
It is apparent that by selecting these criteria for data to be included in the 2017 Proposed FYR fish tissue trend analysis, EPA overestimated the average rate of decline for adult sport fish in the Upper Hudson. For comparison, recovery times to reach interim concentration goals were calculated for each weighted average recovery rate using a simple exponential decay equation (Table 3).

Table 3 – Recovery Times for Species and Length Weighted Average Recovery Rates (Current Average Wet Weight Concentration: 1.3 mg/kg)

Approach	Rate	Years to 0.4 (mg/kg)	Years to 0.2 (mg/kg)
HE Trend A	-0.05	22	35
HE Trend B (EPA)	-0.07956	15	24
Aroclor Trend A	-0.04393	27	43
Aroclor Trend B	-0.0692	17	27

Exponential Equation:

$$y = ae^{-kt}$$

$$t = \frac{\ln\left(\frac{y}{a}\right)}{-k}$$

a = Current average wet weight concentration (1.3 mg/kg)
y = Interim goal wet weight concentration (.4 or .2 mg/kg)
k = Exponential decay rate
t = Time to interim goals in years

More important than the difference between average rates, however, is the difference between individual rates with variation in the method. For example, consider largemouth bass trends for RS2 (Figure 2). Removing the rib-out samples from the

analysis causes the recovery rate to drop from 7.63% per year to 3.2% per year in $TPCB_{HE}$ measurements and from 10.78% per year to 2.44% per year in $TPCB_{Aroclor}$ measurements. The difference in half-life values calculated from these rates is an additional 10-20 years for TPCB concentrations to reach half of their present value. If inclusion of the rib out data produced a trendline truly representative of fish tissue MNA recovery, then the rate of recovery would not be consistently slower across species and River Sections once those data are removed. It is these individual recovery rates and half-life predictions that are relevant to the protectiveness of the remedy, not an oversimplified average.

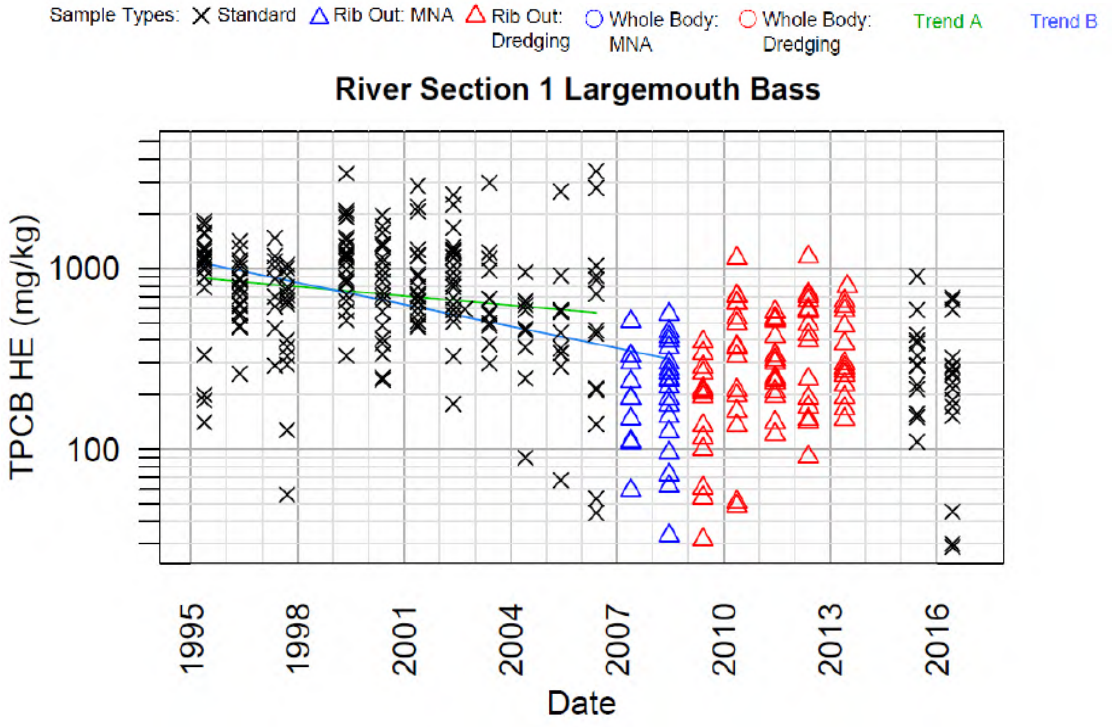

Figure 2 – Example Plot of Differences in Trend (A and B, in Green and Blue, Respectively) due to Exclusion of Rib Out (A) and Inclusion of Rib Out (B). The Trends Produce Very Different Half-life Estimations, with Trend A at 17.5 Years and B at 7.5 Years.

As a further illustration of the large uncertainty and unsuitability of the fish data for determining the protectiveness of the remedy, pre-dredging data were sectioned into three intervals: 1995-1999, 2000-2004, and 2005-2008. For each of these intervals, as a

basic test of the consistency of the rate of decline for each species and RS combination, a different trendline was plotted. These plots (Figure 3, Figures 4 A-R at end of document) show extreme variability of trends between time periods, demonstrating that choosing arbitrary parameters for data to be included in the analysis is not successful in capturing the data to derive a reproducible rate for PCB decline. The annual mean TPCB concentration was also plotted for each species and River Section combination (Figure 4, Figures 5 A-R at end of document).

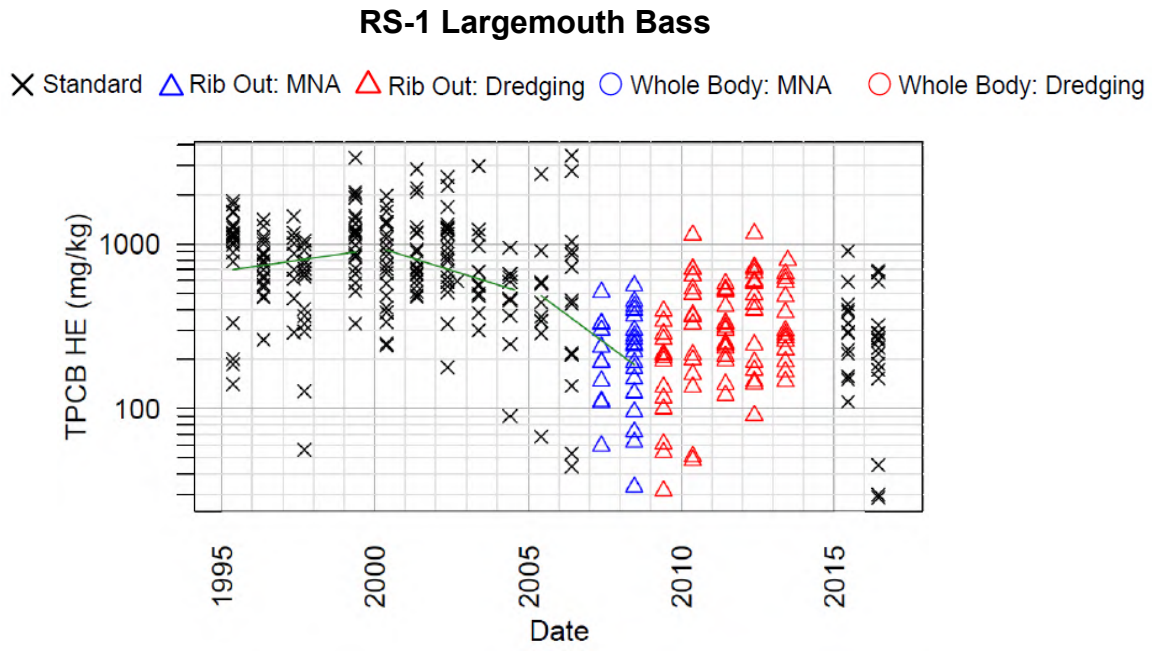

Figure 3 – Example Plot of Differences in Trend (Green Lines) due to Selection of Sediment Data Intervals (1995-1999, 2000-2004, and 2005-2008). The Slopes for These Trends Range from Positive to Negative, Highlighting the Uncertainty Associated with Trend Determination.

Annual Mean TPCB Concentration, RS 1 Largemouth Bass

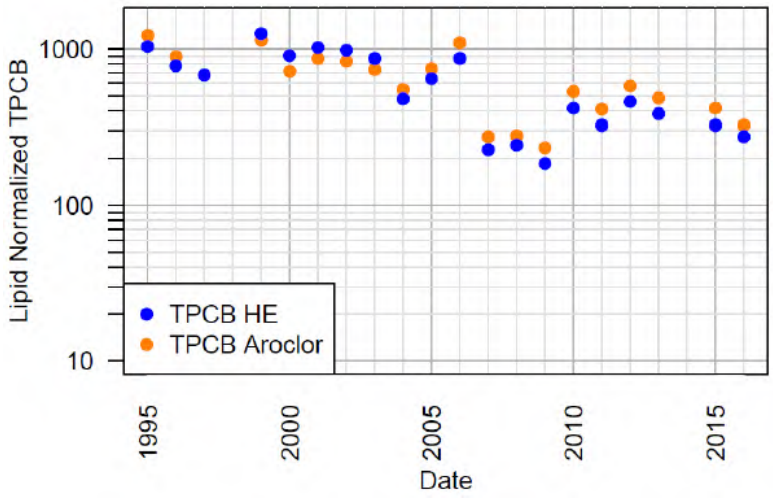

Figure 4 – Example Plot of Annual Mean TPCB Concentration. The Change in the Relationships Between the $TPCB_{Aroclor}$ to $TPCB_{HE}$ Concentrations Shows the Two Methods Are Not Equivalent.

5. Surface Sediment Cumulative Plots

- Preliminary cumulative distribution plots of surface sediment TPCB concentrations show a general improvement between SSAP (2002-2005) and OM&M (2016) datasets.
- When the SSAP dataset is separated into dredging and non-dredging area sample sets, cumulative distribution plots show lesser degrees of improvement in non-dredging areas than the improvement shown by plotting all SSAP samples. Non-dredging areas in River Section 1 show very little or no improvement (Figure 5A).

As a comparative exercise to examine pre-dredging vs post-dredging surface sediment data, TPCB values (Aroclor sum measurements) from the SSAP and 2016 OM&M programs were plotted on the x-axis vs cumulative probability on the y-axis. Each plot (Figures 5-7) show an improvement in surface sediment concentrations between the two programs. However, the plots are precursory due to differences between sampling programs. Specifically, the SSAP was designed with the goal of delineating dredging areas and therefore focused in River Sections 2 and 3 on consolidated sediments of suspected elevated contamination, while the OM&M program sampled only non-dredging areas in all three River Sections. EPA acknowledges the difficulties and biases created by these differences in the Proposed FYR.

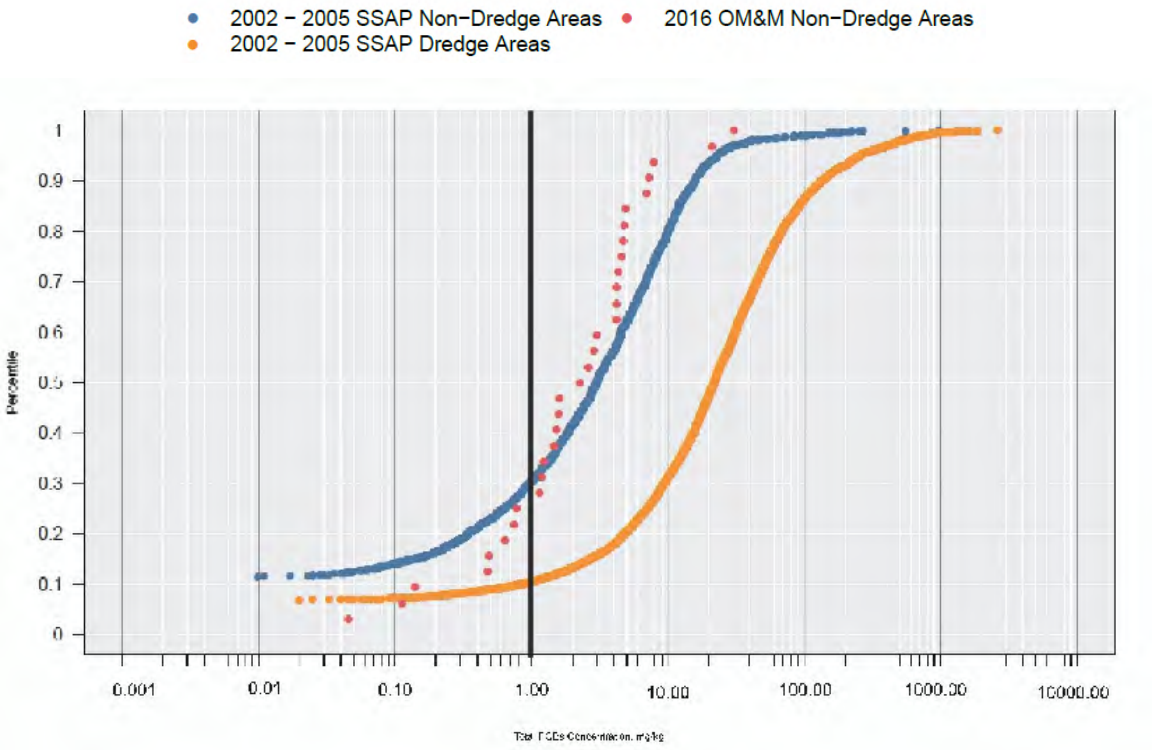

Figure 5A – Cumulative Distribution of TPCB Concentration in Surface Sediment Samples (Top 12 inches or less) in River Section 1

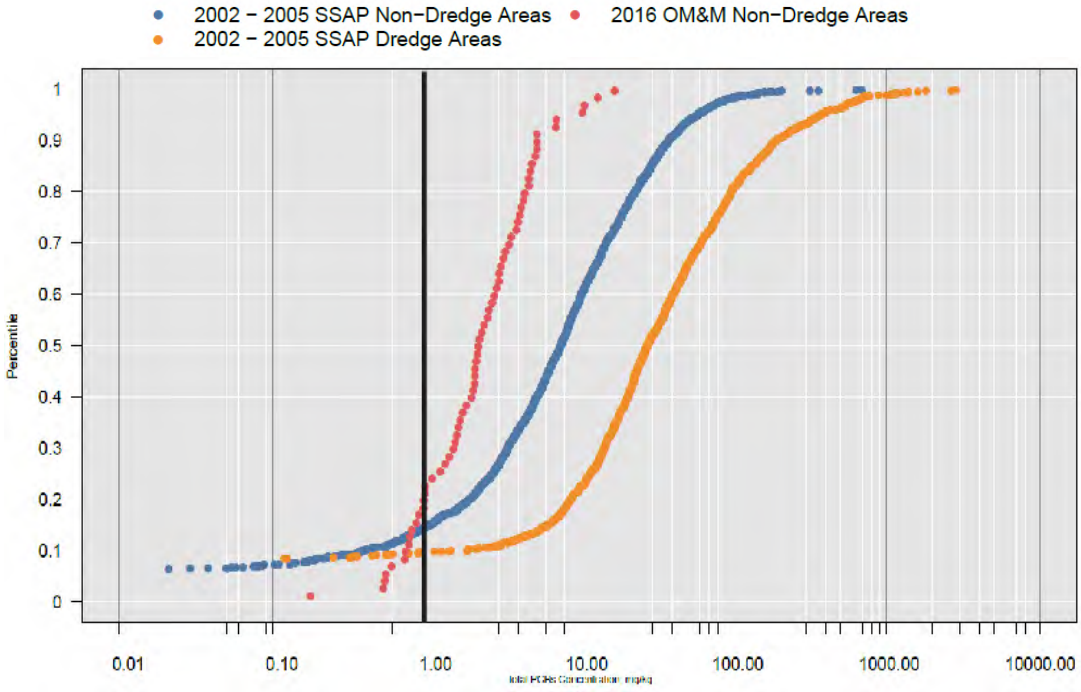

Figure 5B – Cumulative Distribution of TPCB Concentration in Surface Sediment Samples (Top 12 inches or less), in River Section 2

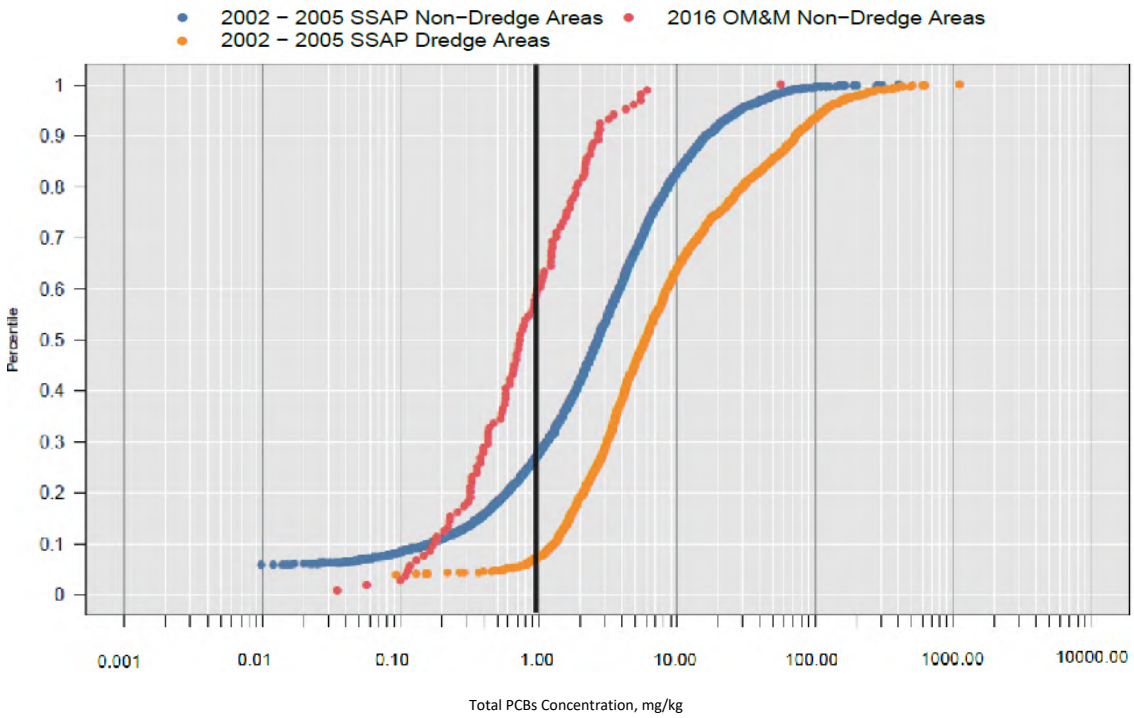

Figure 5C – Cumulative Distribution of TPCB Concentration in Surface Sediment Samples (Top 12 inches or less), in River Section 3

6. Conclusions and Future Analyses

- Both the analyses presented here and those presented by EPA cannot determine the protectiveness of the remedy with any degree of confidence.
- The slowest fish tissue recovery rates hold more weight when considering the remedy effectiveness, since these species will continue to be a pathway to human exposure past the timeframe asserted by EPA. The use of average recovery rates does not consider the variability in individual recovery rates by species.
- Future analyses should focus on quantifying and minimizing uncertainty both in the data transformation process and the comparability between datasets, including the rib in/out fish data and SSAP/OM&M sediment datasets.

Several concerns regarding the remediation-period data merit continued attention and more thorough investigation. The most pressing of these is the need for a procedure to carry the uncertainty of transforming $TPCB_{Aroclor}$ measurements into $TPCB_{HE}$ measurements into the fish tissue trend analysis. Additionally, more rigorous testing than used here or in the Proposed FYR may show comparability (or a lack of comparability) between $TPCB_{Aroclor}$ and $TPCB_{HE}$ measurements.

An additional question to be pursued is the potential effects of sample depth on rates of PCB decay in surface sediment over time. Surface sediment data is composed largely of samples with start and end depths of 0-2 inches or 2-12 inches, but it is possible that samples from 0 to 6 inches may show a different distribution of concentrations characteristic of the biotic zone. Cumulative distribution plots are expected to be useful for expanding on this question.

Overall, the preliminary analyses here need much refinement and intensification in order to make confident statements about recovery from contamination in the Upper Hudson and to reliably predict achievement of the goals of the ROD. They do show, however, that fish tissue concentration decay rates are extremely variable and that an 8% average decay rate is an highly uncertain, biased high, and oversimplified representation of this variation. The EPA 8% rate of recovery exaggerates the estimate of the rate of

natural recovery in the Hudson River. At present, it cannot be concluded from any of the analyses performed that rates of recovery are on track with the ROD model output. The data does not support EPA's conclusion that the goals of the ROD will be achieved.

7. References

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U.S. Environmental Protection Agency (USEPA) OSWER-OSRTI Environmental Response Team. 2015. the Hudson River PCBs Superfund Site Community Advisory Group (CAG) Meeting, Saratoga Springs, NY, 1 October 2015. http://www.hudsoncag.ene.com/files/MSG_CAG_Oct2015_Hudson%20Fish_v01oct15_FINAL.pdf

United States Environmental Protection Agency (USEPA). 2017. Proposed Second Five-Year Review Report for the Hudson River PCBs Superfund Site. Prepared by EPA Region 2.

FIGURES

Figure 2A: River Section 1 Largemouth Bass
Coefficients: A −3.965 %, B −9.288 %
Half Life: A 17.481 years, B 7.463 years

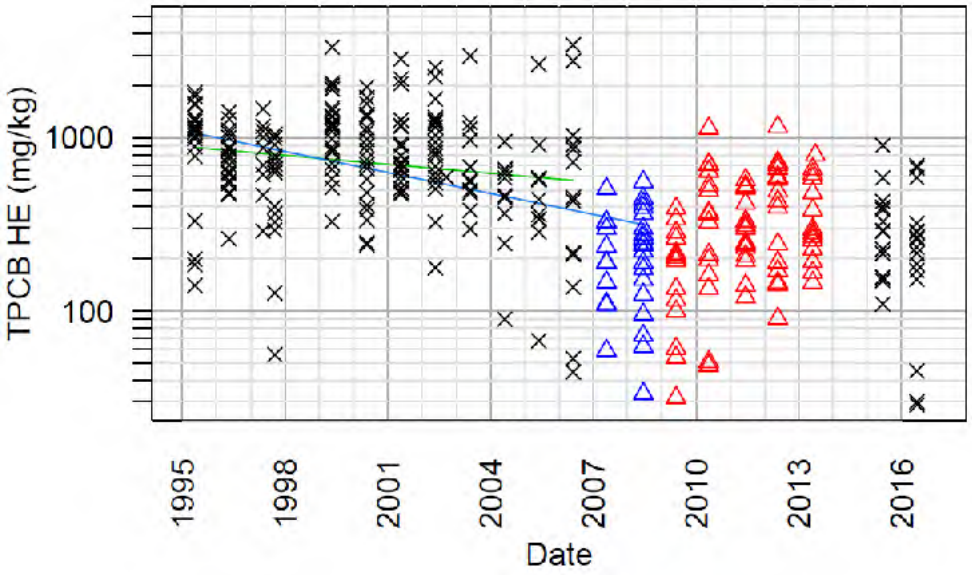


Figure 2B: River Section 1 Brown Bullhead
Coefficients: A −6.534 %, B −8.155 %
Half Life: A 10.608 years, B 8.499 years

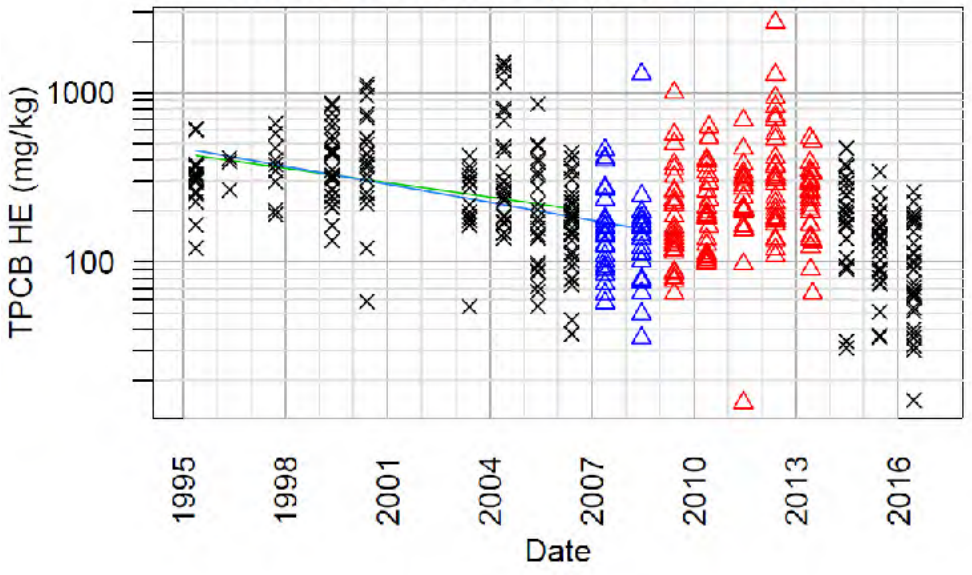


Figure 2C: River Section 1 Yellow Perch
Coefficients: A −10.624 %, B −15 %
Half Life: A 6.524 years, B 4.621 years

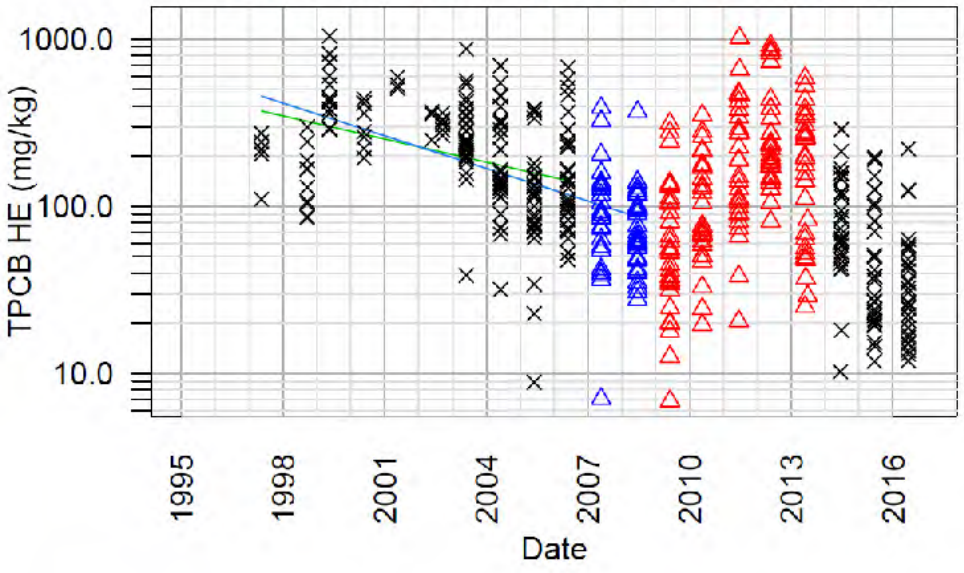
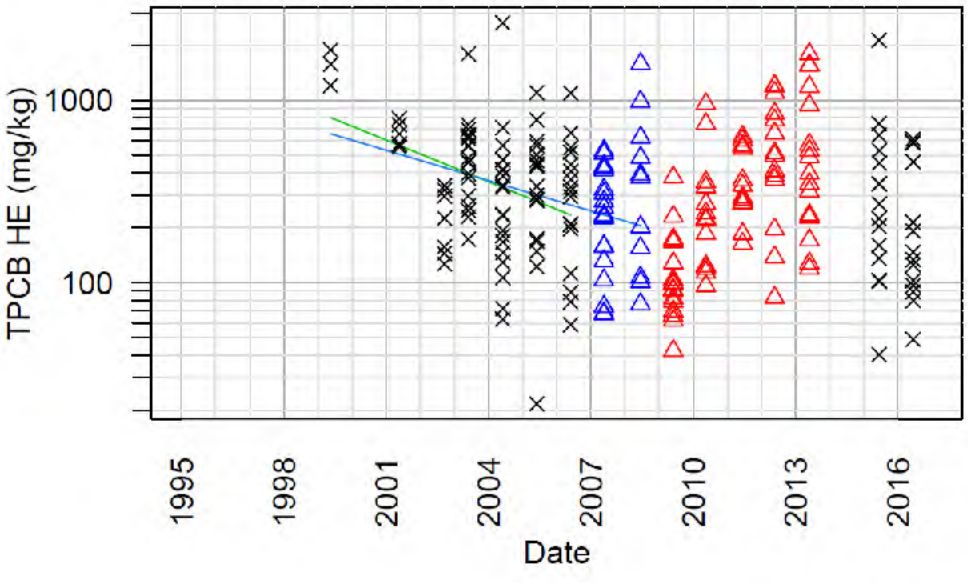


Figure 2D: River Section 1 Smallmouth Bass
Coefficients: A −17.5 %, B −12.763 %
Half Life: A 3.961 years, B 5.431 years



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 2E: River Section 1 Pumpkinseed
Coefficient: −4.998 %
Half life: 13.869

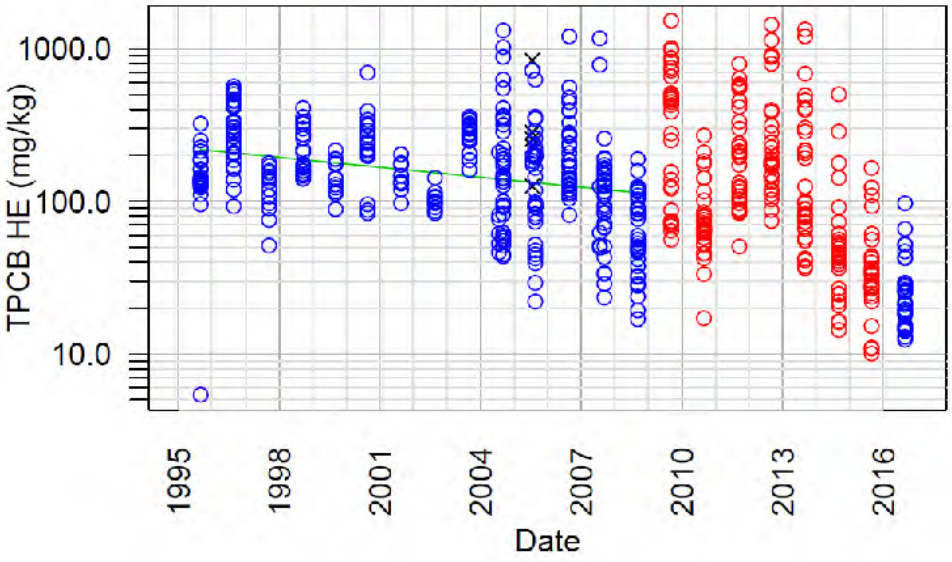


Figure 2F: River Section 2 Largemouth Bass
Coefficients: A −3.202 %, B −7.627 %
Half Life: A 21.644 years, B 9.088 years

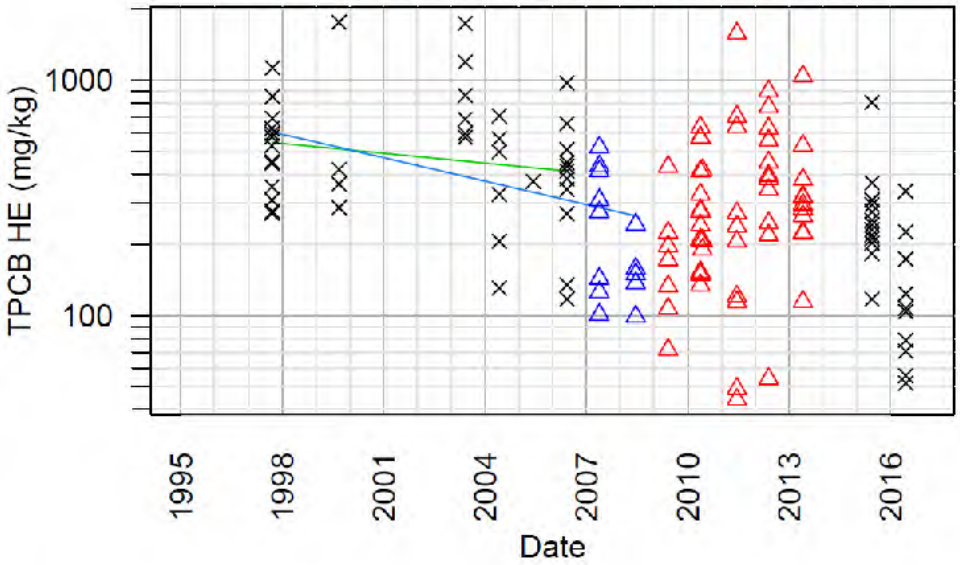


Figure 2G: River Section 2 Brown Bullhead
Coefficients: A 1.811 %, B −2.266 %
Half Life: A −38.28 years, B 30.586 years

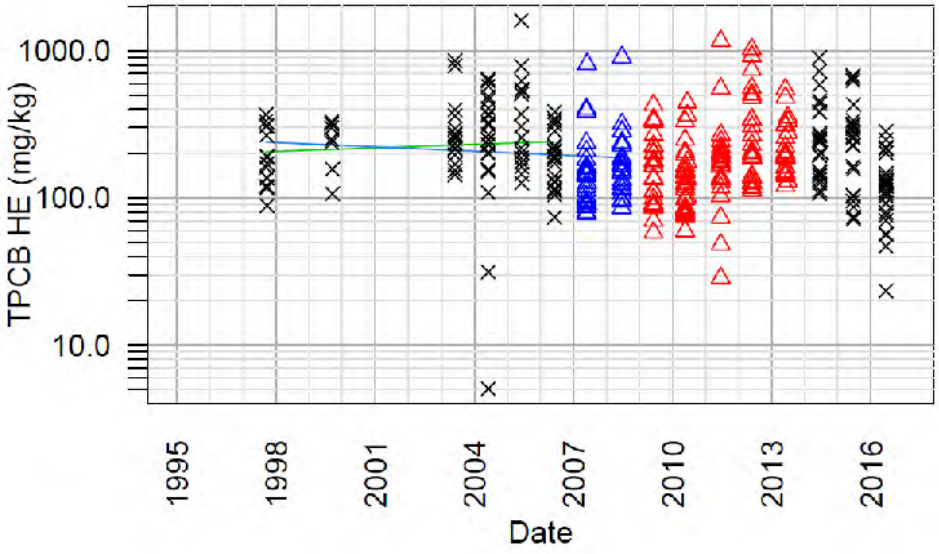
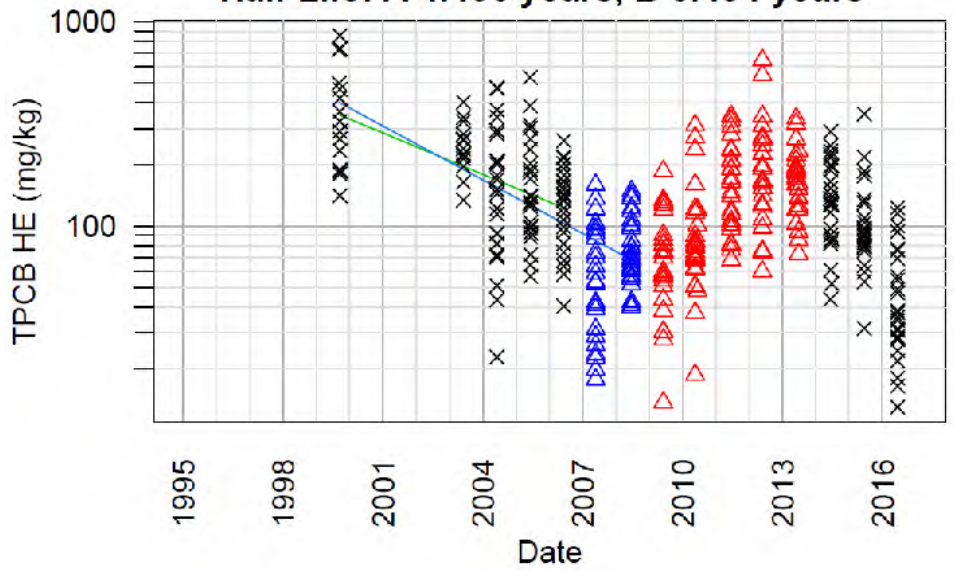


Figure 2H: River Section 2 Yellow Perch
Coefficients: A −15.554 %, B −20.182 %
Half Life: A 4.456 years, B 3.434 years



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 2I: River Section 2 Smallmouth Bass
Coefficients: A −13.865 %, B −15.236 %
Half Life: A 4.999 years, B 4.549 years

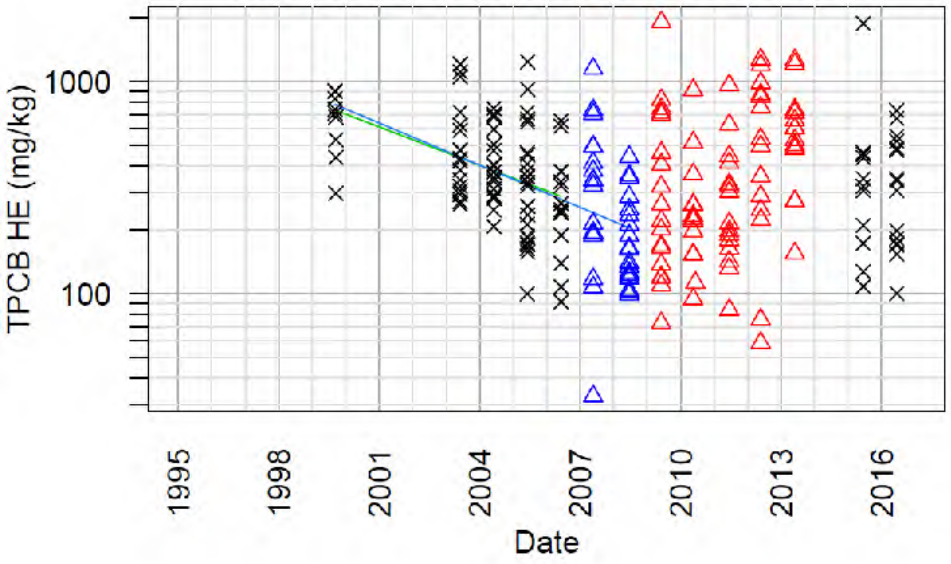


Figure 2J: River Section 2 Pumpkinseed
Coefficient: −5.279 %
Half life: 13.13

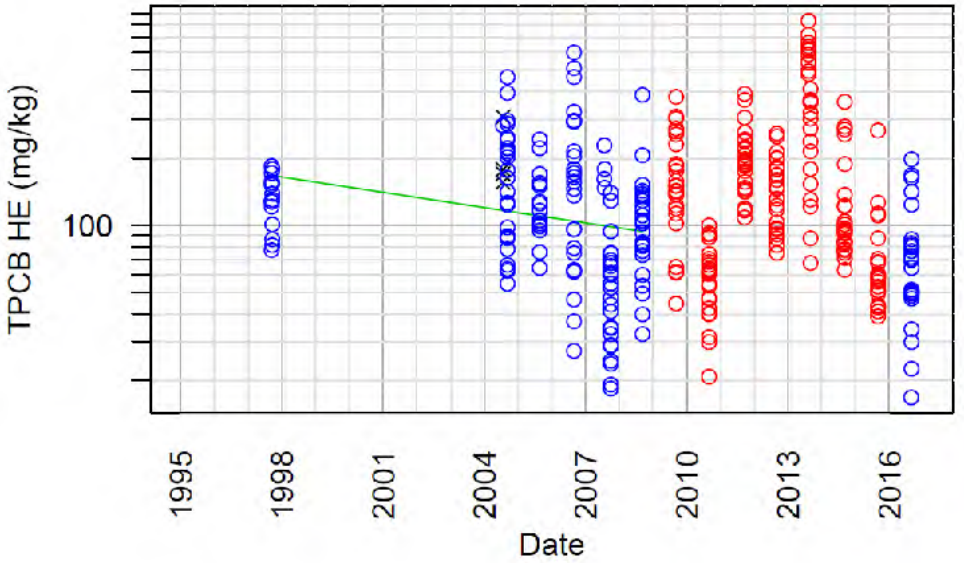


FIgure 2K: River Section 3 Largemouth Bass
Coefficients: A −6.368 %, B −10.75 %
Half Life: A 10.886 years, B 6.448 years

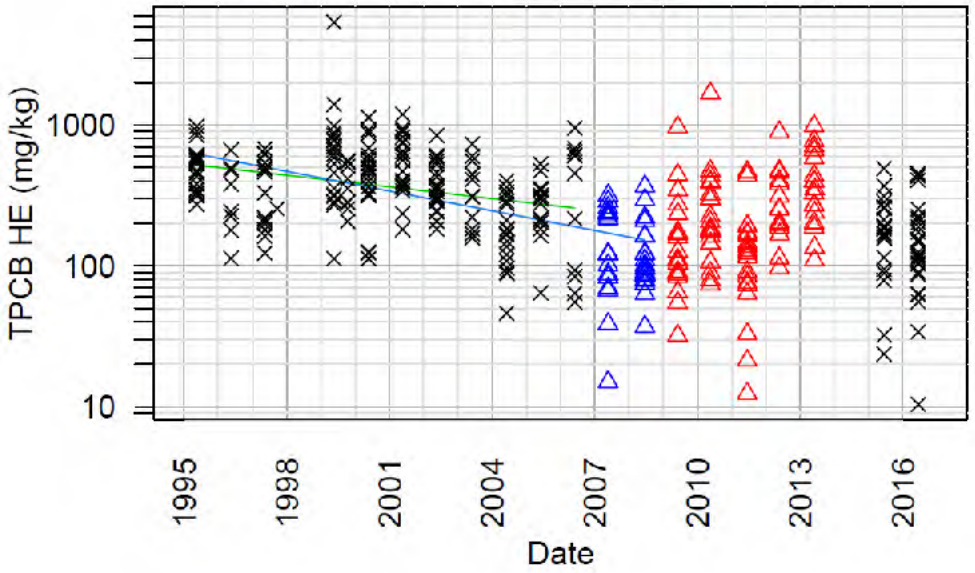
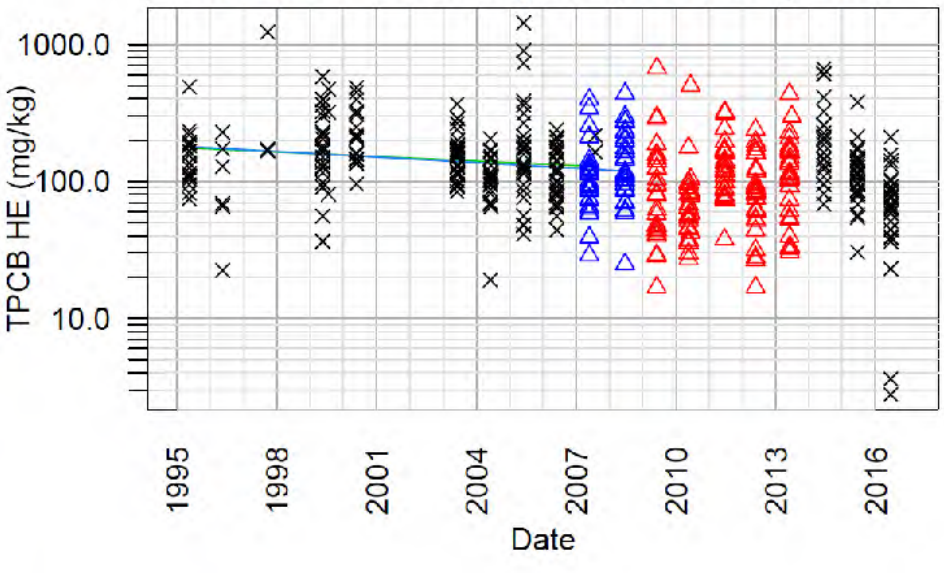


Figure 2L: River Section 3 Brown Bullhead
Coefficients: A −2.533 %, B −3.131 %
Half Life: A 27.369 years, B 22.136 years



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 2M: River Section 3 Yellow Perch
Coefficients: A −18.562 %, B −16.76 %
Half Life: A 3.734 years, B 4.136 years

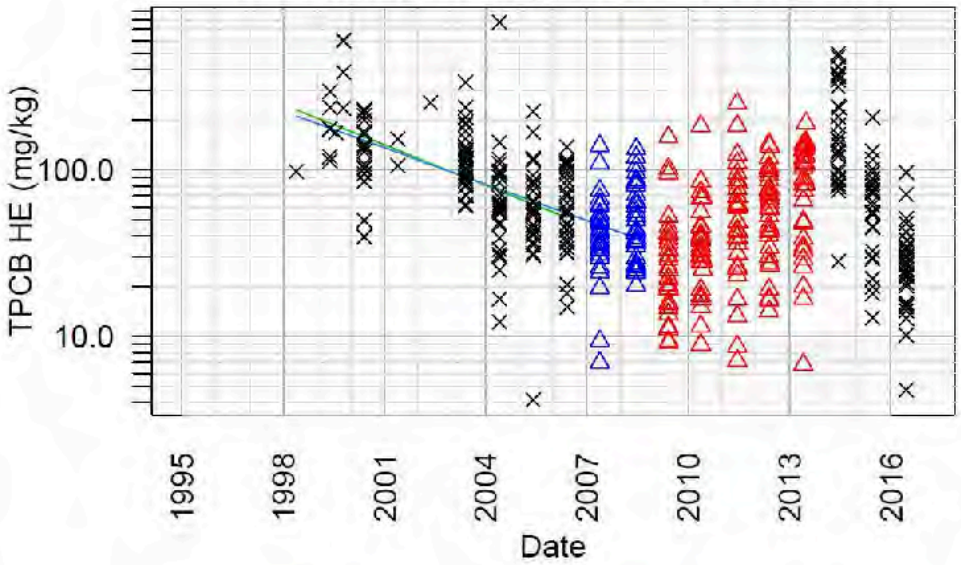


Figure 2N: River Section 3 Smallmouth Bass
Coefficients: A −0.363 %, B −3.865 %
Half Life: A 191.101 years, B 17.936 years

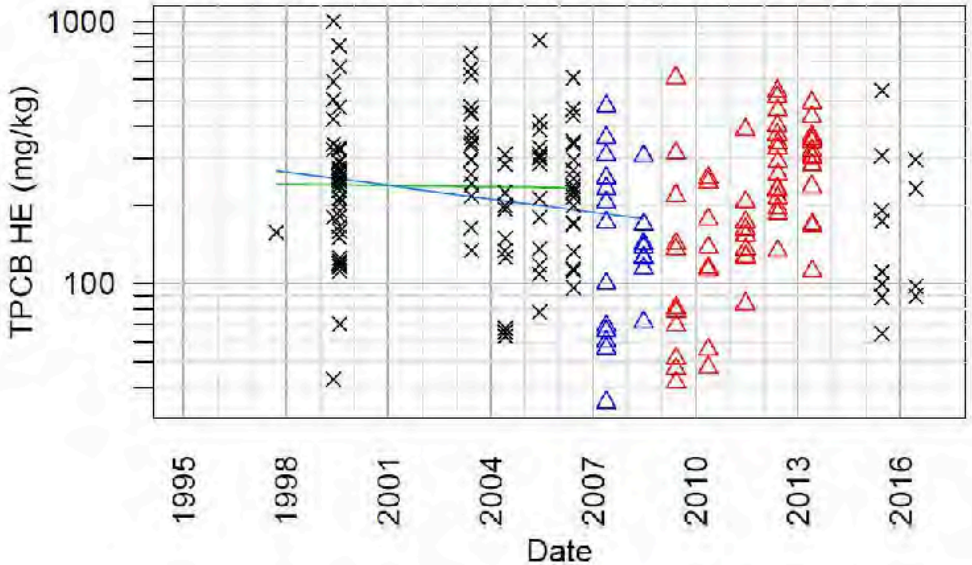
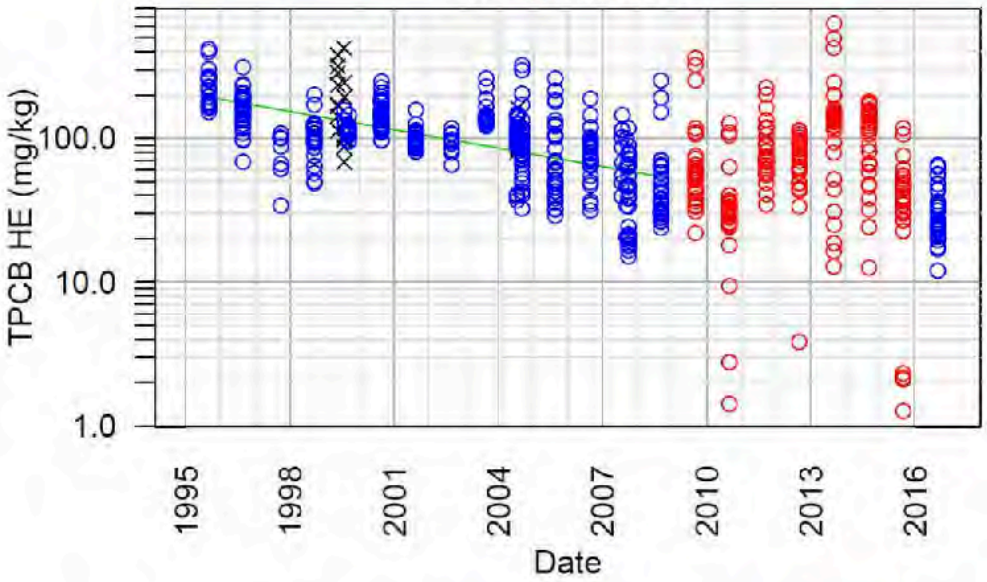


Figure 2O: River Section 3 Pumpkinseed
Coefficient: −9.783 %
Half life: 7.085



Sample Types: Standard | Rib Out: MNA | Rib Out: Dredging | Whole Body: MNA | Whole Body: Dredging | Trend A | Trend B

Figure 3A: River Section 1 Largemouth Bass
Coefficients: A −6.187 %, B −10.118 %
Half Life: A 11.202 years, B 6.851 years

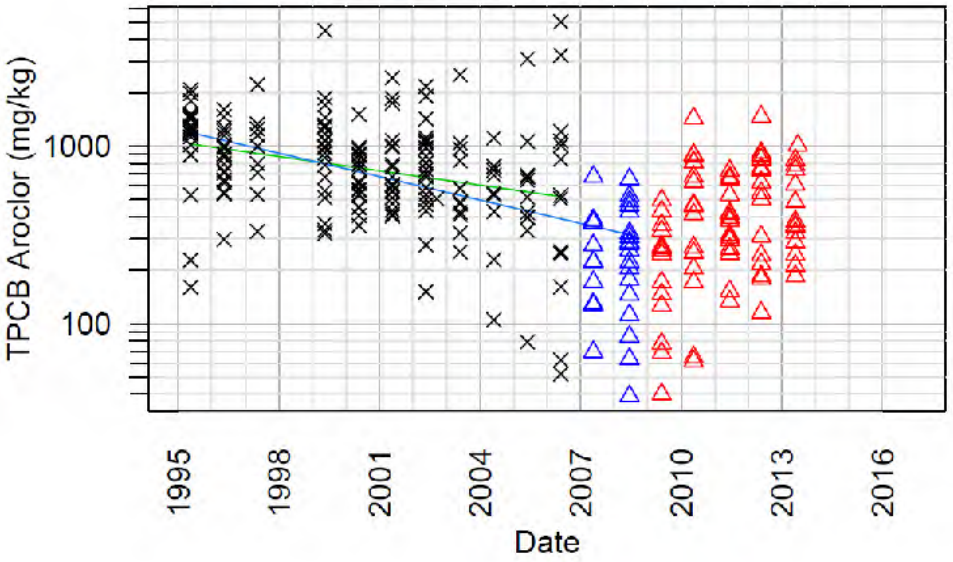


Figure 3B: River Section 1 Brown Bullhead
Coefficients: A −4.635 %, B −6.051 %
Half Life: A 14.954 years, B 11.456 years

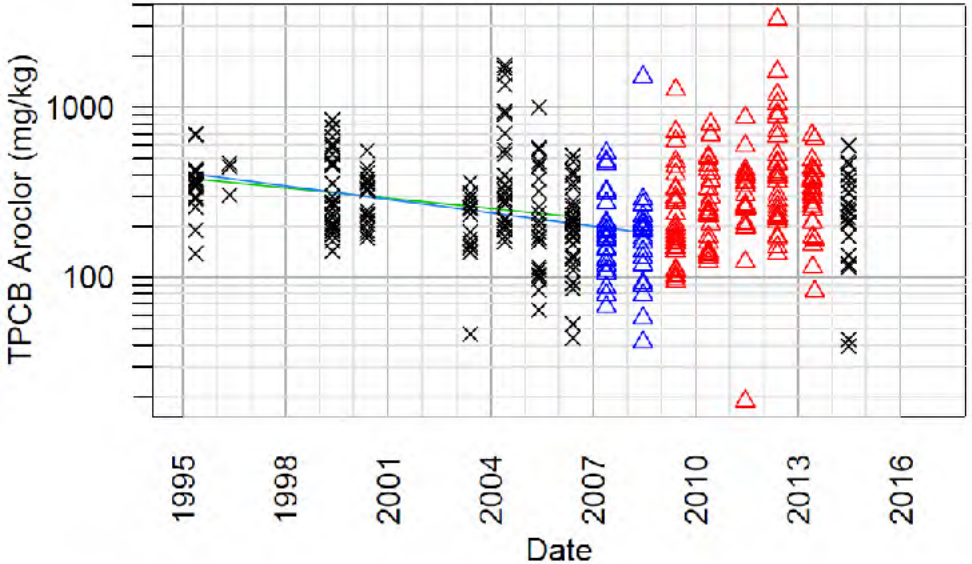


Figure 3C: River Section 1 Yellow Perch
Coefficients: A −8.357 %, B −12.817 %
Half Life: A 8.294 years, B 5.408 years

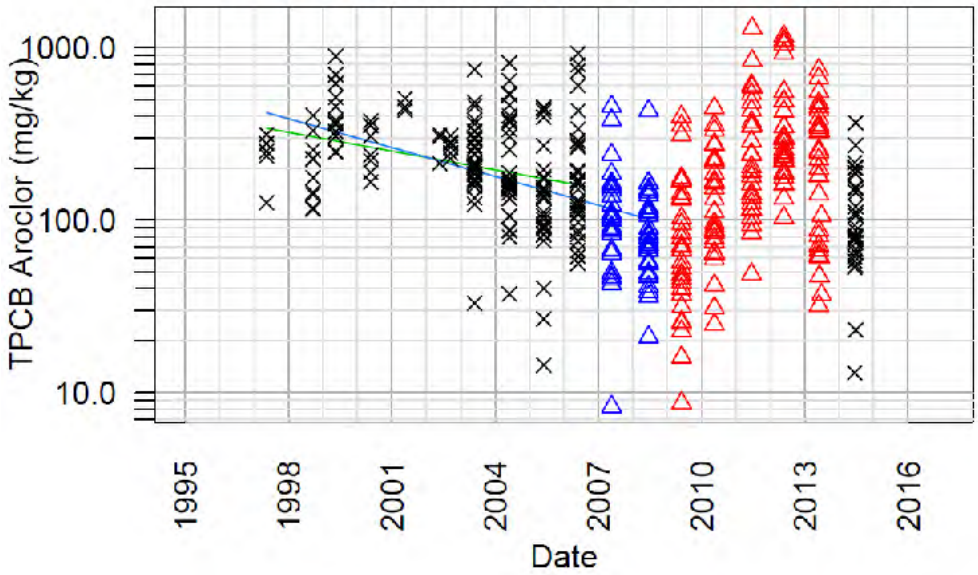
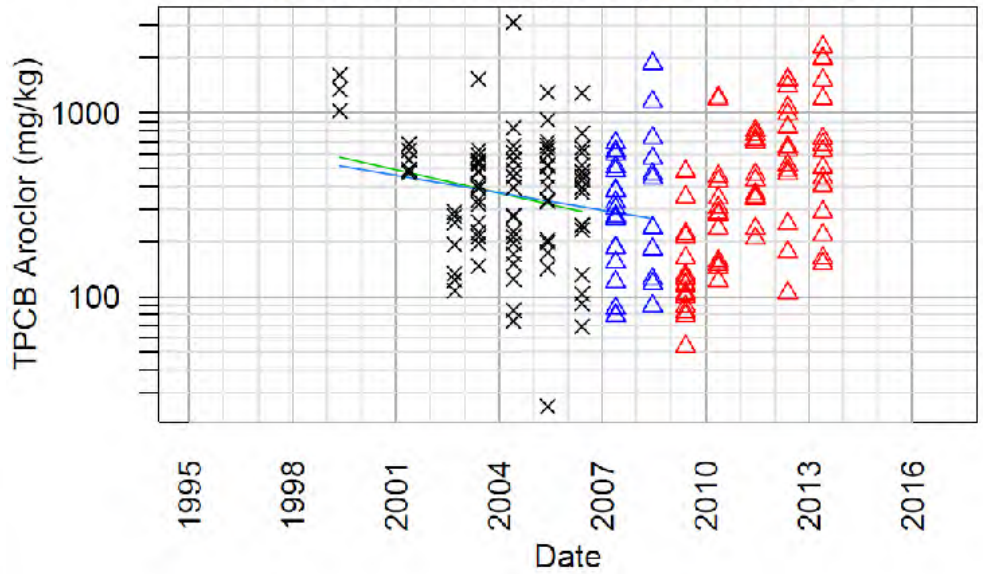


Figure 3D: River Section 1 Smallmouth Bass
Coefficients: A −9.692 %, B −7.29 %
Half Life: A 7.152 years, B 9.508 years



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 3E: River Section 1 Pumpkinseed
Coefficient: −5.505 %
Half life: 12.591

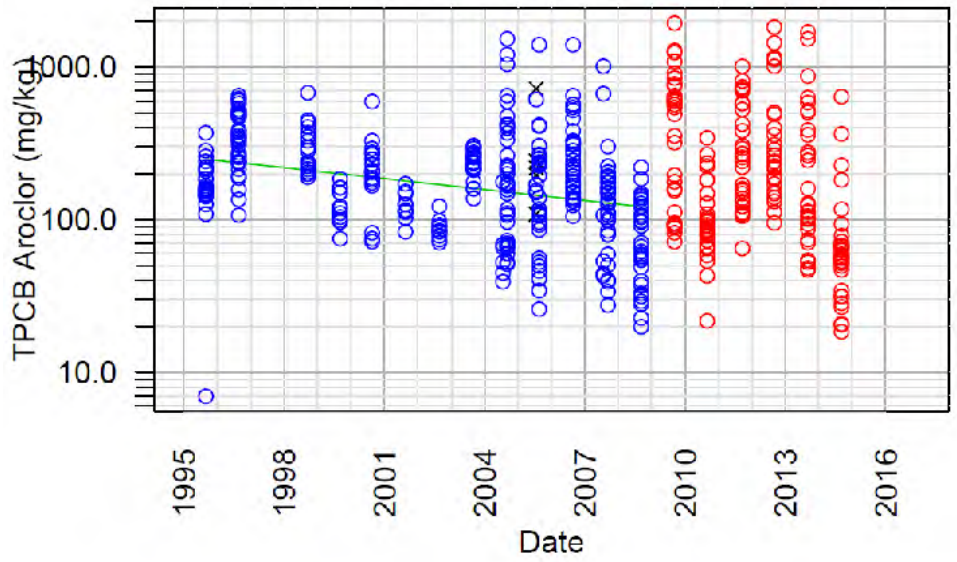


Figure 3F: River Section 2 Largemouth Bass
Coefficients: A −2.437 %, B −10.783 %
Half Life: A 28.443 years, B 6.428 years

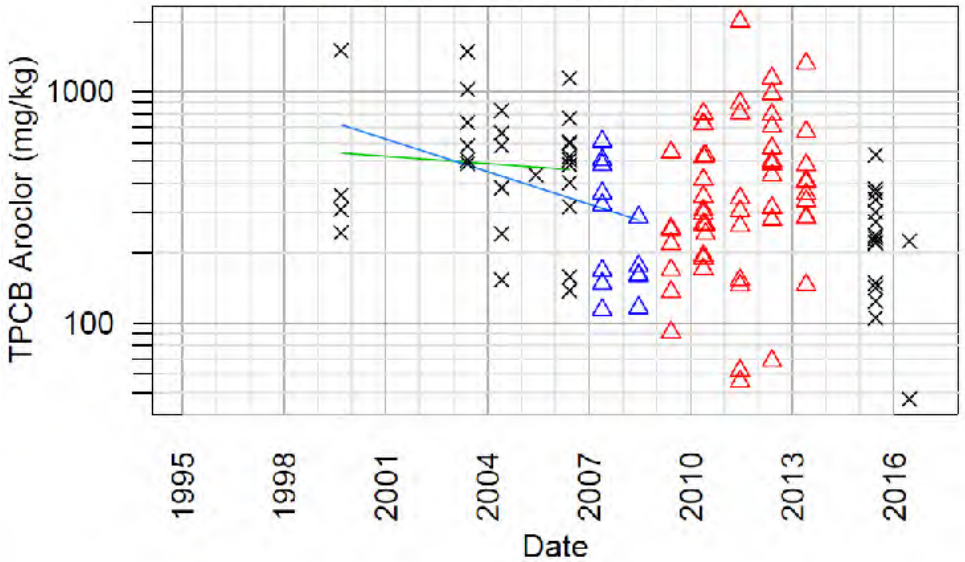


Figure 3G: River Section 2 Brown Bullhead
Coefficients: A 3.338 %, B −3.344 %
Half Life: A −20.764 years, B 20.73 years

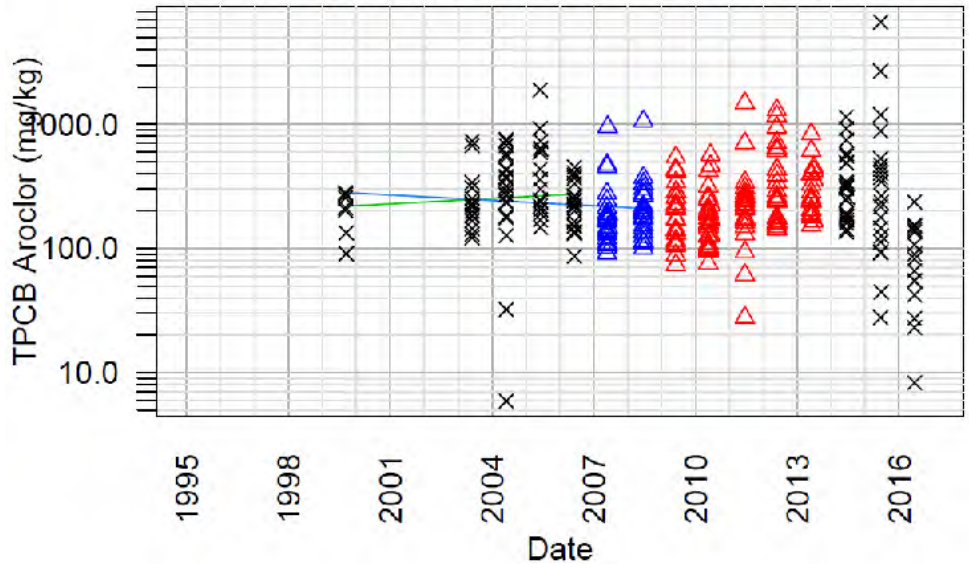
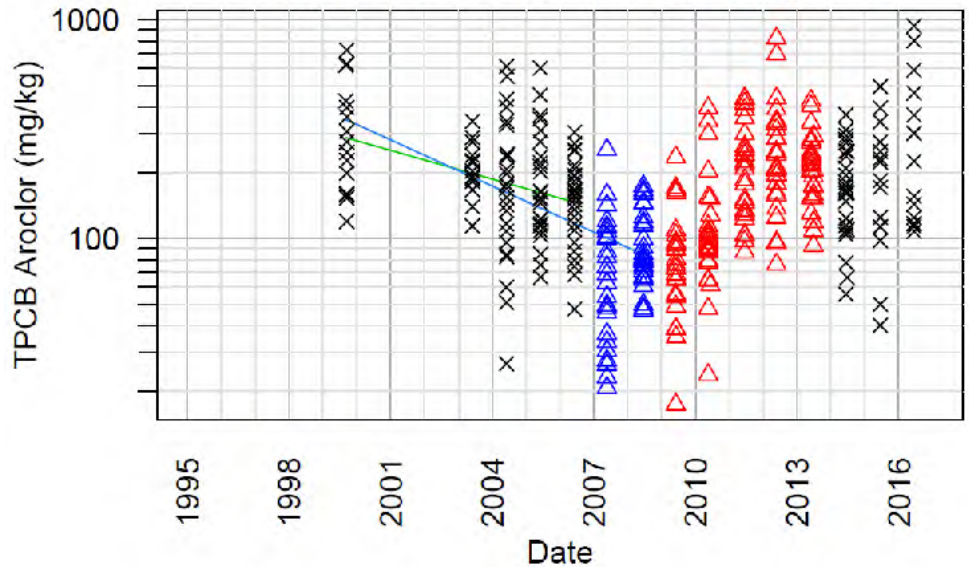


Figure 3H: River Section 2 Yellow Perch
Coefficients: A −10.059 %, B −16.203 %
Half Life: A 6.891 years, B 4.278 years



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 3I: River Section 2 Smallmouth Bass
Coefficients: A −7.421 %, B −10.814 %
Half Life: A 9.341 years, B 6.41 years

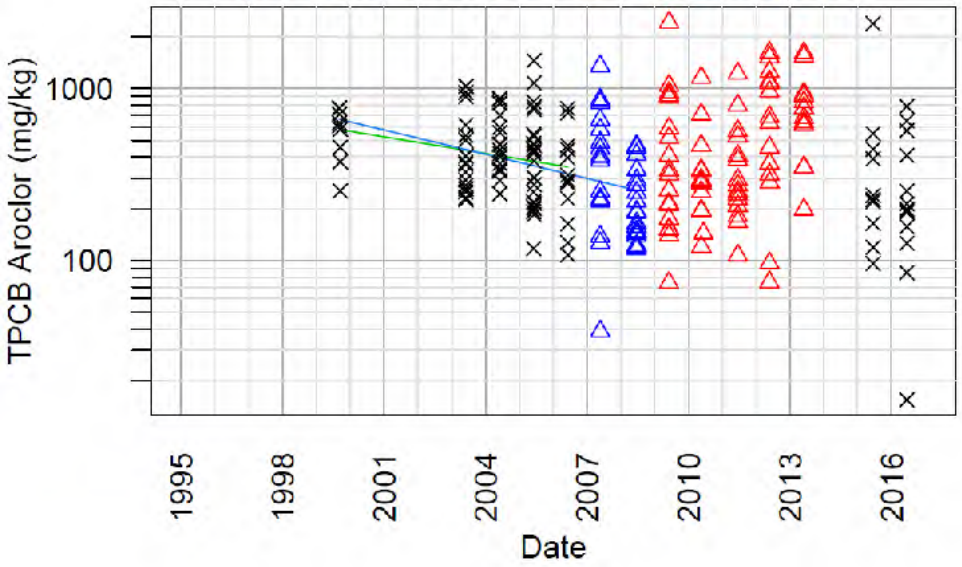


Figure 3J: River Section 2 Pumpkinseed
Coefficient: −17.968 %
Half life: 3.858

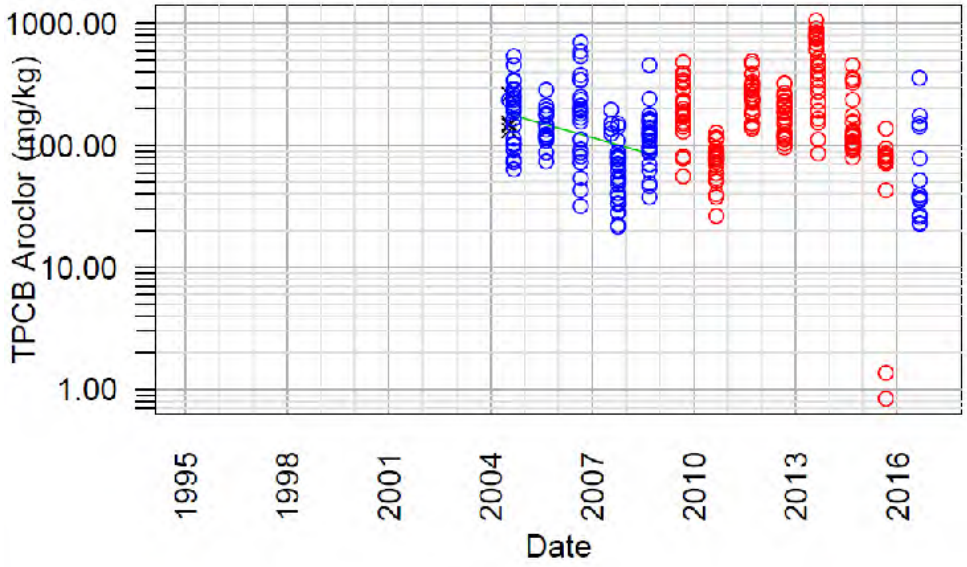


Figure 3K: River Section 3 Largemouth Bass
Coefficients: A −6.275 %, B −9.538 %
Half Life: A 11.046 years, B 7.267 years

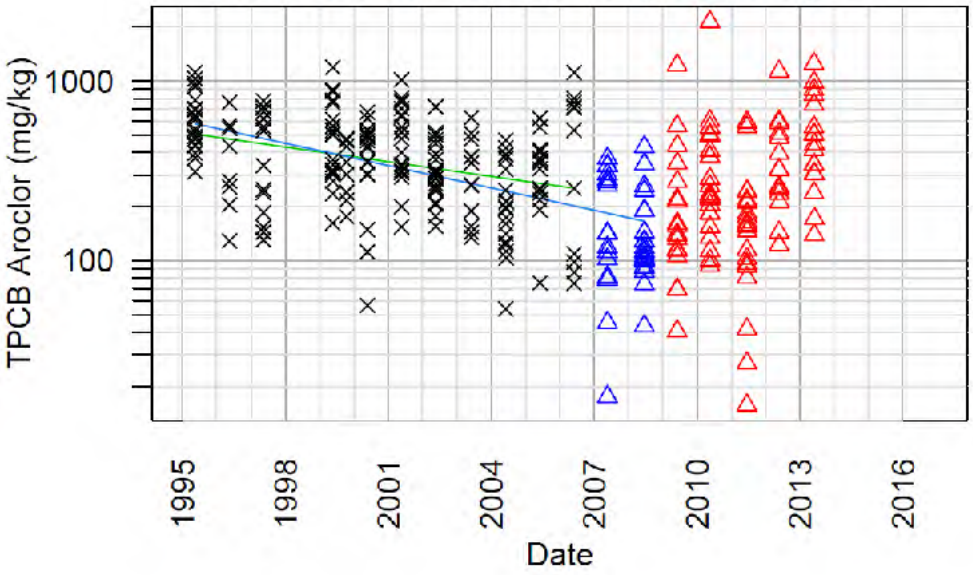
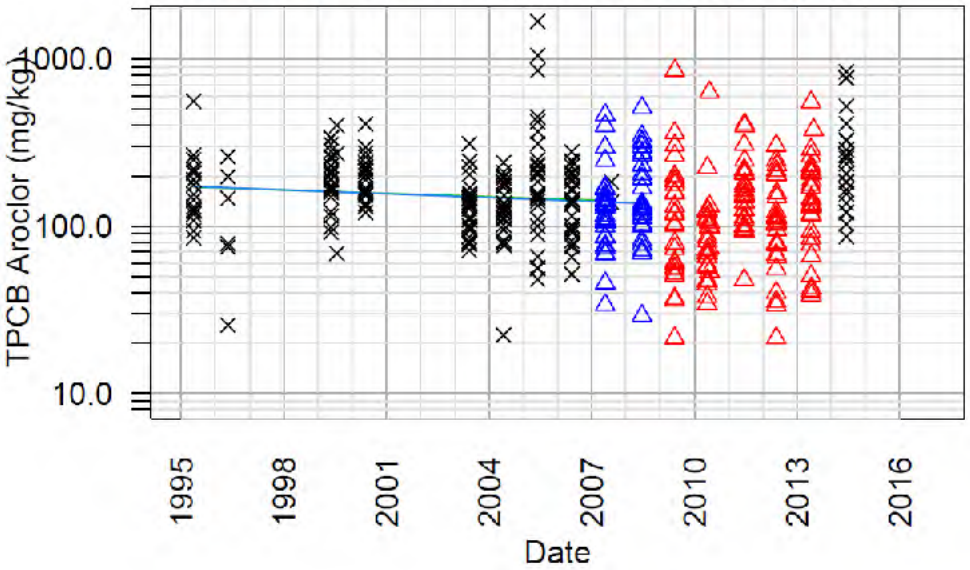


Figure 3L: River Section 3 Brown Bullhead
Coefficients: A −1.539 %, B −1.79 %
Half Life: A 45.035 years, B 38.723 years



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 3M: River Section 3 Yellow Perch
Coefficients: A −13.033 %, B −12.339 %
Half Life: A 5.318 years, B 5.617 years

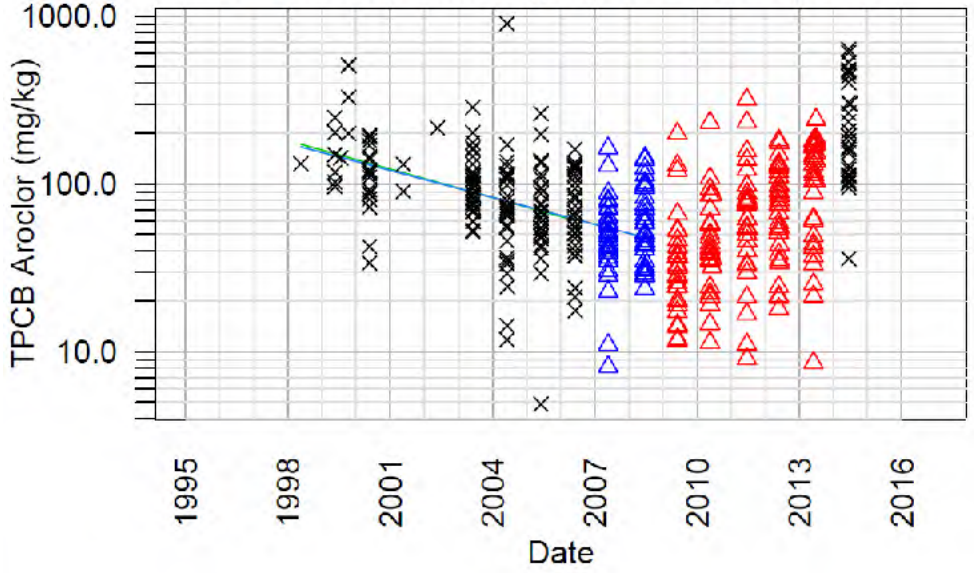


Figure 3N: River Section 3 Smallmouth Bass
Coefficients: A 4.16 %, B 0.376 %
Half Life: A −16.662 years, B −184.468 years

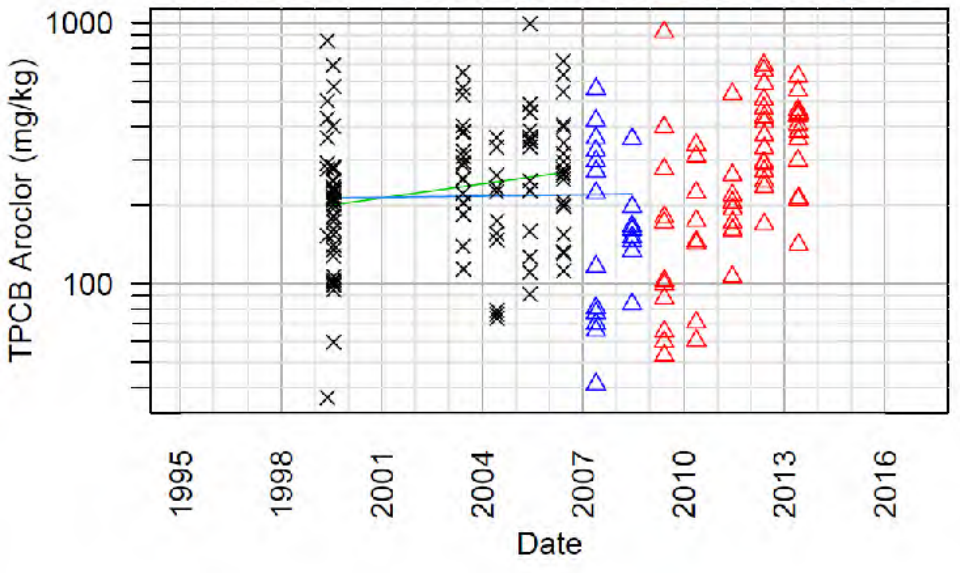
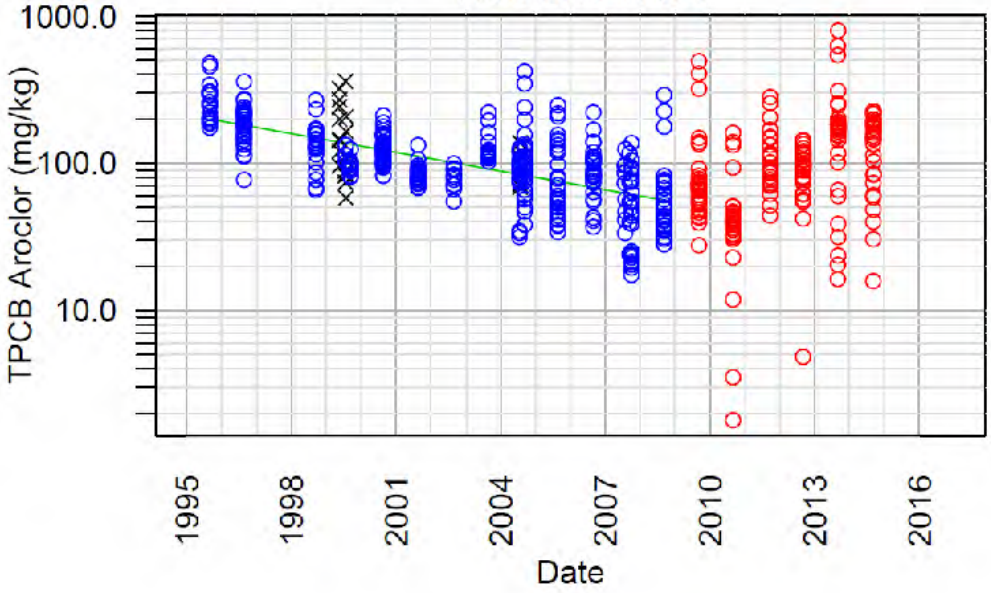


Figure 3O: River Section 3 Pumpkinseed
Coefficient: −9.761 %
Half life: 7.101



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging Trend A Trend B

Figure 4A: RS 1 Largemouth Bass

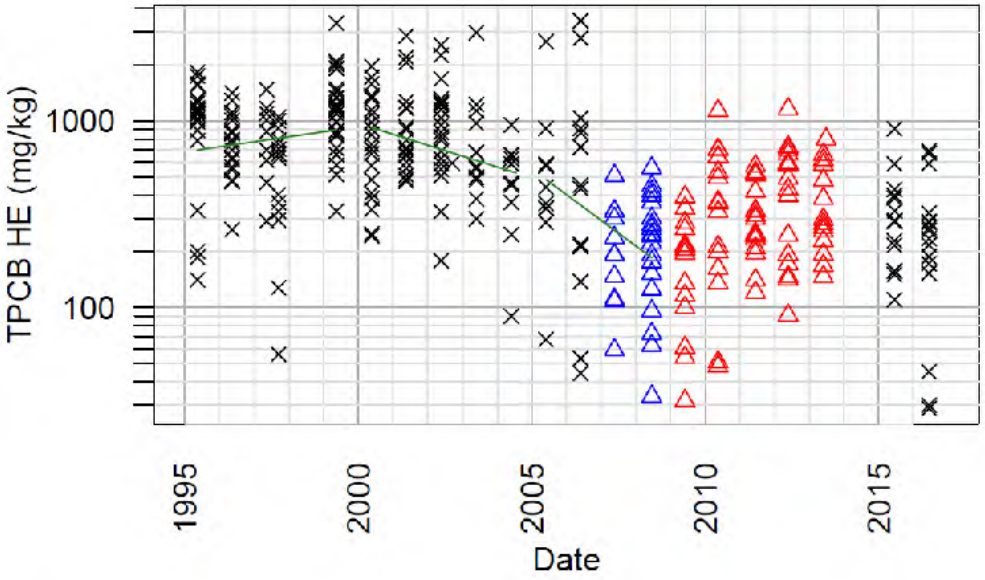


Figure 4B: RS 1 Brown Bullhead

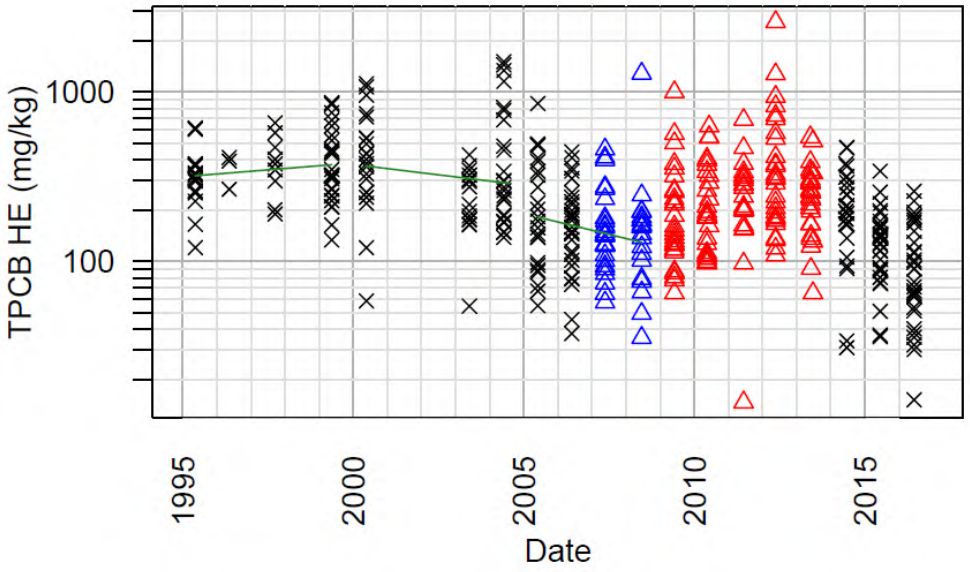


Figure 4C: RS 1 Yellow Perch

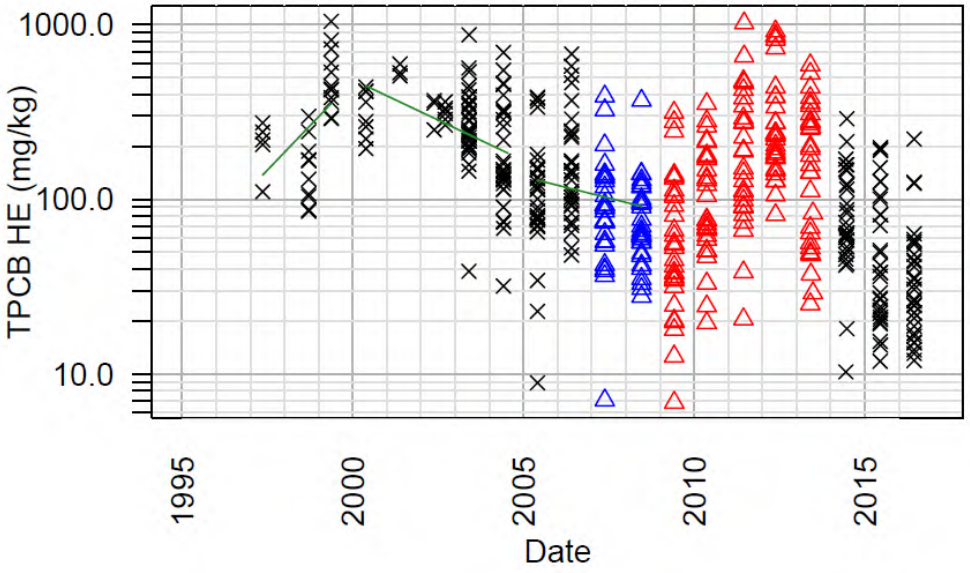
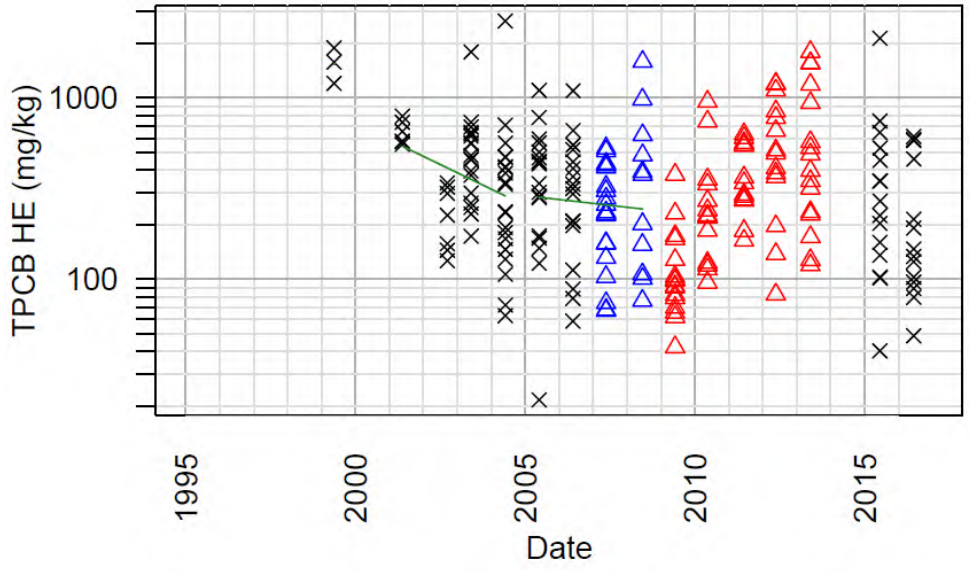


Figure 4D: RS 1 Smallmouth Bass



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging

Figure 4E: RS 1 Pumpkinseed

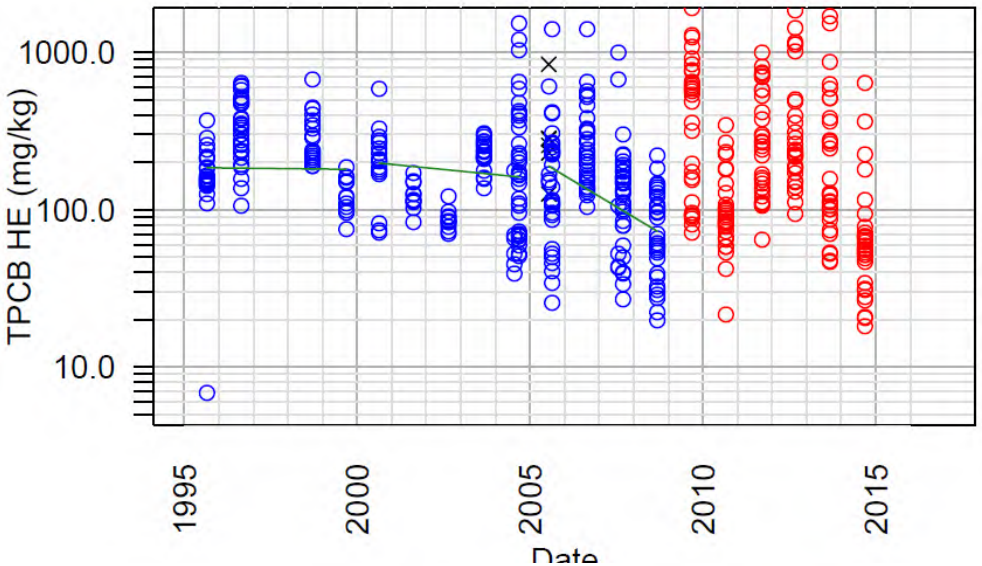


Figure 4F: RS 1 Spottail Shiner

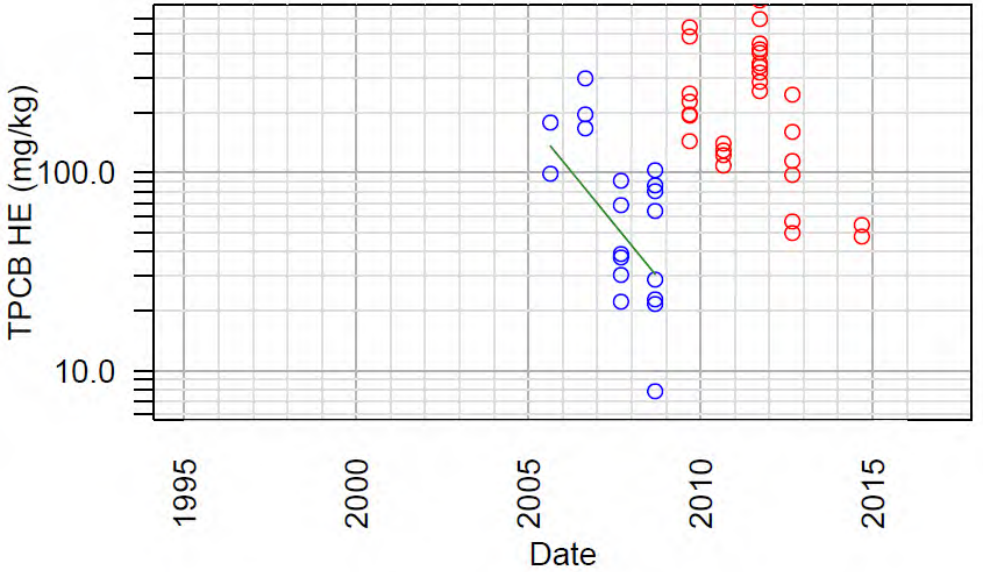


Figure 4G: RS 2 Largemouth Bass

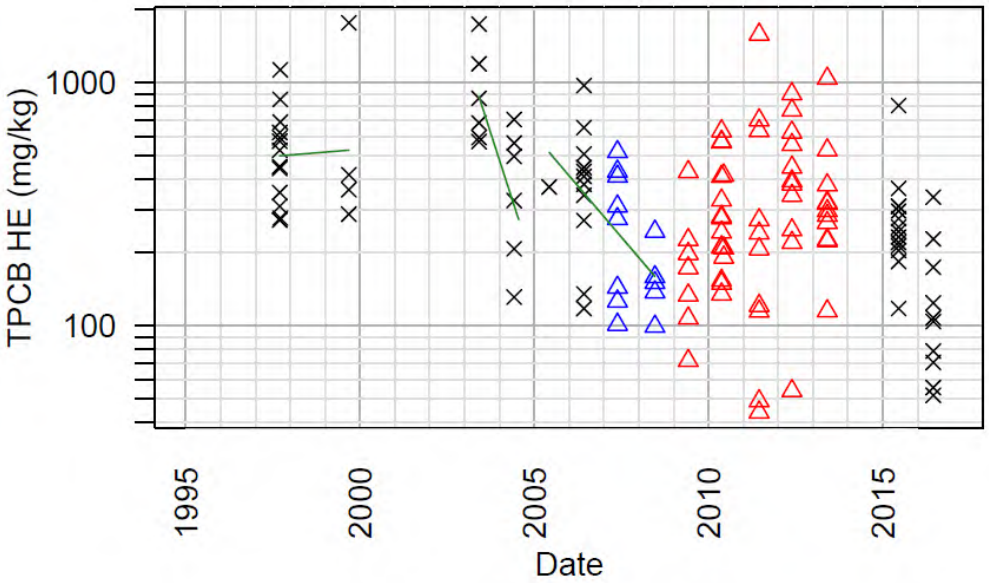
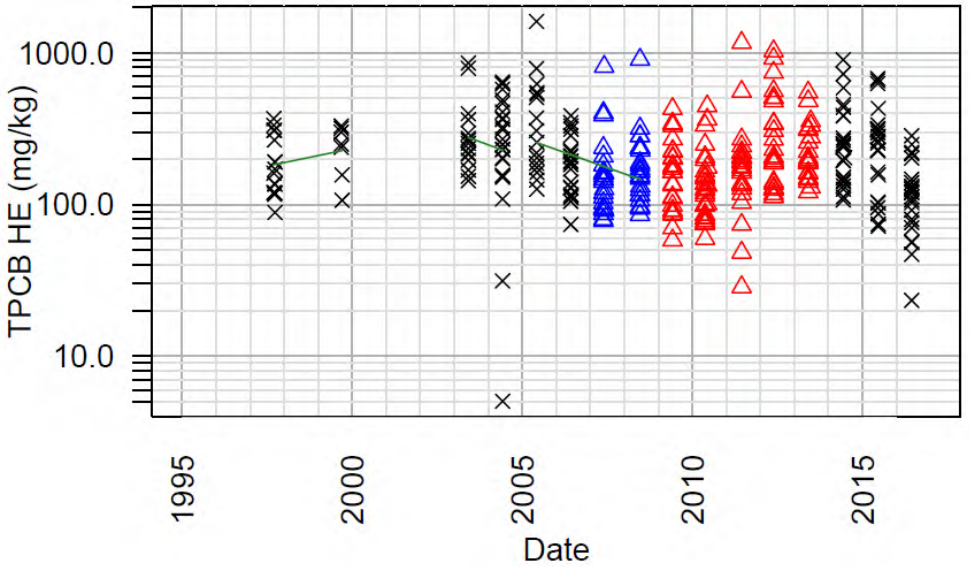


Figure 4H: RS 2 Brown Bullhead



Sample Types: Standard Rib Out: MNA Rib Out: Dredging Whole Body: MNA Whole Body: Dredging

Figure 4I: RS 2 Yellow Perch

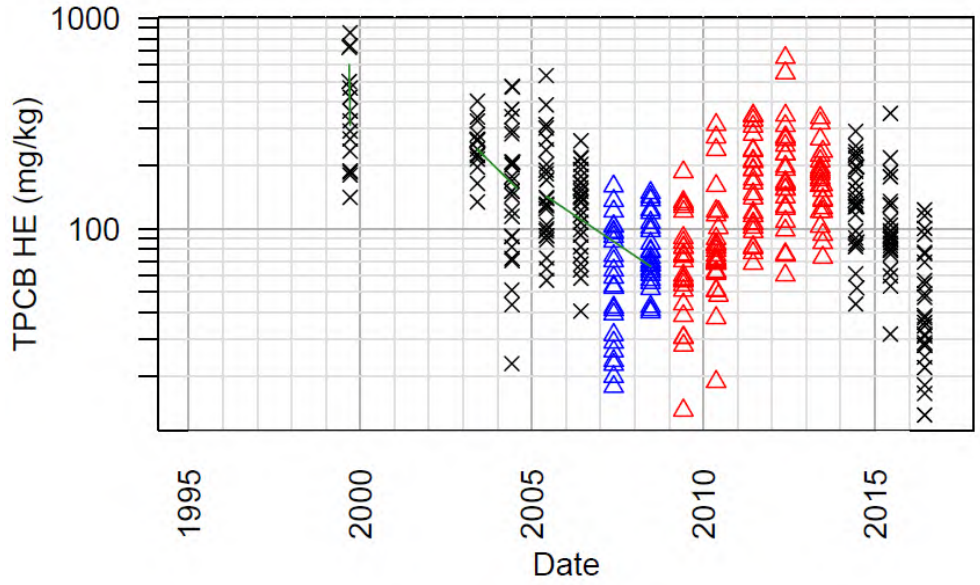


Figure 4J: RS 2 Smallmouth Bass

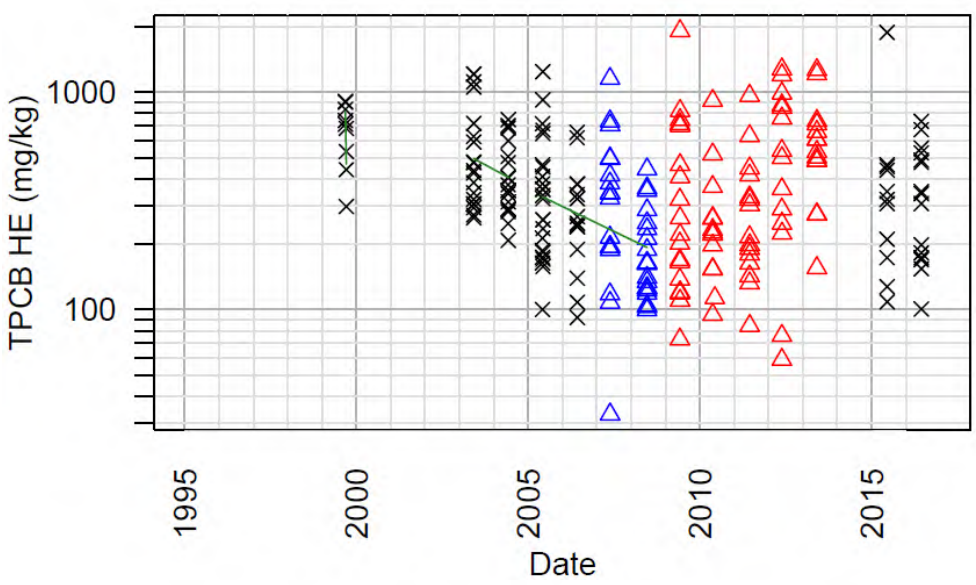


Figure 4K: RS 2 Pumpkinseed

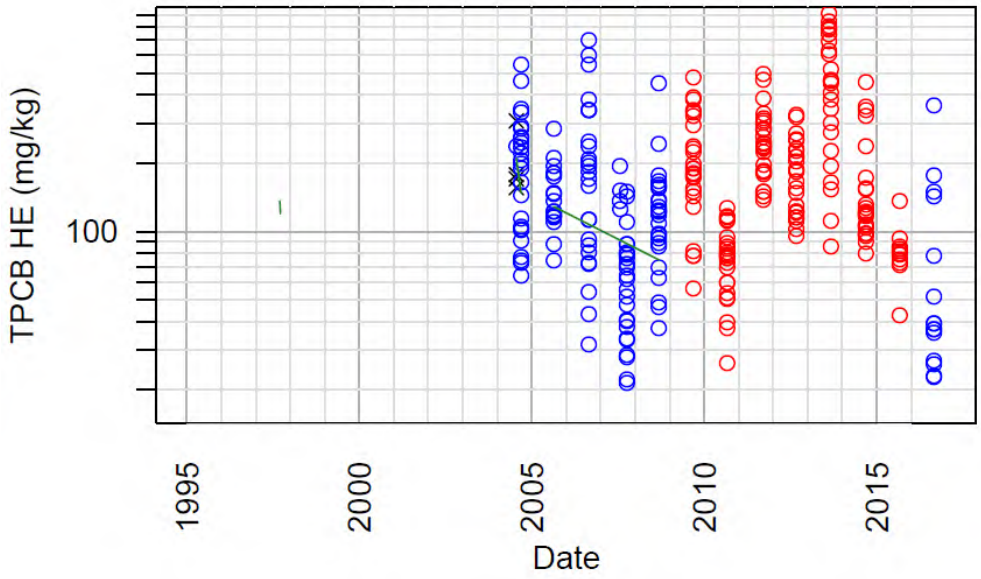
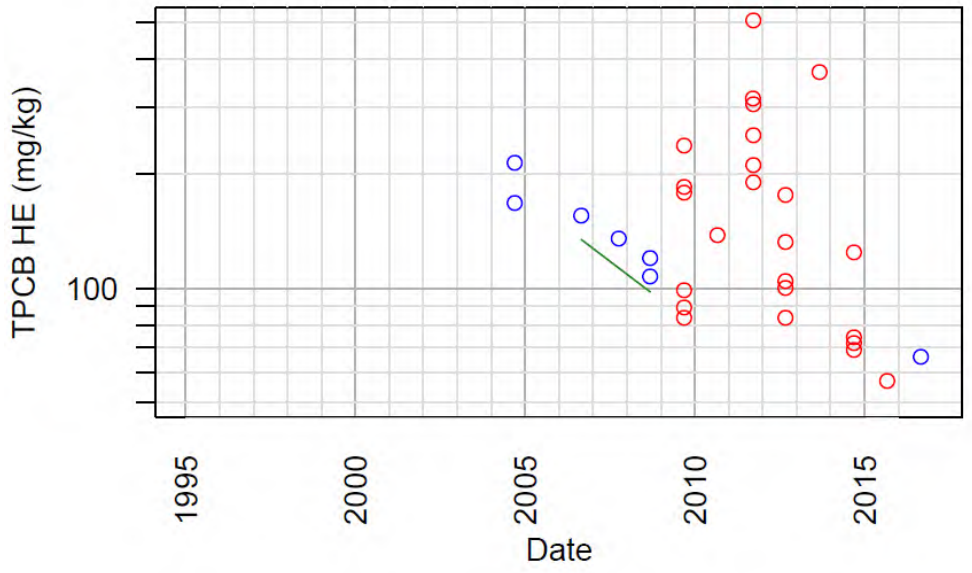


Figure 4L: RS 2 Spottail Shiner



Sample Types: Standard | Rib Out: MNA | Rib Out: Dredging | Whole Body: MNA | Whole Body: Dredging

Figure 4M: RS 3 Largemouth Bass

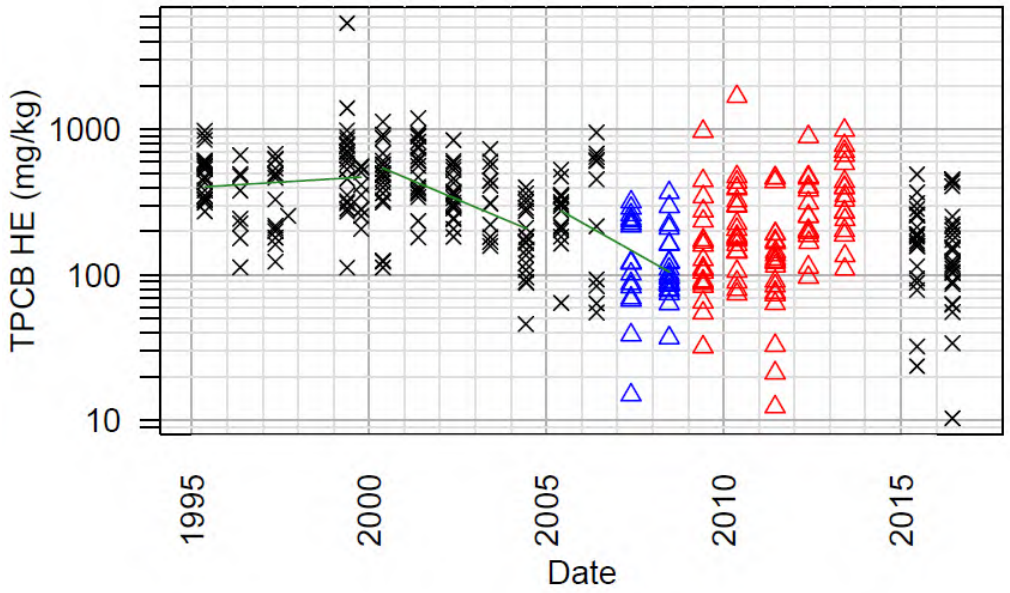


Figure 4N: RS 3 Brown Bullhead

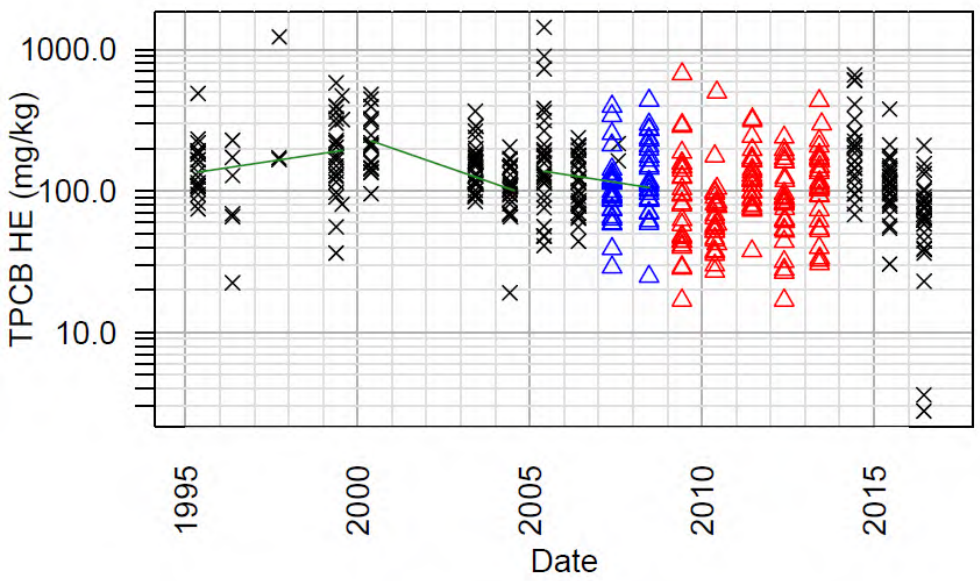


Figure 4O: RS 3 Yellow Perch

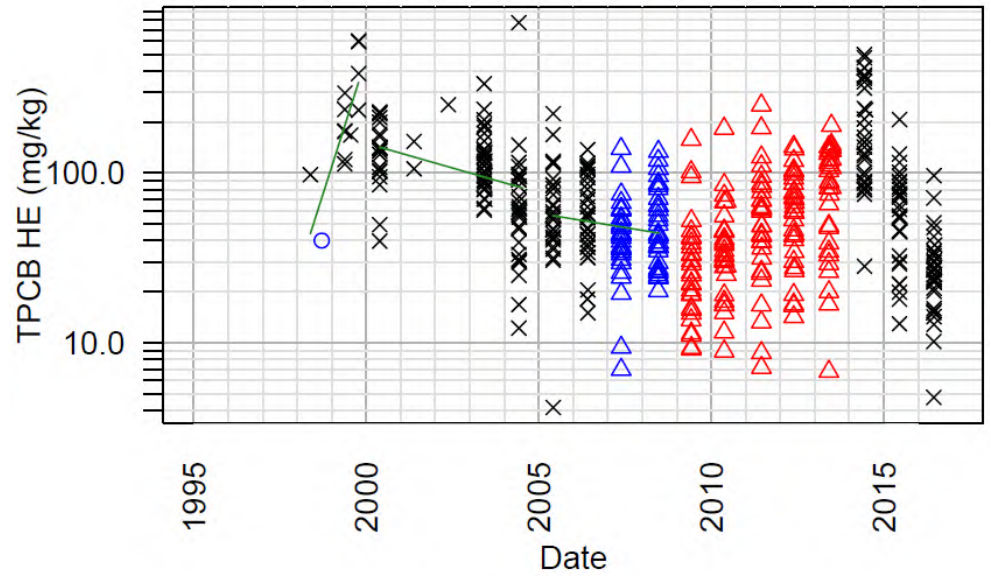
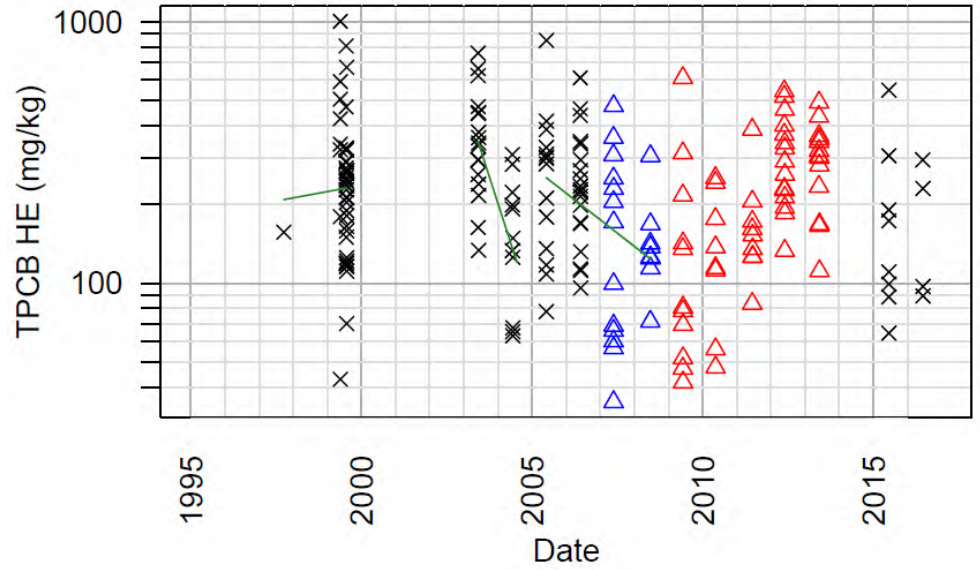


Figure 4P: RS 3 Smallmouth Bass



Sample Types: × Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging

Figure 4Q: RS 3 Pumpkinseed

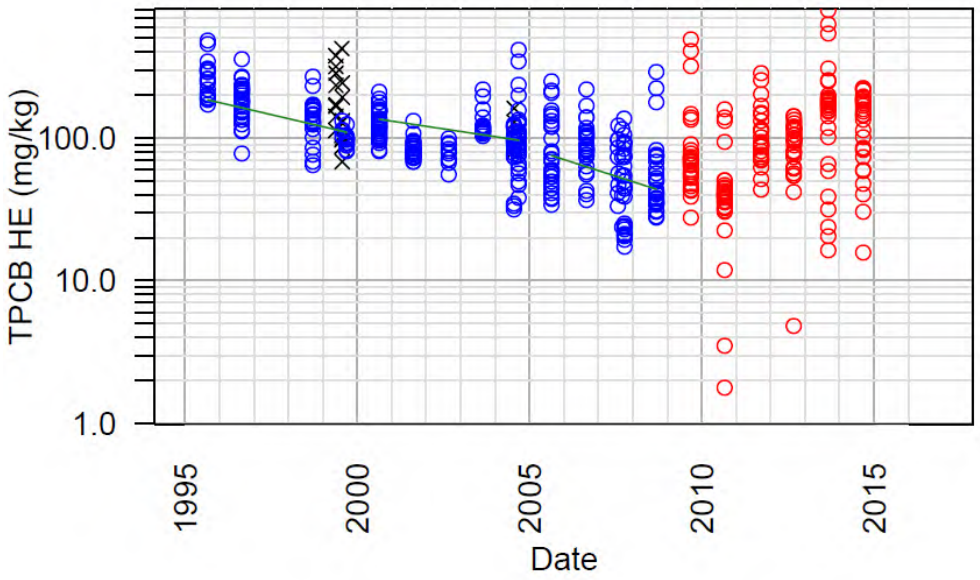
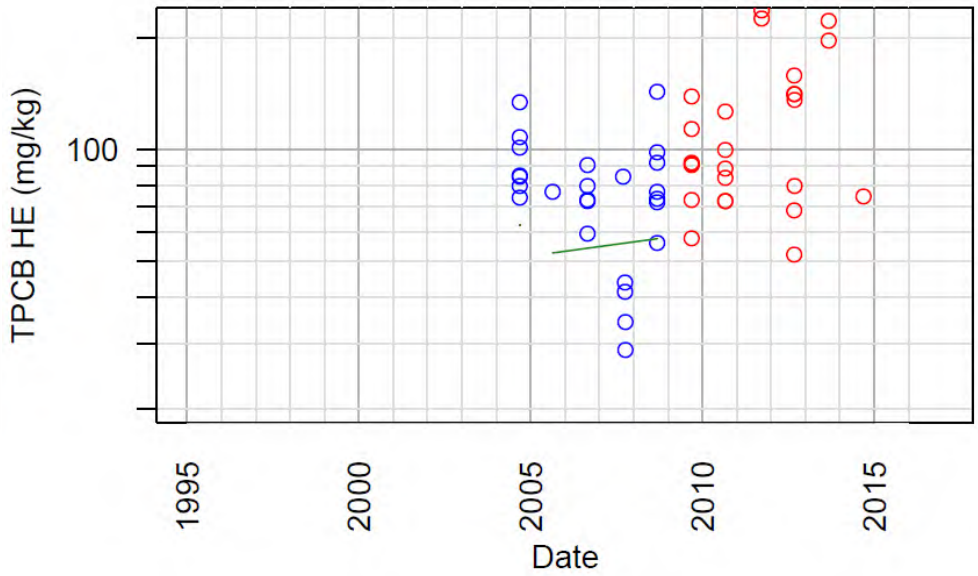


Figure 4R: RS 3 Spottail Shiner



Sample Types: ✕ Standard △ Rib Out: MNA △ Rib Out: Dredging ○ Whole Body: MNA ○ Whole Body: Dredging

Figure 5A: Annual Mean TPCB Concentration, River Section 1 Largemouth Bass

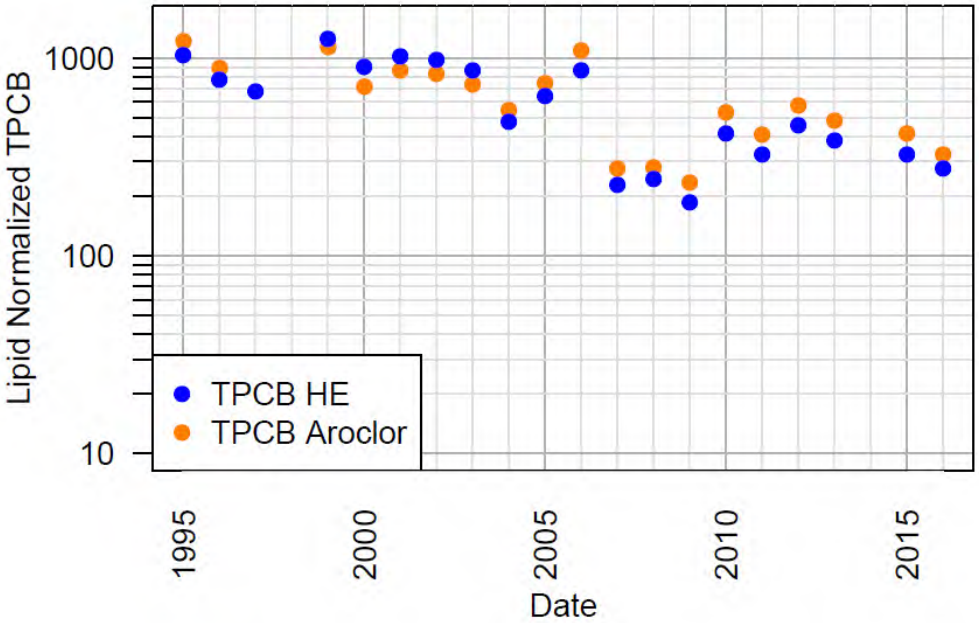


Figure 5B: Annual Mean TPCB Concentration, River Section 1 Brown Bullhead

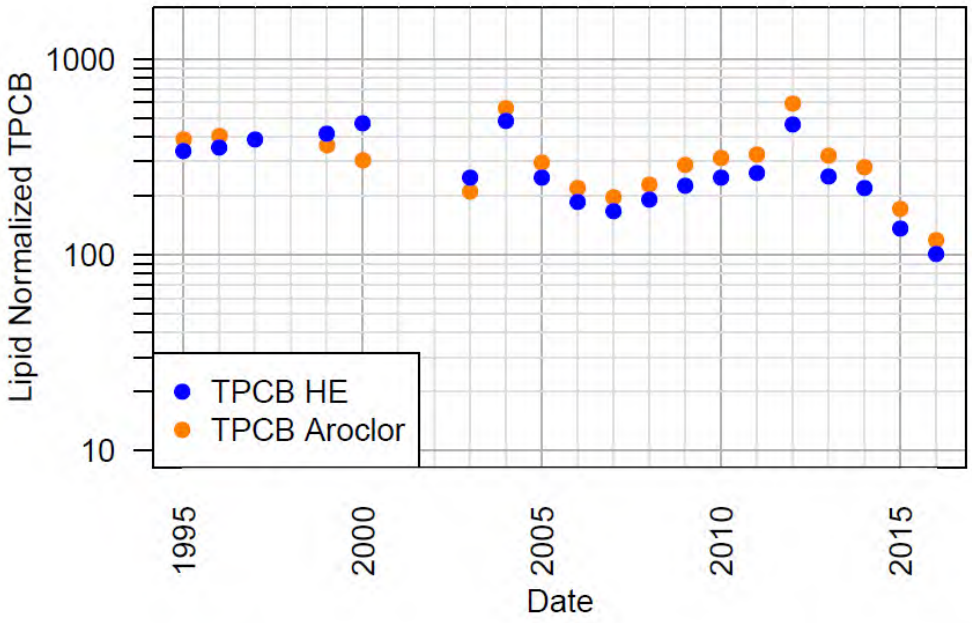


Figure 5C: Annual Mean TPCB Concentration, River Section 1 Yellow Perch

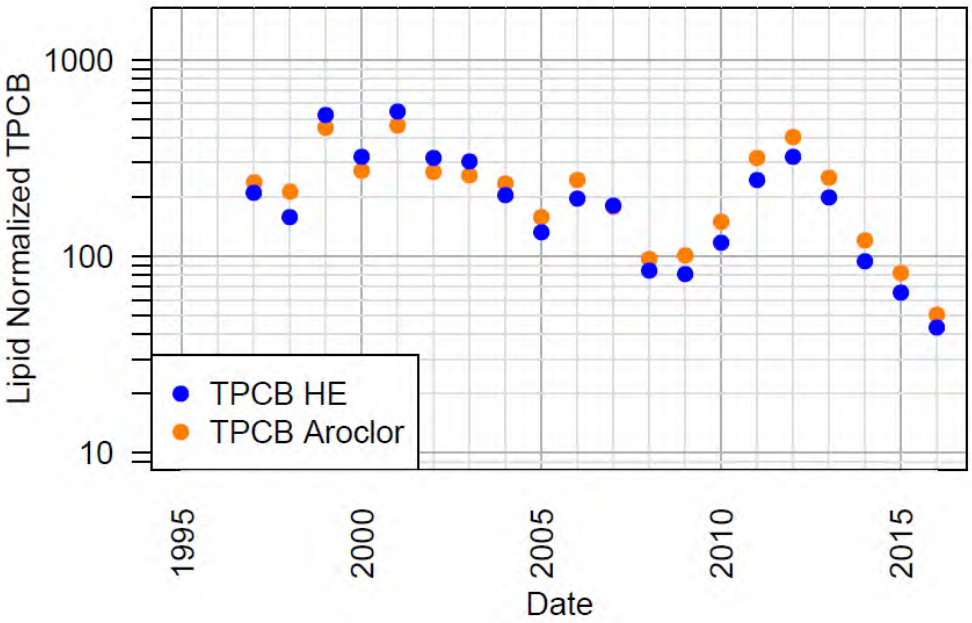


Figure 5D: Annual Mean TPCB Concentration, River Section 1 Smallmouth Bass

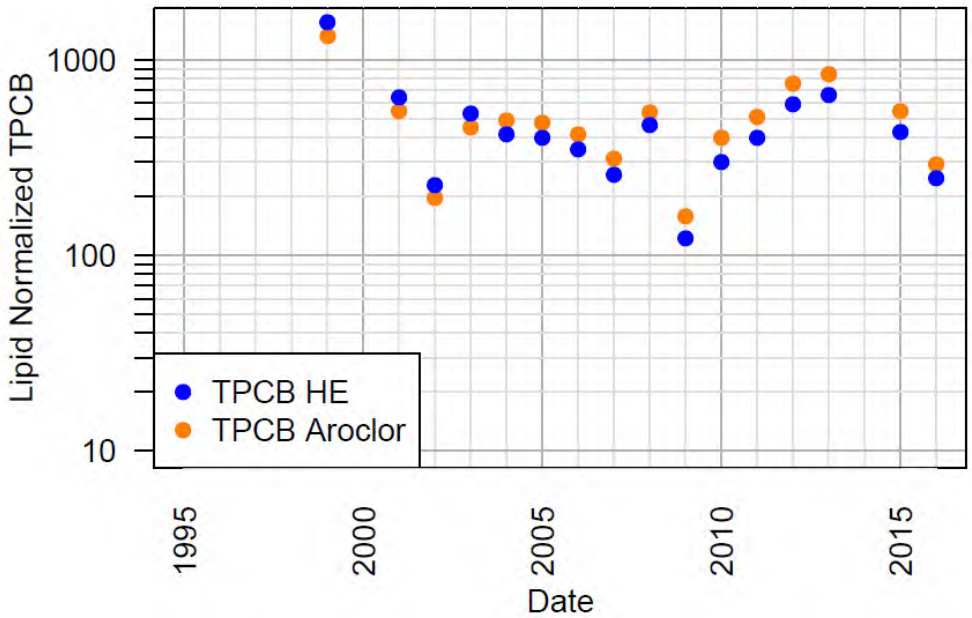


Figure 5E: Annual Mean TPCB Concentration, River Section 1 Pumpkinseed

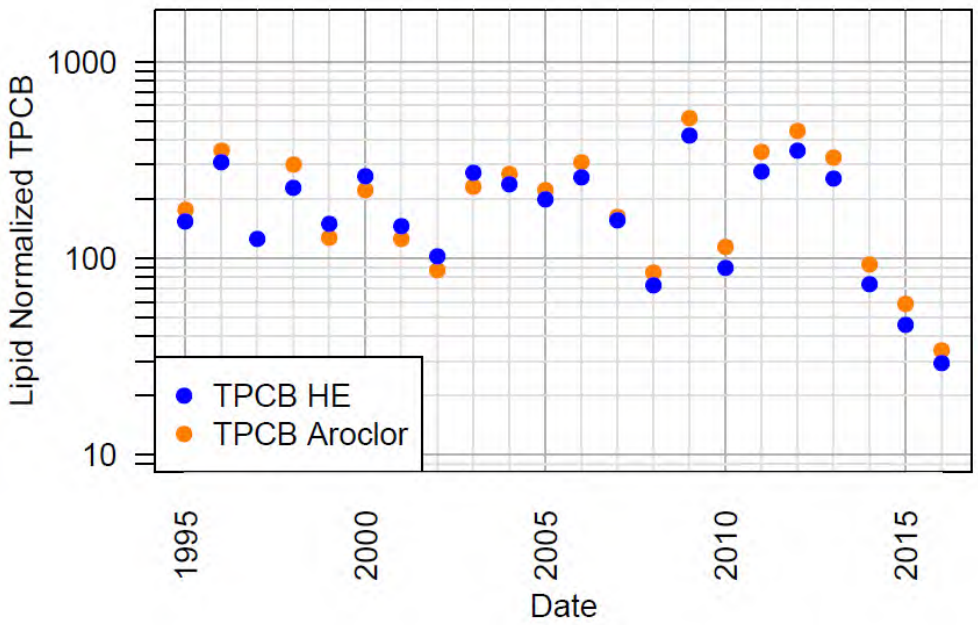


Figure 5F: Annual Mean TPCB Concentration, River Section 1 Spottail Shiner

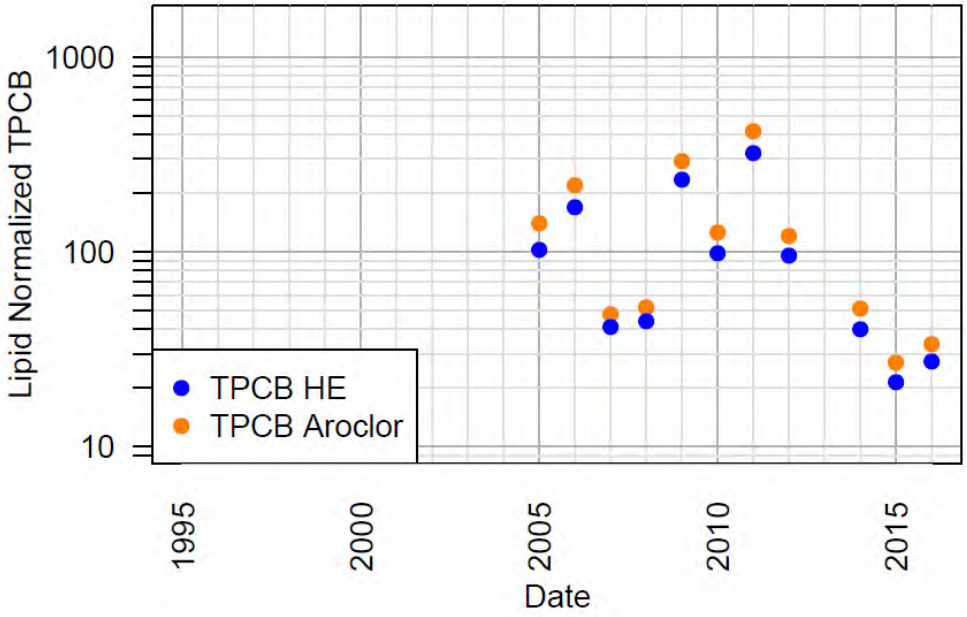


Figure 5G: Annual Mean TPCB Concentration, River Section 2 Largemouth Bass

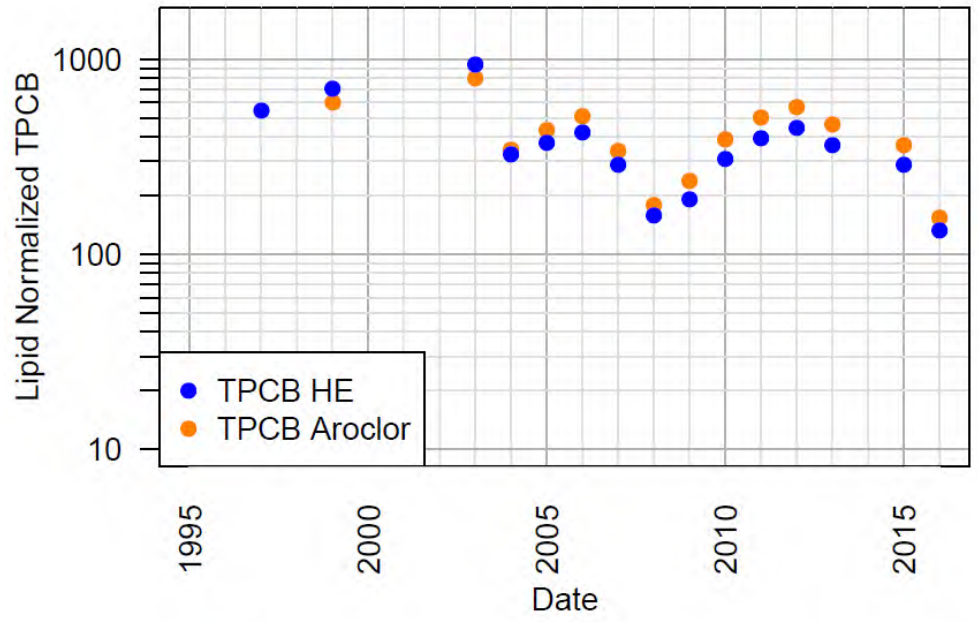


Figure 5H: Annual Mean TPCB Concentration, River Section 2 Brown Bullhead

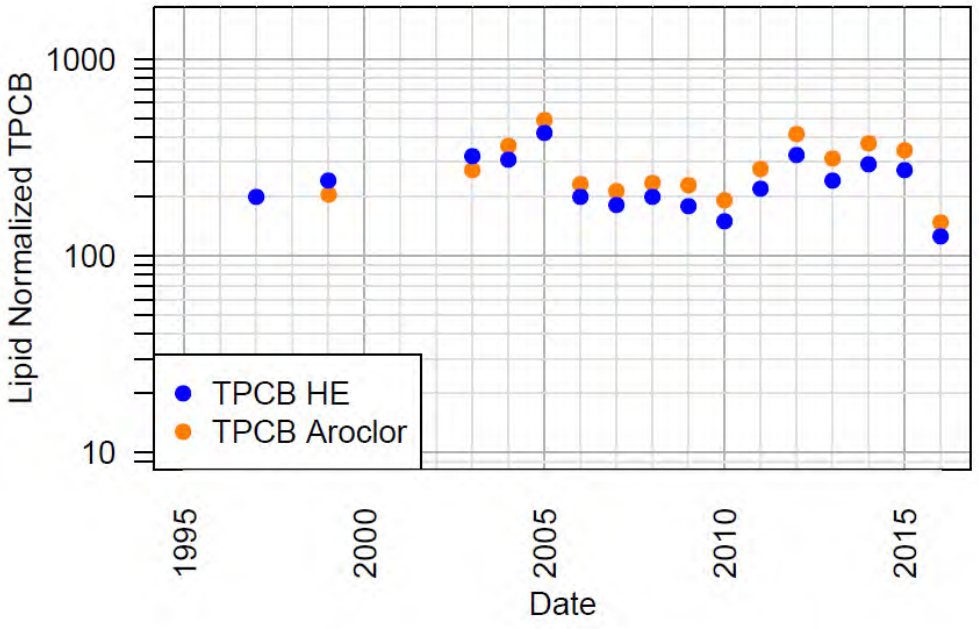


Figure 5I: Annual Mean TPCB Concentration, River Section 2 Yellow Perch

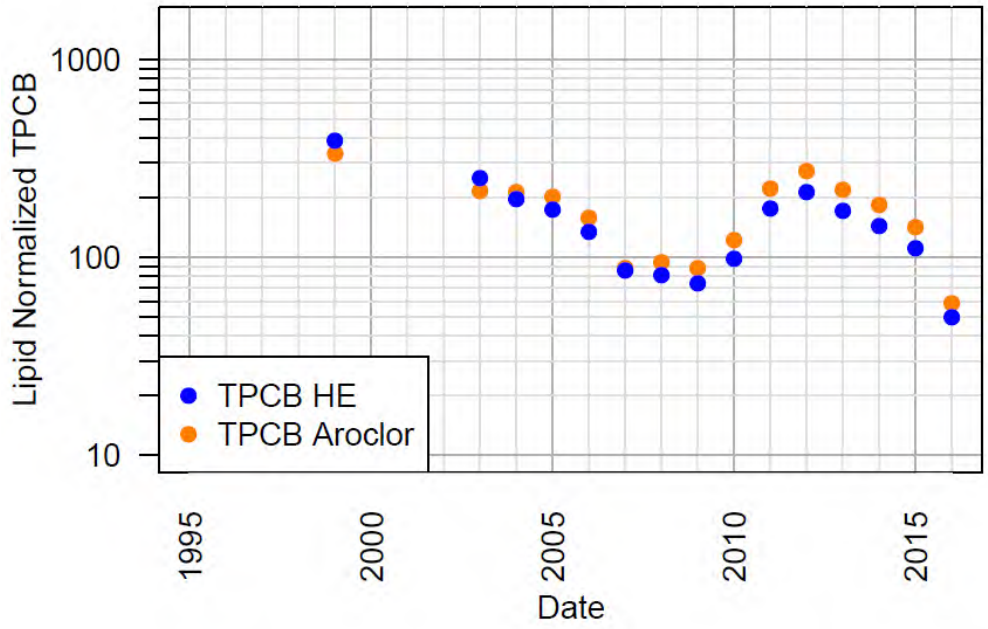


Figure 5J: Annual Mean TPCB Concentration, River Section 2 Smallmouth Bass

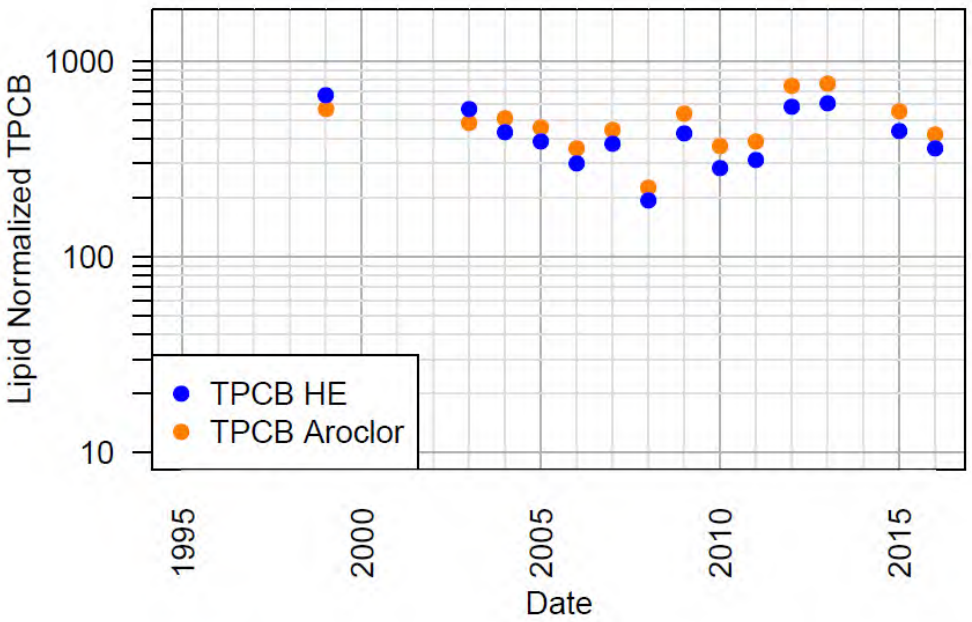


Figure 5K: Annual Mean TPCB Concentration, River Section 2 Pumpkinseed

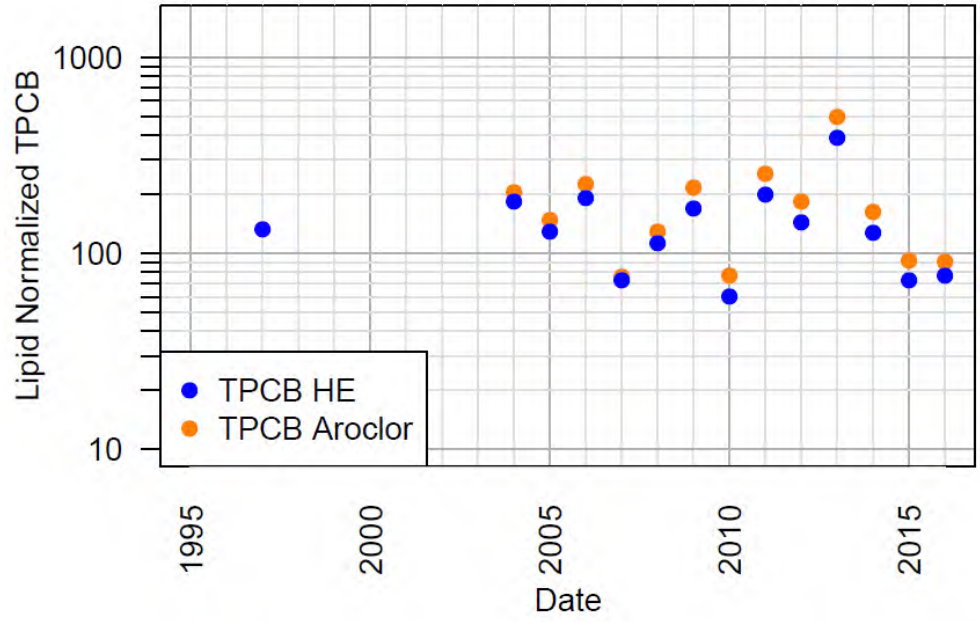


Figure 5L: Annual Mean TPCB Concentration, River Section 2 Spottail Shiner

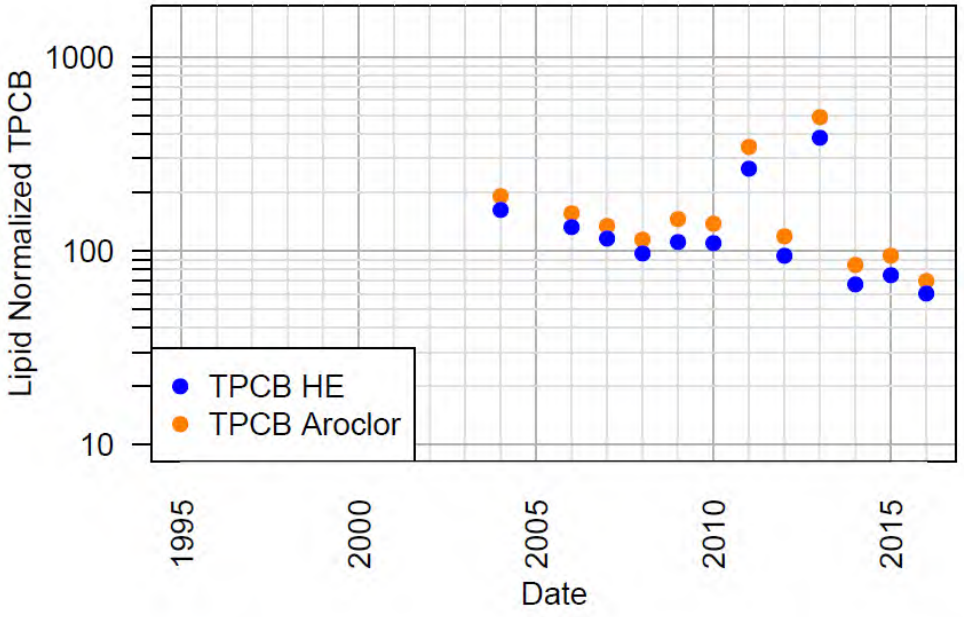


Figure 5M: Annual Mean TPCB Concentration, River Section 3 Largemouth Bass

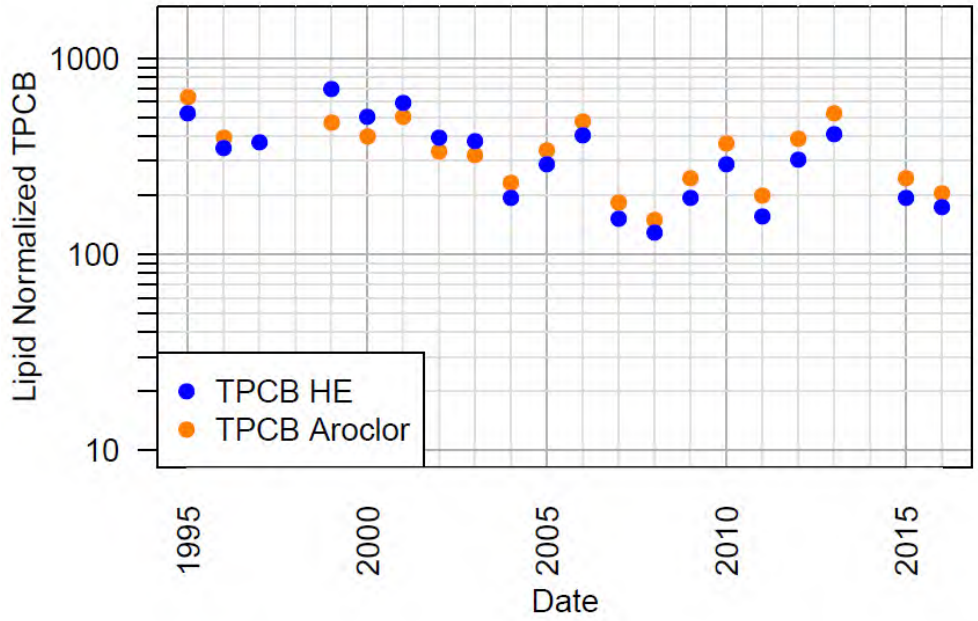


Figure 5N: Annual Mean TPCB Concentration, River Section 3 Brown Bullhead

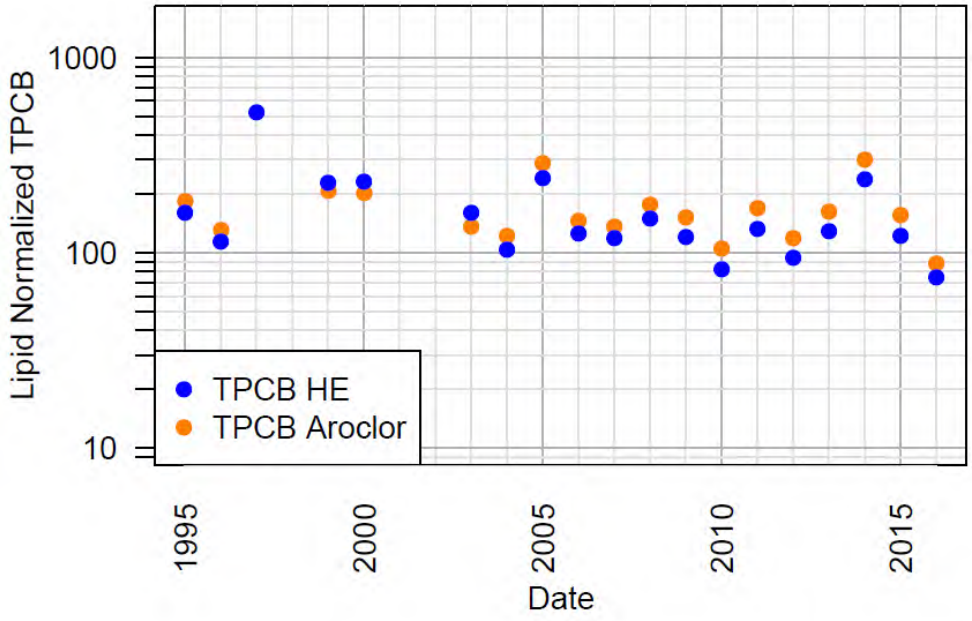


Figure 5O: Annual Mean TPCB Concentration, River Section 3 Yellow Perch

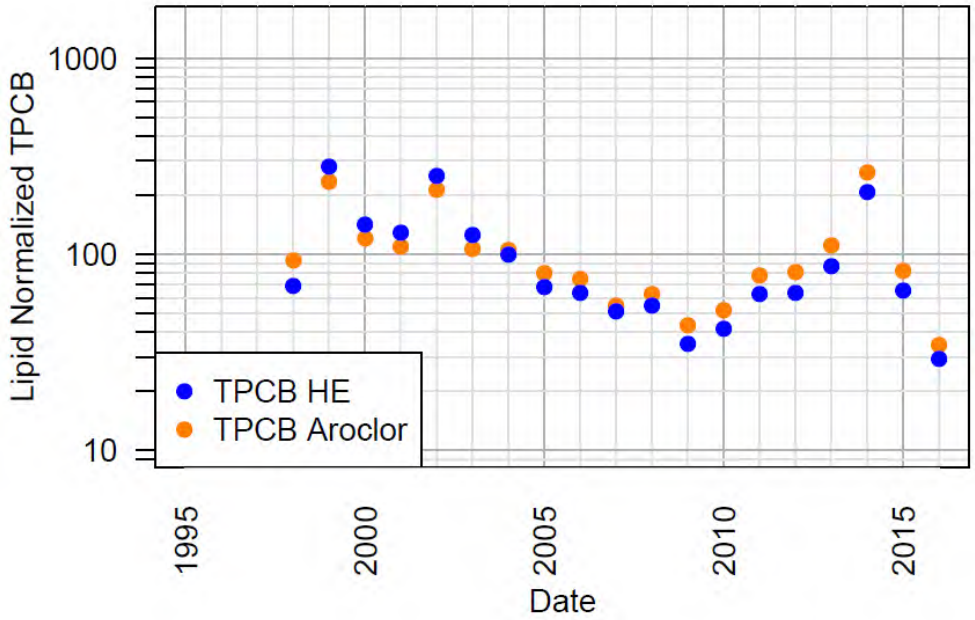


Figure 5P: Annual Mean TPCB Concentration, River Section 3 Smallmouth Bass

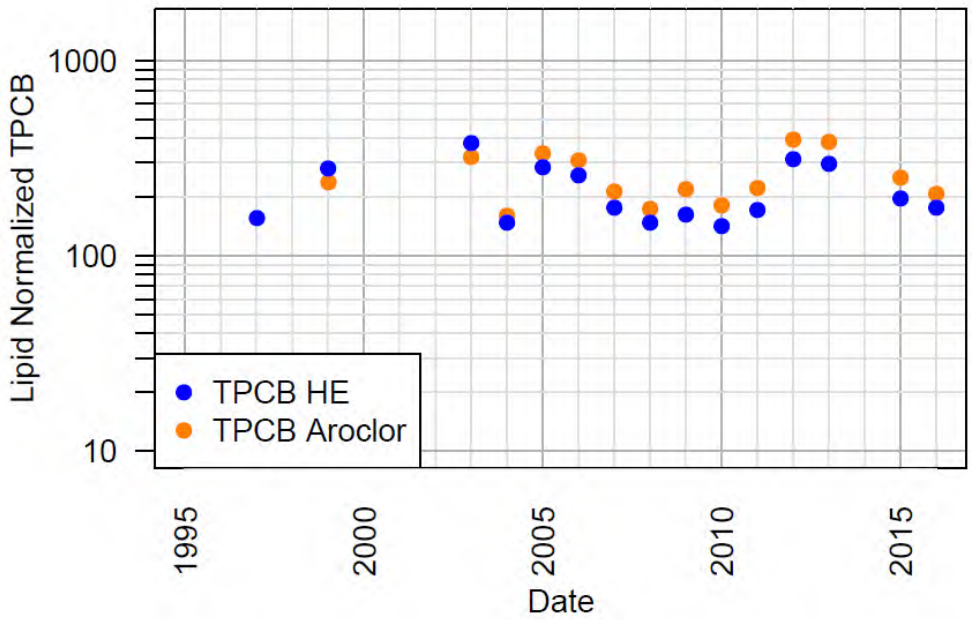


Figure 5Q: Annual Mean TPCB Concentration, River Section 3 Pumpkinseed

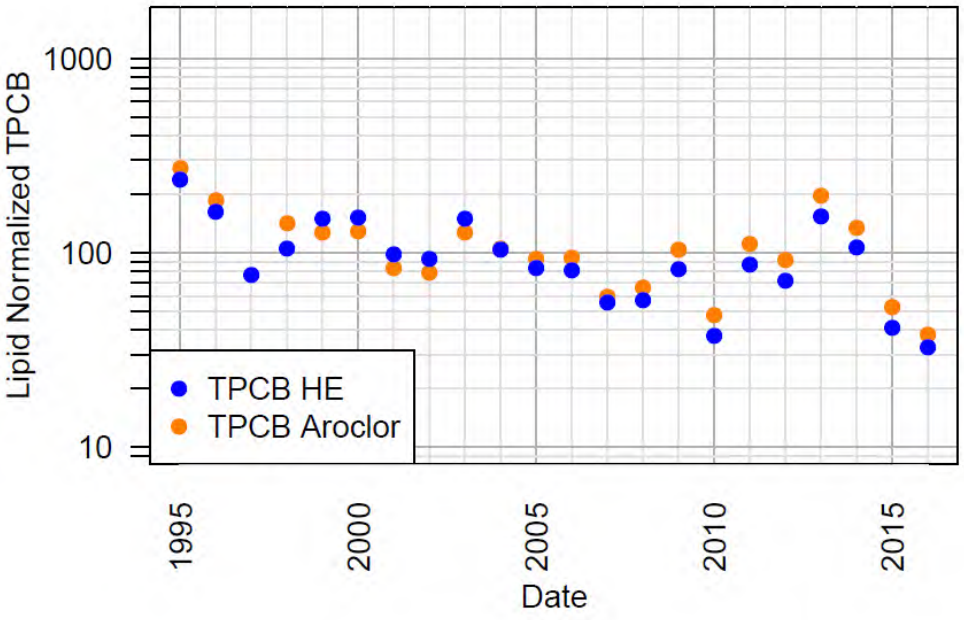
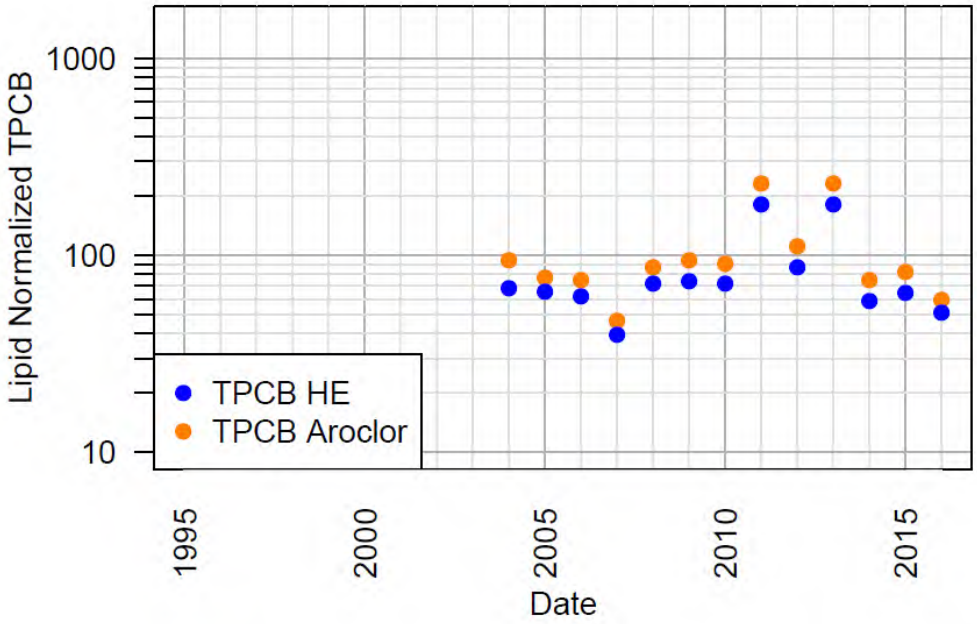


Figure 5R: Annual Mean TPCB Concentration, River Section 3 Spottail Shiner



Society of Saint Ursula

www.societyofstursula.org

50 Linwood Road
Rhinebeck NY 12572

Phone 845 876 2341
Fax 845 876 6544

15 August 2017

EPA Region 2
Mr. Gary Klawinski
290 Broadway
New York, NY 10007

Dear Director Gary Klawinski,

I am a resident of Rhinebeck, NY. I am very concerned about the premature conclusion of the PCB dredging of the upper Hudson. The Hudson River Superfund cleanup has not done the job it was meant to do – to secure the health of the river, its wildlife and the people living along it. PCB contamination in the river remains a significant threat to public health and prosperity as it has for nearly 80 years.

The current levels of contamination in fish, sediment and water are much higher than expected and the lower Hudson River saw little benefit or impact from the dredging project. The only appropriate conclusion for these conditions is "not protective."

It is understood that the original cleanup plan anticipated that some PCBs would be left in the river. However, the EPA discovered - after the remedy was determined - that there was three to five times more contamination in the Upper Hudson than previously estimated. Despite this, the EPA did not expand the cleanup. As a result, despite six years of dredging contamination left in the river is significantly higher than expected.

Riverfront residents of mid and downriver counties, especially those who subsist on the river's fish, face the same health threats today they did before dredging commenced. At the very least, you must undertake an immediate study of downriver contamination and plan for appropriate remedial action.

It is very clear that more data is needed to determine if fish will recover in the reasonable timeframes as agreed upon in the Record of Decision. I urge the EPA to also follow your agency's own guidance for Five-Year Reviews and include credible data and analyses conducted by New York State and federal agencies.

Sincerely,

Kathleen A. Donnelly SU

Kathleen A Donnelly, SU

Historic Hudson - Hoosic Rivers Partnership

Gary Klawinski
Hudson River Field Office
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Gary;

The Historic Hudson-Hoosic Rivers Partnership is a non-profit organization incorporated by the New York State Legislature representing thirty communities within the state-designated preserve. The Partnership fosters collaborative projects with pertinent non-profit and governmental entities with an emphasis on both agricultural and open space protection, economic and tourism development, and the protection and interpretation of our natural and cultural heritage. For us, the river is an important part of our past, our daily lives, and our future.

PCB contaminants within the Hudson River, Champlain Canal, and the floodplain is a detriment to all of our communities. Riverfront parks and boat launches have signs that warn the public of danger. Our plans to organize a fishing tournament are met with replies that the water is too contaminated. Opportunities to utilize the canal and river for freight are dashed because the mandatory depth of the canal cannot be maintained because proper removal of PCB-laden soils. It is clear, that the remedy has not been protective of the long term or even immediate health of our river, its inhabitants, or the economic vitality of the communities.

In response to the Five-Year Review Questions:

1. Is the remedy functioning as intended by the decision documents? NO
   a. It is clear from the initial information on habitat and fish samples that it is taking longer for recovery than anticipated. PCB's were far deeper and more dispersed than the ROD anticipated.
   b. Habitat reconstruction has not resulted in repopulation of species within the parameters that the ROD anticipated.
   c. Resuspension and down river redisposition of sediments into the flood plains has not been addressed.

2. Are the exposure assumptions, toxicity, data, cleanup levels, and remedial actions objections used at the time of the remedy selection still valid? NO
   a. The variability of testing methods has tainted the results to date.
   b. The ROD left behind significant deposits throughout the upper Hudson that are not part of the cleanup. Those deposits are in excess of standards used in other PCB cleanup projects and leave our river subject to additional cleanup costs every time we attempt a project – whether residential or public.
   c. Without data on the level, depth and breadth of contamination of the floodplain this review is fragmented and ill informed. As monitoring

reflected during dredging there were numerous occasions of resuspension of PCB's exceed performance standards often near shore and adjacent to the floodplain. In addition, no assessment of the impacts associated with compliance with the NYS Constitutional requirements of the NYS Canal System and the need for operational dredging of Champlain Canal has been considered or presented.

3. Has any other information come to light that could call into question the protectiveness of the remedy? MOST DEFINITELY YES
   a. The original Champlain Canal was not included in the remedy and it is hydrologically part of the Hudson River. Significant PCB concentrations were found and partially removed from the canal north of Lock 5, yet the original canal was ignored. The original canal is now so silted in with blocked culverts and dead fall that it is often stagnant and overflows the banks during heavy storms.
   b. The ROD ignored the industrial and recreational use of the river when it required dredging only to the depth of the contamination – ignoring the fact that New York State has been unable to dredge to required depths for decades. Additionally, the EPA (with the ROD as an excuse) refilled areas that had silted in over the decades – impeding industrial and recreational use.
   c. The ROD focused on river sections closer to Fort Edward, ignoring contamination of the same toxicity in river sections below Lock 5. Those areas will continue to redeposit PCB's in the upper river, the flood plains and the lower river.
   d. The NYS Canal System has been designated a National Register Property the ability to perform both operational and preventative maintenance on this nationally significant resource has been compromised impacting the integrity of the infrastructure.

For these reasons – I urge the EPA to recognize that the remedy as designed is not protective. Additional dredging is required if those of us in the upper Hudson are to have a clean river. We cannot undertake projects and use of our river with the knowledge that the legacy of PCB's is still lurking in the sediments and floodplains.

Thank You,

Tom Richardson, Partnership Chairperson

The Historic Hudson – Hoosic Rivers Partnership
PO Box 59, Schuylerville, NY 12871
www.HudsonHoosicPartnership.org

Historic Hudson-Hoosic Rivers Partnership
P.O. Box 59
Schuylerville, NY 12871

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31 AUG 2017 PM 1 L

Gary Klawinski
Hudson River Field office
187 Wolf Rd., Suite 303
Albany, NY 12205

12205-113878

WALKWAY OVER THE HUDSON

RECEIVED
AUG 31 2017

August 23, 2017

Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski:

We represent tourism sites along the 200-mile span of the Hudson River Superfund site. The river is the bedrock of the Hudson Valley's current and future economic vitality. It drives the region's multibillion-dollar tourism industry and is in large part responsible for the ongoing recovery of the real estate market in the Lower Hudson since the great recession. The beauty of the river and the myriad parks along it contribute significantly to residents' quality of life and serve as catalysts for attracting visitors and new jobs.

Building upon this momentum depends on a clean, healthy Hudson River. As long as unacceptable levels of PCBs pollute its water, sediment and fish, they hinder lasting economic gains—both the resumption of once-lucrative industries dependent on the river and long-stalled development opportunities along it. More important, they continue to pose a threat to the health of people living in riverfront communities.

For 70 years, the economic, recreational, cultural and scenic values of the Hudson River have been compromised by PCB contamination. This pollution has destroyed a once-vibrant commercial fishing industry, hampered the operation of marinas, led to a severe curtailment of marine transport on the Champlain Canal, tripled the costs of dredging the NY-NJ Harbor, prevented ambitious economic development opportunities on the Upper Hudson similar to those being realized along the Mohawk River, and barred generations of residents and visitors from full enjoyment of this American Heritage River.

For these reasons, we call on the EPA to:

Declare in its Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as the EPA's draft review explicitly states.

Delete the draft review's finding that the remediation "will be protective" in 53 years. The EPA makes this forecast despite admitting eight additional years of research are needed to verify it. Further, data indicate that fish toxicity in the Upper Hudson is almost 300 percent higher than the goal the EPA expected to reach in 2018. If this interim target is so off base, how can the EPA forecast with any reliability that the cleanup "will be protective" in five decades?

Conduct additional cleanup of the Upper Hudson. The draft review fails to incorporate any analysis by the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation showing that the remediation leaves behind contamination equivalent to (in NOAA's words) "a series of Superfund-caliber sites." Both NOAA and the DEC have concluded that additional dredging is needed. An "is not protective" determination will pave the way for this to happen.

Undertake a remedial investigation of the Lower Hudson. The draft review makes clear that PCB levels in fish and sediment in the 160-mile portion of the Lower Hudson have not benefited at all from upriver

dredging. In actuality, downriver contamination is significantly higher than expected. The draft review lays out no plan for investigating and removing this contamination. This oversight must be corrected.

Data confirm that time and nature won't fix this project's shortcomings, as your draft review would lead us to believe. Only additional dredging will make the Hudson healthy as soon as possible. Therefore, we strongly urge the EPA to conclude that the remedy for the entire Hudson River Superfund site is "not protective." Then and only then can we begin to plan for the bright future our children and grandchildren deserve.

Sincerely,

Elizabeth Waldstein-Hart

Elizabeth Waldstein-Hart
Executive Director
Walkway Over the Hudson / ED

PETITION CALLING FOR A NON-PROTECTIVE DETERMINATION REGARDING THE HUDSON RIVER PCB REMEDIATION

To: EPA Administrator Scott Pruitt and Members of Congress

Although better than no clean up, General Electric's removal of toxic PCBs from the upper Hudson River doesn't go far enough to provide New Yorkers with the safe and usable river they deserve. The Environmental Protection Agency's 2005 Consent Decree allowed General Electric to leave what two federal agencies (NOAA and U.S. Fish and Wildlife) have described as "a series of Superfund-caliber sites" in the river that will prevent its full restoration. More needs to be done, not only to restore the river, but also to ensure economic opportunities that would result from lifting long-standing fishing restrictions, reopening the Champlain Canal to major commercial traffic, and a resurgence of tourism and recreation -- all currently hindered by GE's PCBs. Last week, EPA released for comment a misguided decision that the 2009 – 2015 PCB remediation was working as designed in 40 miles of the Hudson above the Troy dam, although "not yet protective" of human health and the environment. However, three times as much PCB-containing sediment is left in the river than originally projected. EPA is willing to speculate and leave it to "Mother Nature" to take care of the remaining PCBs discharged by GE into the Hudson, closing down a multi-million dollar fishing industry. EPA and GE must finish the job -- and not leave New Yorkers holding the bag. EPA should require GE to complete a more robust cleanup to ensure the Hudson River recovers and that PCB levels in fish drop to levels safe for human consumption as soon as possible. EPA and GE have a moral and legal responsibility to make sure the Hudson River is safe, to restore it to its former health and usability, and to ensure GE compensates New Yorkers for the decades of damage it has caused.

That's why we, the undersigned, urge EPA to issue a determination of non-protectiveness, which would require a more robust cleanup of Hudson River PCBs.

NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Doug Winkleman		11545	[redacted]	[redacted]
Tom OConnor		12508		
CHARLES JAMES		20968		
Aimee Allen		12090		
JANICE FAGAN		07042		
PAM YARLEY		35126		
ANDY [illegible]		10512		
JOAN ALSOP		07631		
Brian Polcato		01826		
Mark Jackett		11777		

Please return completed petition to Hudson River Sloop Clearwater, 724 Wolcott Ave., Beacon, NY 12508

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Claire Murphy		19460	[redacted]	
David Goodwin		07480		
Clare Levine		11967		
Andrew Engel		10530		
K Hale		11249		
Carlos Garcia		10538		
Page Oliver		10547		
Jenny Atkins		81002		
Jessica Bernath		10549		
Mike Accacella		06902		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Eve Boshnick	Eve Boshnick	19004	[redacted]	[redacted]
Tom Posusney	Tom Posusney	19007		
DIANA FROST	Diana Frost	10518		
DOUG FRIEDMAN	Doug Friedman	1048		
JOSEPA MANDEL	Joseph B Mandel	10027		
RANDI LEIGH	R Leigh	07748		
JAMES MCG				
Amy Hebard	Amy Hebard	05721		
Mike Fedison	Mike Fed	10562		
Melanie Chirigoni	M Chirigoni	11233		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Carole Braunstein		10987	[redacted]	
Martha Trujillo-Tapp		10549		
Kaitlin Tapp		10579		
Susan Wiener		10954		
Arlen Marks		19344		
MYRIAM BOUCHARD		12561		
Michael Karlsson		10605		
Nicole McNally		10511		
Michelle Brussel		10960		
Anne Larsen		22207		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
RON ELTON	Ron Elton	07631	[redacted]	
Luke Savarstad		12472		
Beth Krueger		06896		
N. Nielsen		06996		
A. WILLIAMS		07030		
M TIFFANY		11566		
J McElwain	J McElwain	11566		
K A Neuman	K. Neuman	10538		
James Neuman		10537		
Jenny Neuman		10009		

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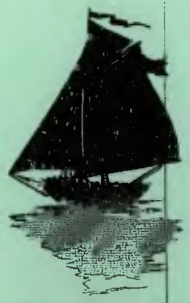

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Ray Bohn		08050	[redacted]	
AUDREY ROSENBLITH		11213		
TANYA BABINSKI		14170		
Alexis Nuccitto		14127		
Daniel Steinman		10926		
NICK FARRON		11211		
Mark Healy		10506		
DAVID WAGENHEIM		10578		
Mike Lowery		13219		
Wendy Russell		11417		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Louise Hazebrouck	Louise Hazebrouck	10509	[redacted]	
Steve Rose	Steve Rose	10509		
Susan Hamilton	Susan Hamilton	05753		
Sean Brown	Sean Brown	19130		
Meryl Brown	Meryl Brown	10968		
Laura Pakala	Laura Pakala	10960		
Peter Grech	Peter Grech	12771		
Brian Staranta	Brian Staranta	06831		
Hope Stanger	Hope Stanger	06902		
James Elliott	James Elliott	10541		

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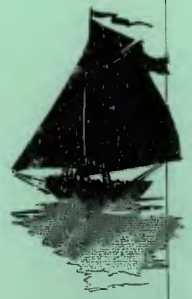

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Eric Harney		12567	[redacted]	
Patricia Perazio		10708		
Michael Iorio		10546		
Michael Simonds		05359		
Blair Waldorf		10001		
Richard Sussman		10960		
Ann Molnar		07753		
Donna Santman		10023		
ERICA ROME		11217		
Scott Sasso		11217		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
STUART EISENBERG	Stuart Eisenberg			
Linda Heitmann	Linda Heitmann			
PAT COLLINS		07013		
Marianne McCabe	M Brahl McCabe	10956		
Meghan Collins		01003		
Rebecca Brahl		20001		
Phillip M. Ciccone		11570		
SCOTT MARTO		1051		
Dan Bender		11763		
Shirley Bush	Shirley Bush	17061		

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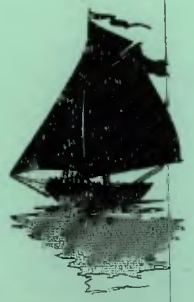

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Hannah Seda		10473	[redacted]	
Nicola Dodd		11215		
Rick Lewis		07003		
Teresa DePuma		19468		
John DeFerrari	John DeFerrari	60601		
Rebecca Cordish		19027		
Sue Lewis		07430		
Kevin Heisig		07430		
Betty Plichta		03748		
Debbie Zinn		10706		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
SR Quackenbush		07090	[redacted]	
Michael Amsalem		10025		
Tammy Amsalem		10524		
Barbara Weill		08904		
Joanne Weill-Greenberg		19127		
Elizabeth Weill-Greenberg		08904		
John C. Parker		01040		
Willow Parker		01040		
Marc Gerlt		11201		
Hailee Wallace		06057		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Jean Bowdish		11106		
Judith Olney		11106		
Mitch Loader		10027		
Sigrid Johannessen		10032		
Christine Pietris		10520		
Phyllis Bannard		12211		
Nicole Pelletier		06903		
Joshua Leonard		10520		
Barbara Gropp		10520		
Lisa Quackenbush		07090		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Anne Stern		05851	[redacted]	
Tim Sevener		07878		
Heather Bock		22902		
Margaret Whitmer-Ferguson		01120		
Sharyn Faranda		12545	[redacted]	
Crystal Kastenell				
Zoë Kaplan-Lewis		11215	[redacted]	
Dan Kaplan		10960		
Kevin Carney		05851		
Dick Crane		20912		
Alison Kofflerdine		10467		

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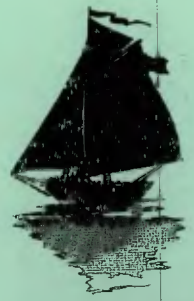

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Laura T Harris		10512	[redacted]	
Jesse Jr Mason		03606		
John O'Donnell		01222		
DEBORAH EBERHARDT		06878		
Rachel Tull		10009		
Amy Wolfe		02478		
Rachel Schwartzman		19119		
Carin Horowitz		10578		
Dakota Peterson		10031		
Tlan Korman		01062		
Shanna Siegel		10579		

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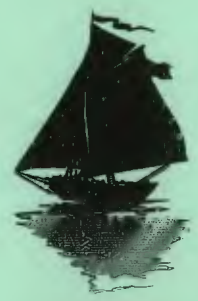

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Jay Lustgarten	Jay Lustgarten	02891	[redacted]	
Robert Reardon	Robert Reardon	10520		
Helen Bowers	Helen Bowers	10541		
Haley Cubell	Haley Cubell	17543		
Shelley Frost	Shelley Frost	03261		
Hannah Leonard	Hannah Leonard	03870		
Sarah Sieger	Sarah Sieger	10560		
Bob Schulof	Bob Schulof	11201		
Ellen Monk	Ellen Monk	10517		
Madeleine Blackburn	Madeleine Blackburn	07042		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Kelly Kohnert		07456	[redacted]	
Gus Myhrberg		12804		
Zoe Bracken		10028		
Anne Hope		10014		
Diane Mallach		07040		
Michelle Gore		12198		
Daniel Marsh		12198		
Guy Velella		12458		
Alan F Gotthardt		18956		
Nancy Pyne		10463		

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PETITION CALLING FOR A NON-PROTECTIVE DETERMINATION REGARDING THE HUDSON RIVER PCB REMEDIATION

To: EPA Administrator Scott Pruitt and Members of Congress

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
John A Burghardt	John A. Burghardt	05525	[redacted]	[redacted]
Tony Marasco	Tony Marasco	12601		
John Winter	John Winter	10517		
Jane Acorn	Jane Acorn	11792		
Rachel Hermann	Rachel Hermann	10801		
Dean Gallea	Dean Gallea	10591		
Jeanne Kiracofe	Jeanne Kiracofe	93022		
Mary Marcia Tupper	Mary Marcia Tupper	4		
SUSAN KATZ	Susan Katz	10128		
Jeremy Kelly	Jeremy Kelly	12180		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
James Uhlis		12553	[redacted]	
Anthony Maresca		12601		
Karen Delaney		12203		
KEVIN YAMAGUCHI		07645		
Mark Heffernan		12180		
Carrie Will		12208		
MONICA STAATS		12180		
Ethan Duffany		12186		
Lauren Gilbert		10940		
Victor Melendez		10940		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Linda Freitag	Linda Freitag	11375	[redacted]	
John Wackman	John Wackman	12401		
Ethan Coplan	Ethan Coplan	07043		
ILONA VRBA	Ilona Vrba	10507		
JACK GACE	Jack Gace	11226		
Joseph Kaminsky	Joseph Kaminsky	08801		
Aaron Shanosky	Aaron Shanosky	10605		
EARL BROWN	Earl Brown	12603		
Ryan Stewart	Ryan Stewart	12603		
GARY FLAUM	Gary Flaum	10510		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Tara Heneghan	Tara Heneghan	10597	[redacted]	
David Fenner	David Fenner	12603		
AL GASSMAN		10604		
Emily Hamilton	Emily Hamilton	10566		
Kathi Borgmann	Kathi Borgmann	14850		
ECKA B. FIELDS		10706		
Susan Stillman	S. Stillman	10606		
CAROLYN HOFFMAN		10603		
IVELISSE KOOPAN		12508		
Will Denson, Jr.	Will Denson	11561		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Tomasyn Goode		06226	[redacted]	
Andrew Feron		10561		
Joshua Kagan		12508		
BROOKE EVANS		10957		
Brian Reid		12571		
Logan Reid		12571		
Peter Coffman		22824		
Arlin Roy		10570		
Debbie M. VanRyn		11209		
Monte Silberg		11576		

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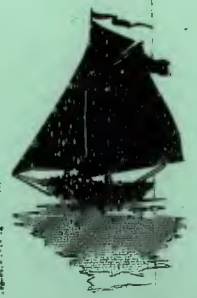

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Erik Ewald		20851	[redacted]	
Christina Vecchio		10954		
Nancy Gold		10536		
Meryl Kaviatkowska		10923		
RIKKI ABZUG		07434		
China Brady		07458		
Aurora Abzug		12504		
Walter Hughes		10526		
Brandy Tiesto		12582		
Catherine Moore		12528		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Olga Tverkhin		10707	[redacted]	
JoAnne Jones		10918		
Nora Reiser		10463		
Mary Kunicki		11375		
Katherine Daniel		06855		
Polly Armour		12525		
A Caudill		10562		
Priscilla Hlavac		10025		
Cynthia Regna		10516		
Sherry Moore		3220		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Steven Dolce	Steven Dolce	11703	[redacted]	
JAMES REQUA	James Requa	10516		
Milissa Amato	Milissa Amato	10591		
Milise Amato	Milise Amato	165?		
Kathy Lessuck	Kathy Lessuck	02906		
Benjamin Faber	Benjamin Faber	10520		
Elena B Blum	Elena B Blum	10025		
Sheri Neuroth	Sheri Neuroth	0702		
LINDA FRANCHELL	Linda Marie Franchell	14216		
MAXINE Falyar	Maxine Falyar	22640		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
JAI MEYERHOFF		14883	[redacted]	
Denise Schmidt		19475		
Deborah O'Neill		10548		
Nicolle Krauss		10562		
Judy D'Errico		10989		
Bob D'Errico		10989		
Stuart Stoffman		05346		
David Nagel		10980		
DAN COHEN		10960		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
MAKRAND BHOOT		10016	[redacted]	
Lila Severer		07878		
Katrina Severer		07878		
Victor Trujillo		87109		
Galadriel Severer		87110		
Alex Kaminsky		18064		
Liz Raskopf		02130		
Mariah Warren		12603		
Adrian Truini		10001		
Jonathan Meyer Jr		14883		

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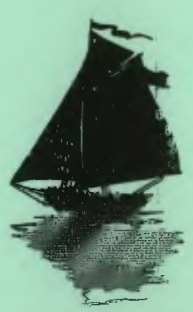

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
CLARE FRANCIS	Clare Francis	10706	[redacted]	
ERIC WALMAN		87461		
Lauren Hamm	Lauren Hamm	12569		
Maria Lorca		12603		
Barrett Roberts	Barrett Roberts	12601		
Isaac Flam		10510		
Andrew Ulrich	Andrew Ulrich	10954		
Lorraine Tapia		10954		
Carla Knauf	Carla M. Knauf	12601		
Tara Heneghan	Tara Heneghan	10597		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Marnie Vyfhuis	MK Vyfhuis	07046	[redacted]	
ORI ALON		12508		
MARY FLORIN-MCBRIDE		10520		
GUY CARANDENTE		10520		
Lisa Burton	Lisa Burton	07421		

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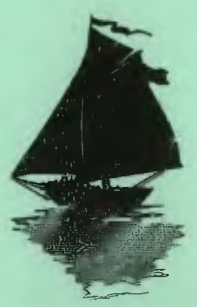

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Alex Peterson	Alex Peterson	02120	[redacted]	
Riverstone Company	[illegible]	05857		
Laura Hagang-Frye	[illegible]	02120		
Megan Joyce	Megan Joyce	02120		
Cat Virgo	[illegible]	12571		
Kathie Aspinall	[illegible]	12569		
Kenneth Siegel	[illegible]	11558		
T. Cichetti	[illegible]	10594		
SARAH WOMER	[illegible]	12504		
Barbara Cangiano	[illegible]	10550		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
S Murphy		14850	[redacted]	
A Bober		14850		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Norah Delaney	Norah D.	12308	[redacted]	
Elissa Pierse		12561		
Deirdre Delaney		12308		
Chrystal Jenkins		14450		
Justin Forlenza		11105		
Rebecca Forlenza		11105		
Amanda		12603		
Susan Cuat		05301		
Eric Carlson		11374		
Louis Grinsberg		11362		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Nora Gallardo	Nora Gallardo	12589	[redacted]	
Jill Benzer	Jill Benzer	10009		
Lyn Hardy	Lyn Hardy	15498		
T. Foley		0607		
Bill Wurtz		1963		
Anko Pletch		12561		
Michael Vega		12571		
Emily Boshi	Emily Boshi	10005		
George Bogrod		08904		
Jessica Epstein		07666		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Doreen Burden	Doreen Burden	10562	[redacted]	[redacted]
Daniel Berge	Daniel Berge	12561		
Eric Berger	Eric Berger	10463		
Arthur Lebowitz	Arthur Lebowitz	10960		
Allen Cubell	Allen Cubell	17543		
Nancy Bell	Nancy Bell	12480		
Lisa Reid	Lisa Reid	1257		
Maureen Crisman	Maureen Crisman	22824		
Meaghan A Crisman	Meaghan A Crisman	22824		
BRUCE CLAPPER	Bruce Clapper	01226		

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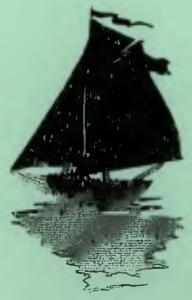

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Alison Hall	Alison Hall	07450	[redacted]	[redacted]
James O'Connor		10024		
Avi Roltzy		12601		
Katherina Kratter	Katherina Kratter	10025		
Vren; Rodgers	V. Rodgers	10580		
Thomas Connelly		11365		
James Connelly	James Connelly	11361		
Barry Clark		10552		
Kevin Hayden		12528		
Lyz Morabito		10570		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Kyle Schmidt		06831	[redacted]	
Alex Oliver		10520		
[illegible]		10520		
Sam Cohen-Angle		10520		
Elizabeth Faber		10520		
Madeline Rhum		11215		
Howard Wentle Rollis		12309		
Douglas DeFeo		11940		
Nicole Crook		11217		
[illegible] Cunha		10591		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Blaine Griffiths	Blaine Griffiths	10549	[redacted]	
Chris DeAngelis	Chris DeAngelis	10543		
Sandra Schmucki	Sandra Schmucki	01520		
Donna Stein	Donna Stein	10019		
George Klein	George Klein	10562		
T. M. NAGAI	T. M. Nagai	10701		
Kim Izzarelli	Kim Izzarelli	10510		
J. Murdoch	J. Murdoch			
Dee Brindis	Dee Brindis	10009		
Merry Brown	Merry Brown	1252[illegible]		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Katherine Anderson	Katherine Anderson	10522	[redacted]	
DALE BLUMENTHAL		10538		
MICHAEL REILLY		10567		
CAROLYN REILLY		10567		
Mary G Bagley	Mary G Bagley	10956		
Amy White	Amy White	11213		
KORONA		10939		
Tamara Bradley	Tamara Bradley	10009		
Mike Bultener	Mike Bultener	14886		
Barbara Bultener	Barbara Bultener	14581		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
PARKER MACDONALD		14850		
Bryn hildur Stefansdottir		14850		
WILLIAM SACCARDI		08889		
Matthew Bissell		11232		
Melissa Tubo		10512		
Morgan Beach		06734		
Tom Fisher		10509		
Wendy Cobb		12590		
Art Lowenstein		10524		
ANN PATTON		10524		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Peter Rothenberg		11203	[redacted]	
Dan Straks	Dan Straks			
Francine Nelson	Francine Nelson	07040		
ROBERT KAMMER		07950		
Marlene Von-Schaumer		1280 8		
Dillon Reilly		10567		
Charles Mahler		14617		
Pete Brechet		19061		
Emily Goldman		10706		
Kim Cleary		02120		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
MATTHEW FAHR				
Tan Lyons		12564	[redacted]	
Linda Hall		12721		
Jeff Hall		12721		
Lou Benson		10510		
LORRAINE PRICE		10594		
Carole Benson		07631		
[illegible]		10594		
Neil Benson		10591		
VIRGINIA MOSS		10011		

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Henry Neal 6/18/2017

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
T. Henry Neale Jr	T Henry Neale Jr	10583	[redacted]	
Carol Sue Neale	Carol Sue Neale	10583		
Donald A. Moore	Donald A. Moore	12534		
[illegible]	[illegible]	12567		
Audrey Enders	Audrey Enders	12526		
Gina Moore	Gina Moore	12534		
Lesley Koegel	Lesley Koegel	12567		
John Koegel		"		
Emily Brighia	Emily Brighia	11238		
Mikaila Arthur	Mikaila Arthur	02906		

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PETITION CALLING FOR A NON-PROTECTIVE DETERMINATION REGARDING THE HUDSON RIVER PCB REMEDIATION

To: EPA Administrator Scott Pruitt and Members of Congress

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Paul C Bermanzohn		12472		
Sally A Bermanzohn		12472	—	—
Barry Almazer		10514		
Janet Weinstein		10530		
Robert P Hansen		11780		
Judy Cooper		11795		
Maria Stavel		11205		
Myrda Frank		11516		
John D'Ingillo		11516		
Pierre Peters		19138		

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6/17/2017

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Justin White		10549	[redacted]	
S. OSBo				
Dwight Arthur		10541		
A Peterson		10457		
Bill Meyer		10549		
Daria Gregg		10562		
Tara Topping		10954		
Roxanne [illegible]		10954		
Matthew Wohl	Matthew Wohl	07110		
Charles [illegible]		01757		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
DELIA MARX		10607	[redacted]	
Meira Marom		11222		
[illegible]		06901		
Donald Gardner		10706		
Roger Brannan		12553		
Monika Granholm		10706		
Daryl Wise		12498		
James Murphy	James E Murphy	14850		
Emily Bingham	Emily Anne Bingham	02825		
Sarah Hadbeck		11209		

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Henry Neale 6/18/2017

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Joseph Cabibbo	Joseph Cabibbo	12477	[redacted]	
Ruby Cabibbo	Ruby Cabibbo	12477		
VLVERT MOORE	[illegible]	1257		
Sharon Zurick	[illegible]	06891		
Sherry Reeves	[illegible]	10520		
Cliff Elkind	[illegible]	10034		
Phoebe Gennado	[illegible]	01672		
Carolyn Zucker	[illegible]	01672		
Robert Fuhrich	[illegible]	10543		
Lacy Verhave	[illegible]	12570		

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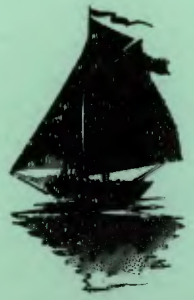

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Julia Colon		190	[redacted]	
Betty Garthwait		12401		
Debra Given		10025		
Lise Brown		10025		
Miki Weiss		10552		
Rachel Corke		10562		
Robert Corke		10562		
M. Hardy	Martin Hardy	37570		
Donna Chapman		12569		
Richard Miller		12561		

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Hany Nada 6/18/2017

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Nadine Boyce	Nadine Boyce	11205	[redacted]	
Kelly D'Arcy	Kelly D'Arcy	07461		
Debby Davis	Debby Davis	07461		
Carol Ann Rivadeneyra	Carol Ann Rivadeneyra	11363		
Stephen Baer	Stephen Baer	10707		
Christine Nota	Christine Nota	10598		
Julie Baez	Julie Baez	11427		
Jim Byrons	Jim Byrons	12561		
Dorian Burden	Dorian Burden	10562		
Jane Schwartz	Jane Schwartz	98029		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Sharon Steel		11385		
Ruth Greene		10805		
Paul Greene		10805		
David Goodwin		10003		
Joette Kane		12533		
Susanne Lowen		20912		
Layla Huckabey		29464		
Mark Trambley		03049		
Karen Streisfeld		10520		
Kirsten Seberg		10604		

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Amy Nack 6/18/2017

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Jonathan Bryant		3210	[redacted]	
Timothy Helgers		12401		
Anna Rubeo		12487		
Vera Rubeo		12487		
Hudson Lovell		10516		
W. MATTHEWS	(Wendy Matthews	06801		
Grace Doyle		12508		
DONNA BARKMAN		10562		
Peter Stott		[illegible]		
Sharon Alley		12211		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Bobbi Siegelbaum		10463		
Ellyn Rem	Ellyn Rem	06877		
Ilsa Harlewood		06880		
Isabelle Harlewood		06880		
Terry Kan		0730-		
Lucy Carney		10458		
Molly Carney		10458		
Thea Dent		10591		
JUDITH LIPSTEIN		10901		
Becca Lipstein		10901		

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Henry Neale 6/18/2017

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Sue Gamache	Sue Gamache	12582	[redacted]	

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Hannah Gretto		02125		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Jeanne Starke	[illegible]	101 28		
Rudy Kopecky	[illegible]	10562	[redacted]	
Elizabeth O'Donnell	[illegible]	01222		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
John Cronin		10516	[redacted]	
Bob Stiller		10960		
Alex Wolf		12572		
Ian Debruler		12561		
David Coplin		07043		
Danielle Fontaine		12498		
Samara Horwitz		12779		
Lucille Tracy		1070[illegible]		

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PETITION CALLING FOR A NON-PROTECTIVE DETERMINATION REGARDING THE HUDSON RIVER PCB REMEDIATION

To: EPA Administrator Scott Pruitt and Members of Congress

Although better than no clean up, General Electric's removal of toxic PCBs from the upper Hudson River doesn't go far enough to provide New Yorkers with the safe and usable river they deserve. The Environmental Protection Agency's 2005 Consent Decree allowed General Electric to leave what two federal agencies (NOAA and U.S. Fish and Wildlife) have described as "a series of Superfund-caliber sites" in the river that will prevent its full restoration. More needs to be done, not only to restore the river, but also to ensure economic opportunities that would result from lifting long-standing fishing restrictions, reopening the Champlain Canal to major commercial traffic, and a resurgence of tourism and recreation — all currently hindered by GE's PCBs. Last week, EPA released for comment a misguided decision that the 2009 – 2015 PCB remediation was working as designed in 40 miles of the Hudson above the Troy dam, although "not yet protective" of human health and the environment. However, three times as much PCB-containing sediment is left in the river than originally projected. EPA is willing to speculate and leave it to "Mother Nature" to take care of the remaining PCBs discharged by GE into the Hudson, closing down a multi-million dollar fishing industry. EPA and GE must finish the job -- and not leave New Yorkers holding the bag. EPA should require GE to complete a more robust cleanup to ensure the Hudson River recovers and that PCB levels in fish drop to levels safe for human consumption as soon as possible. EPA and GE have a moral and legal responsibility to make sure the Hudson River is safe, to restore it to its former health and usability, and to ensure GE compensates New Yorkers for the decades of damage it has caused.

That's why we, the undersigned, urge EPA to issue a determination of non-protectiveness, which would require a more robust cleanup of Hudson River PCBs.

NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Carlos Vazquez		11377	[redacted]	
A. Berogenfeld	A Berogenfeld	11375		
Fred Mayer	Fred Mayer	07717		
Susan Mayer	Susan Mayer	07717		

Please return completed petition to Hudson River Sloop Clearwater, 724 Wolcott Ave., Beacon, NY 12508

Wed Aug 30 12:05:31 EDT 2017
Gaines.Cynthia@epamail.epa.gov
FW: Delivering information to EPA for Administrator Pruitt and setting up a follow up call.
To: CMS.OEX@epamail.epa.gov

From: Matthews, Demond
Sent: Monday, August 28, 2017 1:52 PM
To: Hope, Brian <Hope.Brian@epa.gov>; Gaines, Cynthia <Gaines.Cynthia@epa.gov>
Cc: Bowles, Jack <Bowles.Jack@epa.gov>
Subject: FW: Delivering information to EPA for Administrator Pruitt and setting up a follow up call.

Good Afternoon All,

Here are some additional petitions I “just received” around lunch time from Hudson River Sloop Clearwater via email. These signatures are from Vermont per the organization. I wanted to forward them to ensure they are added to the ones I presented about a week ago.

If you have any additional questions, please let me know.

Thanks

V/r,

DEMOND L. MATTHEWS

Presidential Management Fellow

Small Communities Intergovernmental Liaison

EPA Office of Congressional & Intergovernmental Relations

1200 Pennsylvania Ave NW

Mail Code 1301-A

Washington, DC 20004

Phone: 202-564-3781 Cell: 202-738-3201

From: Stuart Strothman [mailto:sstrothman@gmail.com]
Sent: Monday, August 28, 2017 12:13 PM
To: Matthews, Demond <matthews.demond@epa.gov>
Cc: mannajo@clearwater.org; Thomas, Katie (Sanders) <Katie_Thomas@sanders.senate.gov>
Subject: Re: Delivering information to EPA for Administrator Pruitt and setting up a follow up call.

Dear Mr. Matthews,

Attached are more signatures regarding the EPA's direction on environmental regulation--one of the same petitions you've already received, but new signatures, primarily from Vermont.

Thanks very much.

Kind regards,

Stuart Strothman

On Fri, Aug 18, 2017 at 3:04 AM, Manna Jo Greene <mannajo@clearwater.org> wrote:

https://drive.google.com/drive/folders/0B0prHFXTGX-mRDBLS2k1YmNYUWc?usp=sharing

On Thu, Aug 17, 2017 at 3:03 PM, Manna Jo Greene <mannajo@clearwater.org> wrote:

Demond,

Many thanks for agreeing to meet and receive the municipal resolutions and petitions regarding Clean Water and related topics.

Here is the link to the scanned versions, as requested.

https://drive.google.com/drive/folders/0B0prHFXTGX-mRDBLS2k1YmNYUWc?usp=sharing

We would like to meet at 11:30 a.m.this morning, if that works for you.

I will call when we are nearby.

Many thanks,

Manna

On Thu, Aug 17, 2017 at 10:33 AM, Matthews, Demond <matthews.demond@epa.gov> wrote:

Good Morning Legislator Greene,

I would be more than happy to take your resolutions and petitions. Just give me at a minimum an hour notice prior to your arrival. Also, I will find out which office I should give them to.

Keep in touch.

V/r,

DEMOND L. MATTHEWS

Presidential Management Fellow

Small Communities Intergovernmental Liaison

EPA Office of Congressional & Intergovernmental Relations

1200 Pennsylvania Ave NW

Mail Code 1301-A

Washington, DC 20004

Phone: 202-564-3781 Cell: 202-738-3201

From: Manna Jo Greene [mailto:mannajo@clearwater.org]
Sent: Wednesday, August 16, 2017 10:19 PM
To: Matthews, Demond <matthews.demond@epa.gov>
Cc: Stuart Strothman <sstrothman@gmail.com>; Eargle, Frances <Eargle.Frances@epa.gov>
Subject: Delivering information to EPA for Administrator Pruitt and setting up a follow up call.

Demond,

Sending greetings. I spoke with Fran Eargle and am wondering if I could meet briefly with you on Friday morning to deliver 30 municipal resolutions and a set of petitions with 6,000+ signatures we collected at Clearwater's Great Hudson River Revival festival June 17-18?

Perhaps we could arrange a follow up call after the 4th of July holiday.

I have limited email access while here in DC, but will be with Stuart Strothman, who has, so I am cc'ing Stuart as well.

I can be reached by phone at 845-807-1270.

Many thanks for your help with this.

Manna

--

Clearwater is sailing to Washington, DC, leaving from Revival on August 17 to bring a our cargo of concern and strong message of support for environmental protection, especially the gains we have made since the Clean Water Act was passed in 1972. Please donate to support our Sail to DC at
https://interland3.donorperfect.net/weblink/weblink.aspx?name=clearwater&id=12
All our waters are connected. All our waters must be protected.

Many thanks,

Manna

Manna Jo Greene, Environmental Director
Hudson River Sloop Clearwater, Inc.
724 Wolcott Ave., Beacon, NY 12508
845-265-8080 x 7113 Fax: 845-831-2821
845-807-1270 (cell)
845-687-9253 (home office)
www.clearwater.org

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Clearwater's Sail on Washington

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Petition In Support of Environmental Protection and Clean Water

TO: SCOTT PRUITT, ADMINISTRATOR, ENVIRONMENTAL PROTECTION AGENCY; cc: CONGRESS

We, the undersigned, support the Environmental Protection Agency (EPA) and strong federal environmental regulations that protect pub health and safety, and ensure a sustainable future for generations to come. Americans want clean air and water for themselves and their children. Because pollution doesn't stop at state borders, the federal government has an essential role to protect these vital resources and must not abdicate it responsibility to do so. In 1970 Clearwater sailed to Washington DC in support of the Clean Water Act, which was passed in 1972. Now, on its 45th anniversary, we face new challenges – including the pollution of drinking water in Flint, Michigan and in Hoosick Falls and the City of Newburgh, New York, among others. We also urgently need action to address climate change. We strongly support the EPA and its accomplishments – and look to the federal government continuing to protect our waterways, which are essential to the health, well-being and economic revitalization of our communities and watersheds.

NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Kathi Tighe	K. Tighe	02139		[redacted]
Steve Mindel	Steve Mindel	05301		
Amy Hathaway	Amy Hathaway	05401		
Lynn Arnold	Lynn Arnold	03455		
Marcia L Nelson	Marcia L Nelson	05301		
William Cogburn	William Cogburn	05701		[redacted]
Mitch Diamond	Mitch Diamond	02155		

Please return completed petition to Hudson River Sloop Clearwater, 724 Wolcott Ave., Beacon, NY 12508

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Eric Breck		02140	[redacted]	
Emily Breck		02140		
Jeff Bary		11201		
Barbara Moloney		08628		
Sarah Mason		03037		
JOHN C. CARPENTER		11215		
Ginny Briggs		01752		
Ava Grey		01452		
Miranda Johnston		02474		
Maeve Tyler-Penny		02446		
Kira Young		02460		
Henry Manlick		01742		
Valerie Fathergill		01742		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Aaron Huyden		01002	[redacted]	
BRUCE ORDEN		13760		
Susan Wallach		10516		
Sarah Heron		10001		
Ellen Cookson		11218		
Margaret Barry		11201		
Deirdre Biolo-Padin		11215		
Joan Scim		10570		
Amelia Mason		02144		
Anna Blackman		10009		
Amanda Barbour		08852		
Aidin Carey		02144		
Lisa Keegan		02143		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Morgan Ingalls		05301	[redacted]	
ANDREA MATTHEWS	Andrea Matthews	0534[illegible]		
Emily Lin	Emily Lin	05345		
Lindsey Weaver		05301		
N. Arielle Colm		05101		
Sophie Baby-Kyle		05301		
Guy Wood	Guy Wood	05301		
Margaret Barrand	Margaret Dale Barrand	05301		
Keith Murphy		05301		
Lauren Breining		05301		
Patrice Murray	Patrice Murray	05301		
Robin Y Davis	Robin Y Davis	05301		
Emma Davis	Emma Davis	05301		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Adrienne Major		05346		
Whitney Field		03466		
Rich Holschuh		05301		
Saralinda Lobrose		01070		
Andrea K O Rogers	Andrea K O Rogers	01072		
	Elizabeth M. Smith	01349		
DAVID G REES		01072		
Janice P. Mason	Janice P. Mason	01093		
Natasha Diamondstone	Natasha Stone	05301		
Malena Ordnung		07042		
	Michael Kohout	05301		
TED HABER	Ted Haber	01354		
Owen Diamondstone-Kohout	Owen	05301		

[redacted]

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Lucia Morey		05301	[redacted]	
Audrey maples		05346		
Cayden Leavey		05301		
Rhys Glennon		05301		
Eliza Price		05301		
Lucy Congleton		05301		
Bill Congleton		05301		
Robert Glennon		05301		
Edward Dodd		[illegible]		
Kerry Bourne		05345		
Aubrey Bourne		05345		
Barbara Bourne		" "		
Abraham Noe-Hays		05346		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Bethany Morrow-Crabbs		05346	[redacted]	
Amy Cann		05346		
Randy Major	RANDOLPH MAJOR	05341		
Ann Coakley		05346		
Stefan Amidon		05301		
Amanda Whitman		05301		
Peter Amidon		05301		
Rebecca L Tracy		05301		
Laurie Courson		05346		
Peg Alden		05346		
Ellen Kaye		05346		
David Eggleton		01801		
Jake Hamelin		05341		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Timothy Breer	Timothy J Breer	05158	[redacted]	
MARY LOU MASSUCCO	Mary Lou Massucco	05154		
Dayna Roebuck	Dayna Roebuck	05301		
Catherine Morris	Catherine Morris	05361		
Alden Witman	Alden Witman	05301		
Arthur Davis	Arthur Davis	05346		
Peter Merritt	Peter Merritt	05301		
John Bruce	John Bruce	05744		
Rafe Rosen	Rafe Rosen	05301		
Gil Rosenberg	Gil Rosenberg	05301		
Todd Blackman	Todd Blackman	05301		
Geoff Rogers	Geoff Rogers	01077		
Sam Green	Sam Green	05301		

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POLITICAL ACTION AT WORK? YES! (STUART IS COLLECTING SIGNATURES + WILL GO TO DC IN JUNE TO DELIVER THESE TO CONGRESS, ALONG WITH THE CREW + HUDSON RIVER SLOOP CLEARWATER). SIGN IF YOU WILL! THANKS.

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Stuart Strothman		05301	[redacted]	
DANA AQUADRO		05346		
Robert Oakes		05346		
CHRISTY JENNETT		0556		
Cliff DesMarais		05340		
Dania Cloagh		05101		
Christopher Brewer		05301		
Erica Bitaoui		05149		
Susan Rugg		05154		
Alexis Esposito		05146		
Gwen Bear		01301		
Solianna Bear		01301		
Susanna Strothman		05301		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
STORM		01301		[redacted]
D Rosen		05141		
Helene Lene		01096		
Amy Chapman		01027		
Mark Lena		01096		
Victoria Rosen		0544[illegible]		
Remy Loeb		01070		
Debbie Potter		03303		
Helen Debra		05602		
Alexandra Zuser		03450		
Tom PUTNICK		03457		
Valerie VanMeter		03457		
Heidi Eide		01379		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Emma Schneider	Emma Schneider	02144	[redacted]	
JOHN SMITH	John D Smith	05346		
Elena Dodd	Elena Dodd	05346		
Jackson Levy-Lord	[illegible]	05301		
Andrew Davis	Andrew M Davis	05301		
[illegible]	[illegible]	05301		
Susan Wyse	Susan Wyse	05346		
Sandra McKee	Sandra McKee	05672		
Virginia S Bunker	Virginia S Bunker	05346		
Jillian Hunsdon	[illegible]	05301		
Steve Mindel	Steve Mindel	05301		
Rachel Diamondstone	Rachel Diamondstone	01364		
Louisa Strothmann	[illegible]	11207		

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NAME (Please Print)	SIGNATURE	ZIP	PHONE	EMAIL
Bari Shamas	Bari Shamas	05346		
Harvey Liss		05345		
Ellen Nuffer		05345		
John Durham		05344		
Andy Ingalls		05301		
Suzanne d'Corsy	Suzanne d'Corsy	05345		
Cynthia Jacelon	Cynthia Jacelon	01301		
John E Ridgway	John VE Ridgway	01301		
Heather Harrison	Heather Harrison	05301		
John Wilmerding	John Wilmerding	05301		
Alan McArdle		01039		
Nadia Fox	Nadia Fox	01040		
HEATHER TAYLOR		05346		

Please return completed petition to Hudson River Sloop Clearwater, 724 Wolcott Ave., Beacon, NY 12508

BONURA
HOSPITALITY GROUP

August, 2017

Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski:

We represent businesses along the 200-mile span of the Hudson River Superfund site. The river is the bedrock of the Hudson Valley's current and future economic vitality. It drives the region's multibillion-dollar tourism industry and is in large part responsible for the ongoing recovery of the real estate market in the Lower Hudson since the great recession. The beauty of the river and the myriad parks along it contribute significantly to residents' quality of life and serve as catalysts for attracting visitors and new jobs.

Building upon this momentum depends on a clean, healthy Hudson River. As long as unacceptable levels of PCBs pollute its water, sediment and fish, they hinder lasting economic gains—both the resumption of once-lucrative industries dependent on the river and long-stalled development opportunities along it. More important, they continue to pose a threat to the health of people living in riverfront communities.

For 70 years, the economic, recreational, cultural and scenic values of the Hudson River have been compromised by PCB contamination. This pollution has destroyed a once-vibrant commercial fishing industry, hampered the operation of marinas, led to a severe curtailment of marine transport on the Champlain Canal, tripled the costs of dredging the NY-NJ Harbor, prevented ambitious economic development opportunities on the Upper Hudson similar to those being realized along the Mohawk River, and barred generations of residents and visitors from full enjoyment of this American Heritage River.

For these reasons, we call on the EPA to:

Declare in its Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as the EPA's draft review explicitly states.

Delete the draft review's finding that the remediation "will be protective" in 53 years. The EPA makes this forecast despite admitting eight additional years of research are needed to verify it. Further, data indicate that fish toxicity in the Upper Hudson is almost 300 percent higher than the goal the EPA expected to reach in 2018. If this interim target is so off base, how can the EPA forecast with any reliability that the cleanup "will be protective" in five decades?

Conduct additional cleanup of the Upper Hudson. The draft review fails to incorporate any analysis by the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation showing that the remediation leaves behind contamination equivalent to (in NOAA's words) "a series of Superfund-caliber sites." Both NOAA and the DEC have concluded that additional dredging is needed. An "is not protective" determination will pave the way for this to happen.

Undertake a remedial investigation of the Lower Hudson. The draft review makes clear that PCB levels in fish and sediment in the 160-mile portion of the Lower Hudson have not benefited at all from upriver dredging. In actuality, downriver contamination is significantly higher than expected. The draft review lays out no plan for investigating and removing this contamination. This oversight must be corrected.

BONURA
HOSPITALITY GROUP

Data confirm that time and nature won't fix this project's shortcomings, as your draft review would lead us to believe. Only additional dredging will make the Hudson healthy as soon as possible. Therefore, we strongly urge the EPA to conclude that the remedy for the entire Hudson River Superfund site is "not protective." Then and only then can we begin to plan for the bright future our children and grandchildren deserve.

Sincerely,

Joseph A Bonura Jr

Owner

176 Rinaldi Boulevard, Poughkeepsie, NY 845.486.4700

BONURA
HOSPITALITY GROUP

176 Rinaldi Boulevard
Poughkeepsie, NY 12601

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Gary Klawinski
Director, Hudson River Field Office
US Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

12205-113878

Hudson River Public Comment - NASA Backed PCB remediation technology

Ian Doromal <ian@ecospears.com>

Fri 9/1/2017 9:54 PM

To: epahrfo@outlook.com <epahrfo@outlook.com>;

Dear Gary Klawinski,

My name is Ian Doromal, Vice President of ecoSPEARS. We are an environmental solutions provider of patented, non-invasive NASA SPEARS (sorbent polymer extraction and remediation system) to extract and remediate PCBs from sediments, safely and effectively.

For our communities, marine ecosystem and marine life at stake, and for those families living near the Hudson River and affected throughout the years, we must continue the fight to clean up the Hudson. THE JOB IS NOT FINISHED. In fact, dredging has DONE NOTHING to fix the PCB issue. All dredging has done is resuspend the PCBs downstream. The Hudson River is as toxic as ever!

I am writing to you and to all of EPA that we have an Enhanced Monitored Natural Recovery (EMNR) technology solution to extract and remediate PCBs in a non-invasive and cost-effective way. This NASA-licensed technology targets and remediates PCBs at the source.

With two pilot demonstrations sponsored in part by Golder Associates, our technology has shown a 75-90% success rate in totally removing PCBs from contaminated sediment over the course of 3-6 months, with higher rates of success expected in areas of PCB concentration higher than three (3) parts-per-million and/or three (3) months. Our technology has also shown success in removing PAHs, and our scientists are currently testing solutions which successfully remove PCBs from dry soil and landfills.

In light of the EPA's outreach to the Hudson River community for comments on its cleanup initiatives and proposal for the cleanup, I wish to bring our technology to your attention as a potential alternative.

The SPEARS technology is a 6-8-inch hollow spike, made from recycled polymer plastic, lined with resin for structural integrity and filled with an ethanol-based solution. The addition of the ethanol solution into the hollow interior allows the molecules of the polymer to expand, which in turn allows hydrophobic PCBs to enter the polymer and become trapped in the ethanol. A series of SPEARS are anchored to mats allowing for easy implementation and removal.

Budget cuts to the EPA and state-sponsored cleanups threaten to allow the toxicity of Superfund sites to further spread into our water, land, and food.

Our mission at ecoSPEARS is to introduce a solution for PCB remediation which not only meets the EPA's evaluation criteria, but removes toxic PCBs from the environment while protecting the surrounding ecosystem by providing a non-invasive and true solution to reducing and removing toxicity levels, mobility, and overall volume of PCBs. ecoSPEARS seeks to provide the environmental remediation industry with a sustainable, long-lasting solution to traditional remediation methods.

As the initiatives continue on the Hudson River Superfund site cleanup, it is my sincere hope that you will consider the innovative and sustainable technology solutions ecoSPEARS provides for immediate, safe, and long-lasting PCB remediation.

All of our information, including published reports on our pilot demonstration studies with Golder Associates, can be found online. If you wish to learn more about who we are, a link to our website will be provided in this submission. Thank you.

EPA REMEDIATION GUIDELINES:

ecoSPEARS - *how do we fit?*

1. ecoSPEARS is non-invasive: it protects the delicate ecosystem in/around the remediation site without disturbing the natural process of wildlife.

2. ***LOOK INTO FEDERAL/STATE ENVIRONMENTAL LAWS re: REMEDIATION***

3. ecoSPEARS absorbs PCBs and PAHs at the source, removing them for good and providing long-term relief to the environment from these toxic chemicals.

4. ecoSPEARS is the **ONLY** remediation solution that actively reduces the levels of toxic man-made chemicals in contaminated sediment and water.

5. ecoSPEARS begins working **IMMEDIATELY** and remains effective as a true solution for short-term remediation **AND** long-term projects.
6. ecoSPEARS are easily implemented in areas containing soft sediment or soil; in rockier, more flocculent areas, dredging will be required before SPEARS can be implemented.

7. ecoSPEARS cuts the cost of capping or dredging. Because the SPEARS begin working immediately upon implementation and continue to passively absorb PCBs as time goes on, the cost of SPEARS works cyclically and requires minimal manpower to implement. The costs of machinery, 24-hour work pay, and federal and/or state fines are slashed.

8. ***THIS WILL LIKELY VARY BY STATE - EXPECT POLITICAL AND/OR LEGAL OPPOSITION/LOBBYING AGAINST US***

9. ecoSPEARS provides communities with the world's only available non-invasive and **TRUE** solution to PCB remediation ***(EXPECT QUESTIONS FROM COMMUNITIES SIMILAR TO ONES RECEIVED AT RCVP COMPETITION)***

GAP ANALYSIS:

- Each and every proposed solution to the Hudson River cleanup project **DOES NOT** remove/reduce toxicity, mobility, or volume of PCBs: ecoSPEARS does, so how do we communicate that in layman's' terms?

- The chosen alternative… *"has less impact to habitat and surrounding properties than other options, protects against erosion and would help maintain flow in the river channel. It is less costly than alternatives A-6, A-7*

and A-8, protects human health and the environment, and provides short- and long-term effectiveness while complying with applicable or relevant and appropriate requirements, known as ARARs."

- It is our hope that we generate word of mouth in the Hudson River community to lead to a pilot study for implementing SPEARS at different hotspots across the Superfund site to prove that this technology is truly scalable.

Our website is: www.ecospears.com

Please feel free to reach out to me: 407-595-5785 or email me directly at ian@ecospears.com

Regards,

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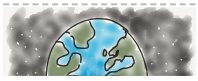

R. Ian Doromal | *Vice President*
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Winter Park, FL 32789
Tel: (407) 595-5785
ian@ecoSPEARS.com
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COMMENTS ON EPA's SECOND FIVE-YEAR REVIEW REPORT FOR HUDSON RIVER PCBs SUPERFUND SITE

Haggard, John (GE Corporate) <john.haggard@ge.com>

Fri 9/1/2017 4:53 PM

To: epahrfo@outlook.com <epahrfo@outlook.com>;

Cc: Gary Klawinski - USEPA (Klawinski.Gary@epamail.epa.gov) <Klawinski.Gary@epamail.epa.gov>; Gibson, Bob (GE Corporate) <bob.gibson@ge.com>;

2 attachments (5 MB)

2017-09-01 Cover Letter - GE 5YR Comments.pdf; 2017-09-01 GE 5YR Comments.pdf;

Please find attached the comments of the General Electric Company on the EPA Report: “Second Five-Year Review Report for the Hudson River PCBs Superfund Site” (May 31, 2017).

Thank-You

John G. Haggard

GE
Leader, Global Remediation
Global Operations, Environmental, Health & Safety
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September 1, 2017

Gary J. Klawinski
Director, Hudson River Office
U.S. Environmental Protection Agency, Region 2
187 Wolf Road, Suite 303
Albany, New York 12205

Re: Hudson River PCBs Superfund Site
GE's Comments on Proposed Second Five-Year Review Report

Dear Mr. Klawinski:

Enclosed are the comments of the General Electric Co. on the Proposed Second Five-Year Review (Second FYR) Report for the Hudson River sediment remedy.

The Hudson River dredging remedy remains one of the largest and most logistically complex environmental cleanups in U.S. history. Together, GE and EPA removed significantly more PCBs than projected, while also mitigating potentially adverse impacts to the fullest extent practicable. That is why, at the completion of the dredging in 2015, EPA aptly described the project as an "historic achievement" and declared the project to be "extremely successful." EPA's Second FYR Report reaffirms those conclusions and that the Hudson River dredging project is on course to achieve EPA's goals of protecting human health and the environment. GE is proud to have completed this unprecedented project that EPA selected, New York State endorsed, and both oversaw, and GE is proud of the environmental improvements that have been achieved so far, the result of a very productive working relationship.

The data collected to date, and summarized in the Second FYR Report, demonstrates that the remedy—chosen by EPA with the concurrence of New York State—is reducing PCB levels as planned and shows why no additional dredging in the Upper or Lower Hudson is recommended or warranted. In the first 12 months since GE completed the $1.7 billion dredging project, PCB levels in water in the Upper Hudson declined at every monitoring station and by as much as 73 percent. PCB levels in fish are near or below pre-dredging levels, as EPA projected. These results indicate that environmental conditions have responded to the remedy and are improving just as EPA projected they would.

The purpose of a Five-Year Review, as EPA's guidance explains, is to assess whether the selected remedy is, or will be, protective of human health and the environment by evaluating whether it is functioning as intended and whether the assumptions underlying that remedy remain valid. Consistent with that guidance, EPA reviewed all of the data collected since 2002 and determined in the Second FYR that the remedy was implemented correctly and is functioning as intended. That determination is consistent with the expectations EPA set forth in its 2002 Record of Decision (ROD) and with its First Five-Year Review in 2012, in which the Agency concluded that the remedy would be protective of human health and the environment based on data and information at that time.

On the sole issue now before EPA—whether the remedy was properly implemented and working as intended—there can be no real dispute. The enclosed comments demonstrate the many ways in which the project's substantial technical record fully supports EPA's analysis and determinations. As we show:

- The selection of the remedy followed 12 years of study and advice from scientists, environmental groups, elected officials and local community representatives. New York State participated fully in EPA's deliberative process, supported and concurred with the remedy when it was chosen, and helped oversee its implementation. In

formally concurring with the final remedy decision in 2002, New York State found "the selected remedy to be protective of human health and the environment" and "will reduce public health and environmental risk," and lauded EPA's approach that "balances the public health and ecological needs . . . with the concerns expressed by the many stakeholders, including local communities." The Hudson River's leading environmental groups also celebrated the remedy as a "victory for the Hudson River cleanup" and "a very important turning point for the Hudson River." Before selecting the final remedy, EPA considered multiple alternatives, ranging from monitored natural recovery (no dredging) to full bank-to-bank dredging. Ultimately, EPA chose a balanced remedy grounded in a careful, quantitative analysis and tailored to the specific conditions of the site. EPA's goal was the strategic removal of enough PCBs to accelerate the decline in PCB levels in fish, while minimizing damage to the river ecology and disruption to local communities. While more extensive and costly remedies were considered, EPA found none would have achieved significantly better results. The data collected to date confirm this conclusion.

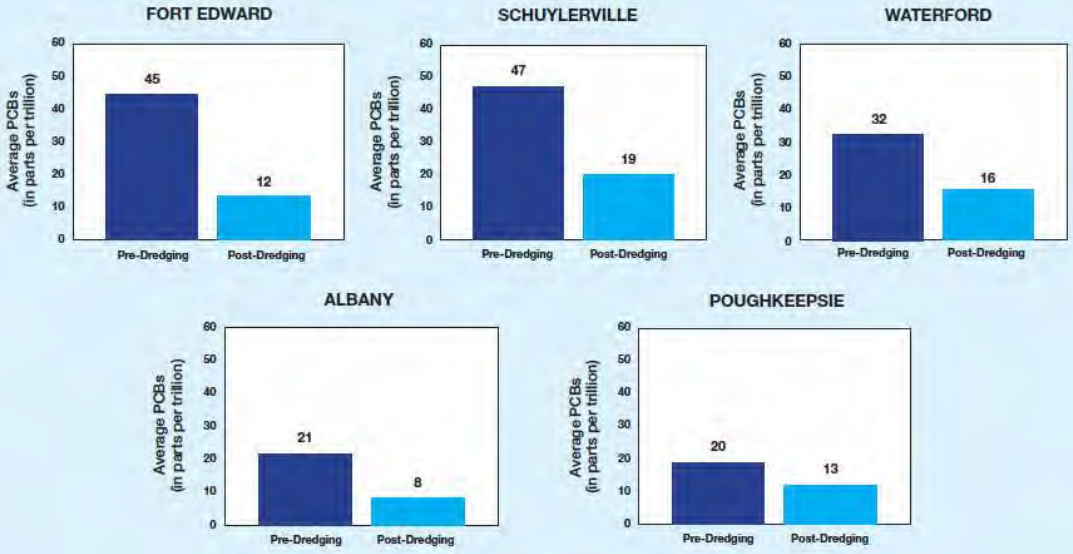
- The final dredging project, as executed, removed more than twice the mass of PCBs as expected in the Record of Decision (ROD)—nearly 150,000 kilograms (kg) versus 70,000 kg estimated in the ROD—and it removed a greater percentage of the PCB mass in the river than anticipated—nearly 80% versus 65% estimated in the ROD. The current estimated PCB mass remaining in non-dredged areas of the river is wholly in line with EPA's estimates in the ROD.

- Water and fish data collected from the river since the completion of dredging show a positive environmental response to the remedy that is consistent with EPA's expectations. EPA recognized in the ROD that, even with the most extensive dredging alternative it evaluated, it would take decades before unlimited human consumption would be feasible based on projected declines in PCB levels. In the interim, EPA concluded that human exposure to PCBs through consuming fish would be controlled, to the extent practicable, through New York State's fish consumption advisories and fishing restrictions. As EPA found in the Second FYR, those controls are functioning as expected. In the 110 miles of the Lower Hudson River from Catskill to New York Harbor, most fish are now considered safe for eating on a weekly or monthly basis for men over 15 and women over age 50, including the prized striped bass, according to the state's fish consumption advisories. While New York State bans consumption of fish from the 40 miles of the Upper Hudson, the River supports a thriving recreational (catch and release) fishery.

- Long-term monitoring after dredging is and always has been an integral part of this remedy. GE has begun this monitoring, and the results from the first year of post-dredging monitoring (2016) are encouraging and consistent with EPA's projections. For example, in the Upper Hudson River north of Albany, where the dredging occurred, PCB levels in water declined as much as 73% from pre-dredging levels. In the Lower Hudson River south of Albany, where PCB levels were already significantly lower prior to dredging, PCB levels declined as much as 36%. These declines are depicted on Figure 1. Post-dredging fish data from 2016 likewise indicate that fish are beginning to recover. Monitoring of water, fish, and sediments will continue for decades to verify that the dredging remedy and ongoing natural recovery will reduce PCB concentrations to the target levels in the expected time frames.

FIGURE 1:

Post-dredging PCB levels in water declining at expected rates



Information about PCB levels in water is important because PCB levels in water and sediment influence PCB levels in fish.

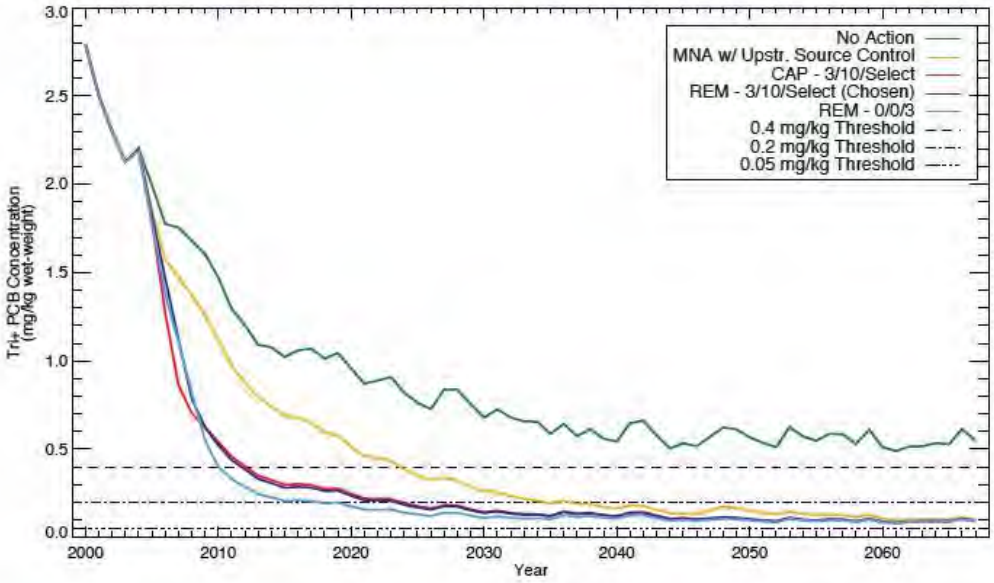
Water data collected from May through December when river flows at Thompson Island in the Upper Hudson River were equal to or below 5,000 cubic feet per second and at Albany during all river flows. Pre-dredging data collected in 2004-2008, and post-dredging data collected in 2016.

Notwithstanding the clear evidence supporting the ongoing success of the remedy, some parties have resurfaced old arguments and argued that EPA should order additional dredging, claiming that more dredging would more quickly achieve EPA's goals. This claim is not supported by scientific evidence; nor is it properly a question for the Second FYR. At its core, this argument improperly seeks to have EPA go back to the drawing board, reopen the remedy selection process and select a different remedy. The sole question before EPA at this stage is whether the selected remedy was properly implemented and is working as planned. EPA has properly concluded that the answer to this question is yes—the remedy was properly implemented and is working as planned, and the scientific data demonstrate that the dredging project is clearly delivering the environmental benefits that EPA envisioned.

Moreover, the calls for additional dredging fail to recognize that, in selecting the final remedy in 2002, and in multiple subsequent reviews, EPA considered the arguments being raised now and rejected them. EPA's analysis has consistently demonstrated that additional dredging beyond the selected remedy will not deliver better results in a significantly shorter time frame. In fact, it showed that even the most extensive removal alternative would not be significantly more protective than the chosen remedy and would not appreciably reduce the number of years to achieve the same target levels, as shown on Figure 2. As EPA recognized in its 2002 ROD Responsiveness Summary, more extensive removal alternatives would "greatly increase costs while yielding little additional public health or environmental benefit," and would therefore fail Superfund's critical requirement that every remedy selected must be cost-effective.

What EPA's Model Predicted in 2002:

FIGURE 2: COMPARING PCB LEVELS BY REMEDY



Upper Hudson River Species-Weighted Fish Fillet Average Tri+ PCB Concentration

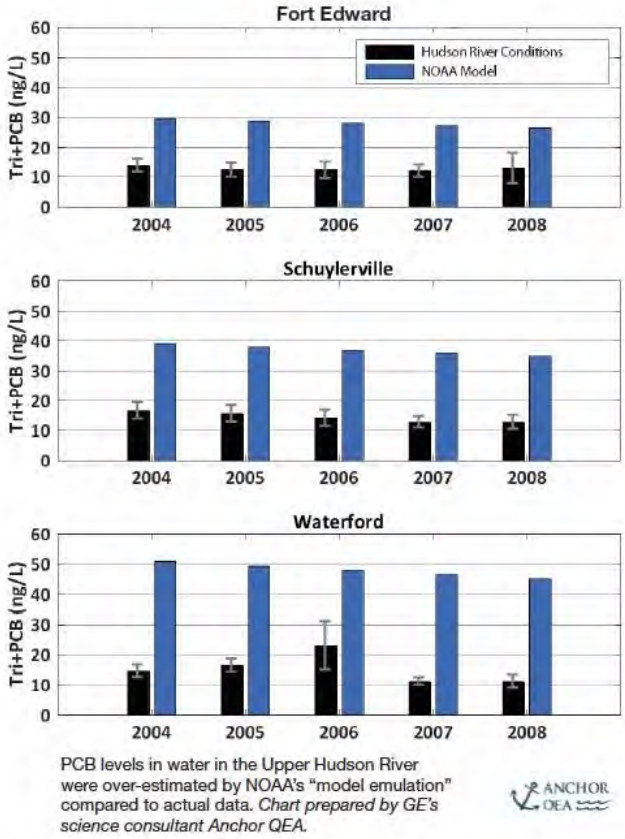
Data Source: Table 11-2 from the Hudson River PCBs Site Record of Decision.
UHR average obtained by weighting RS1, RS2, and RS3 species-weighted results by factors of 0.154, 0.125, and 0.721, respectively.

ANCHOR QEA

Data from EPA's Record of Decision (February 2002, Table 11-2) show the projected timeframes for reductions of PCB levels in fish, based on a range of remedial options from No Action to a major dredging project. All options reduce PCB levels in fish. EPA selected the REM-3/10/Select (dark blue) over more extensive dredging (REM-0/0/3 light blue). The two dredging options show only a modest difference in the levels of PCBs in fish over several years. Chart created by GE's science consultant Anchor QEA from EPA data.

Some advocates for more extensive dredging have relied on a model developed by the National Oceanic and Atmospheric Administration (NOAA), which purports to show that fish in the Lower Hudson River will recover at a slower rate than predicted in EPA's ROD. The NOAA model is demonstrably invalid for several reasons but chiefly because its results are inconsistent with actual, measured data—a critical test for determining a model's validity and reliability. Its predictions for both fish and water are significantly higher than the actual data. For example, as shown in Figure 3, the NOAA model over predicts PCB concentrations in water by two to four times compared to actual measured data.

FIGURE 3:
ACTUAL DATA VS. NOAA MODEL



PCB levels in water in the Upper Hudson River were over-estimated by NOAA's "model emulation" compared to actual data. *Chart prepared by GE's science consultant Anchor QEA.*

EPA has said that it expects the benefits of the remedy to become even clearer as additional data are collected over the next several years. An independent expert report prepared for the Hudson River Foundation concurs, recently concluding that many additional years of monitored natural recovery will be necessary to determine whether any additional remedial action is required.

The appropriate next step is the one the ROD envisions: the collection of scientific data to provide a sound basis for assessing the long-term performance of the remedy. GE will continue to work closely with EPA and New York State to complete this additional work. At this time, there is no justification whatsoever for additional dredging. The data demonstrate this remedy is working. As EPA has determined in its Second FYR, the remedy removed more PCBs than expected, is functioning as expected, and will protect human health and the environment. GE will continue to meet its commitments as it has during every stage of this process.

Please let us know if you have any questions about the enclosed comments.

Sincerely yours,

John G. Haggard
GE Project Coordinator

Enclosure

cc: Walter Mugdan, EPA
Douglas Garbarini, EPA
Douglas Fischer, EPA
Commissioner Basil Seggos, NYSDEC

COMMENTS OF GENERAL ELECTRIC COMPANY ON PROPOSED SECOND FIVE-YEAR REVIEW REPORT FOR HUDSON RIVER PCBs SUPERFUND SITE

September 1, 2017

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LIST OF ACRONYMS AND ABBREVIATIONS

ARAR	Applicable or Relevant and Appropriate Requirement
BMP	baseline monitoring program
CD	Consent Decree
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CU	Certification Unit
cy	cubic yards
DAD	Dredge Area Delineation
EPA	U.S. Environmental Protection Agency's
First FYR	*First Five-Year Review Report*
FS	Feasibility Study
g/m^2	grams per square meter
GE	General Electric Company
HRF	Hudson River Foundation
kg	kilogram
LC20	20% lethal concentration
LOAEL	Lowest Observed Adverse Effect Level
mg/kg	milligrams per kilogram
mg/kg-BW/day	milligram per kilogram body weight per day
MNA	monitored natural attenuation
MPA	mass per unit area
NCI	Nodal Capping Index
NCP	National Contingency Plan
ng/L	nanograms per liter
NOAA	National Oceanic and Atmospheric Administration

NOAEL	No Observed Adverse Effect Level
NRD	natural resource damage
NYSDEC	New York State Department of Environmental Conservation
NYSDOH	New York State Department of Health
OM&M	operation, maintenance, and monitoring
OU	operable unit
PCB	polychlorinated biphenyl
RAM QAPP	*Phase 2 Remedial Action Quality Assurance Project Plan*
RAO	Remedial Action Objective
RG	Remediation Goal
RG	Remediation Goal
ROD	Record of Decision
RS	River Section
Second FYR	*Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site*
SOW	Statement of Work
the Site	Hudson River PCBs Superfund Site
TIP	Thompson Island Pool
TPCB	Total PCBs
$TPCB_{HE}$	Total PCBs based on PCB homologue equivalents
Tri+ PCBs	PCBs with three or more chlorine atoms

EXECUTIVE SUMMARY

The General Electric Company (GE) is submitting these comments on the U.S. Environmental Protection Agency's (EPA's) *Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site* (Second FYR; EPA 2017). That report concludes that the remedy selected by EPA in the 2002 Record of Decision (ROD) for the Hudson River sediments (EPA 2002a), which GE implemented through dredging in 2009 through 2015, is functioning as anticipated and will be protective when the monitored natural attenuation (MNA) component of the remedy is complete, and that in the meantime institutional controls are in place to control human exposure pathways.

EPA provides compelling and detailed evidence to support these conclusions. The report is well organized and clearly written, and addresses all the necessary regulatory and statutory requirements. GE's present comments, after providing some important background information on the sediment remedy, demonstrate that: (a) the existing data and other information support EPA's determination that the remedy is functioning as expected; (b) the ROD's conclusion that the remedy is protective of human health and the environment remains valid at this time; (c) long-term monitoring will be necessary to determine the long-term protectiveness of the remedy; and (d) there is no basis for the additional dredging of the river that some have called for.

Selection and Implementation of Remedy

The ROD selected a remedy from several alternatives after 12 years of study and advice from scientists, environmental groups, elected officials and local community representatives. That remedy involved strategic dredging in the Upper Hudson River and MNA of the polychlorinated biphenyls (PCBs) remaining in the river. A larger removal alternative was rejected on the ground that it would not result in a significant incremental reduction in human health or ecological risks and yet would cost substantially more than the selected alternative.

The selected remedy was based on several expectations and conclusions that all parties understood:

- Recognizing the limitations of the then-existing sediment dataset, the ROD required a massive data collection effort and application of specific numerical removal criteria to define the dredge program based on those data. The removal criteria were constructed in such a way as to result in more or less PCB removal depending on what the actual data showed – in other words, allowing the dredging project to be scaled as dictated by the data.
- PCBs would be left behind in the river, but those PCBs would be either buried or at acceptable levels such that surface sediments would be expected to recover over time.
- Achievement of the ROD's Remediation Goals (RGs), established for fish and water, would take a number of years after the remedy was complete. Although the specific times presented in the ROD to reach target levels were presented for the purposes of comparing the remedial alternatives, not as absolute predictions, it was recognized that, under all alternatives, it would take a substantial period of time to reach levels that would allow unrestricted consumption of fish. For example, under the selected alternative, achievement of the fish RG of 0.05 milligrams per kilogram (mg/kg), which would allow for consumption of one fish meal per week, would not be achieved in River Sections (RS) 1 and 2 within the model period – 59 years from completion of dredging – and would be achieved in RS 3 in 43 years. Achievement of the interim RGs of 0.4

and 0.2 mg/kg, which would allow for lesser amounts of consumption, would still take several years after completion of the remedy. For comparison, the most extensive removal alternative considered would not significantly accelerate these times.

- In the meantime, human exposure via fish consumption would be controlled through the State's fish consumption advisories and fishing restrictions, to the extent practicable.
- The remedy is protective of human health and the environment, even though it would take time after dredging to achieve the RGs for fish and even though it was recognized that the fish consumption advisories and restrictions in the meantime would not completely eliminate all PCB exposure.
- Monitoring after dredging was an integral part of the remedy, and the data to be collected will be critical to an objective evaluation of the remedy.

The State of New York concurred with the remedy specified in the ROD, with the understandings described immediately above.

The pre-design sampling program found more PCBs than described in the ROD, but the remedy was designed to accommodate those findings by using removal criteria that would be applied to the PCB mass and concentrations found as a result of the extensive pre-design sampling program.

GE agreed to perform the remedy under a Consent Decree (CD) executed with EPA, and it did so. This was one of the largest and most logistically complex environmental cleanups in history, removing significantly more PCB mass and a higher percentage of the PCB mass in the river than projected. As described in more detail in these comments, the remedy as implemented was consistent with the remedy selected in the ROD, and the benefits of dredging observed to date are within the expectations reflected in the ROD. The remedy removed over 2.7 million cubic yards of sediments and over 146,000 kilograms of Total PCBs. EPA has estimated that the PCB mass removed was 2.23 times greater that the ROD estimate. By GE's estimates, approximately 78% to 79% of the PCB mass in the river was removed, exceeding the ROD's projection of 65% removal.[1] In the areas targeted for dredging, 97% of the PCBs were removed and the small amount that remained was capped or covered with clean backfill. The estimated mass of PCBs remaining in areas outside of dredge areas is comparable to what was estimated in the ROD to be left behind.

In 2016, GE completed the active remediation specified in the ROD in accordance with the CD and all other requirements established by EPA; and it requested EPA's Certification of Completion of the Remedial Action, which is defined in the CD to exclude post-construction operation, maintenance, and monitoring (OM&M). EPA is required to issue such a certification when it determines that the "Remedial Action," as so defined, has been completed in accordance with the CD.

EPA's Five-Year Review and Support for Its Conclusions

The purpose of a five-year review is very different from the purpose of the original ROD which selected the remedy. Under EPA guidance, the purpose of a five-year review is to assess whether the

[1] See Section 6.1 of these Comments for an explanation of GE's estimates of PCB mass removed and remaining in the Upper Hudson River.

previously selected remedy is or will be protective of human health and the environment by evaluating whether that remedy is functioning as intended and whether the assumptions underlying that remedy remain valid. In the absence of compelling new information, it is not an opportunity to restart the remedial selection process or look anew at remedial alternatives. Instead, its focus is to determine whether the data continue to support EPA's expectations when it decided on the remedy in the first instance, and if not, to develop appropriate recommendations.

Consistent with the purpose of the five-year review, EPA determined that the Hudson River remedy is functioning as intended, consistent with the expectations in the ROD. It determined further that the remedy will be protective of human health and the environment in time, and that in the interim, the state fish consumption advisories and fishing restrictions control human exposure pathways, to the extent practicable, as anticipated in the ROD.

EPA's determination that the remedy is functioning as intended was based on a comprehensive evaluation of the available water, fish, and sediment data. It is supported both by the rates of recovery estimated from data collected from the pre-dredging period (when natural recovery was occurring) and by post-dredging data collected in 2016. These data are consistent with ROD expectations and indicate a decline in PCB concentrations. Indeed, the 2016 results provide indications of a positive environmental response to the remedy. For example, those results indicate that, in the Upper Hudson River north of Albany, where the dredging occurred, PCB levels in water declined as much as 73% from pre-dredging levels, and that south of Albany, where PCB levels were already significantly lower prior to dredging, PCB levels declined as much as 36%. Additionally, the 2016 fish data indicate that fish are beginning to recover. However, as planned, additional water, fish, and sediment data will be collected for the foreseeable future to verify that the dredging remedy and ongoing natural recovery will reduce PCB concentrations to the target levels as anticipated in the ROD.

EPA's conclusions are also supported by the recent independent expert report for the Hudson River Foundation (HRF) (Farley *et al*. 2017), which concluded that monitoring should continue for the foreseeable future to determine whether the remedy plus ongoing natural recovery will reduce PCB concentrations to acceptable levels.

The institutional controls in place in the meantime – i.e., the State's fish consumption advisories and fishing restrictions – are operating as expected. GE provided $4 million to New York State to support these controls, and the New York State Department of Health (NYSDOH) has taken numerous steps to improve outreach and communications. In addition, GE has agreed, as part of long-term monitoring, to conduct supplemental fish sampling for NYSDOH's continued evaluation of the advisories. As a result of these activities, the institutional controls are as effective as practicable to control exposures, as the ROD contemplated.

As shown above, the ROD concluded that the selected remedy is protective of human health and the environment. As also noted above, the remedy is functioning as expected to date. As a result, the ROD's conclusion on protectiveness remains valid. EPA's current protectiveness determination is phrased differently, but has the same effect – i.e., that remedy is expected to be protective and no additional dredging is necessary at this time. As EPA recognizes, long-term monitoring of fish, water,

and sediment will be necessary to evaluate the river's rate of recovery and thus to determine the long-term protectiveness of the remedy.

Lack of Justification for Additional Dredging

Some have argued that the remedy as outlined in the ROD is not protective and that additional dredging is necessary. These calls for additional dredging fail to recognize that, in selecting the remedy in the ROD, EPA already found that additional dredging would not deliver better results in a significantly shorter time. They are also inconsistent with the purpose of a five-year review and are unsupported by sound evidence.

One of the documents on which the advocates for more dredging place primary reliance is a publication by the National Oceanic and Atmospheric Administration (NOAA), presenting the results of a model which NOAA claims show that the fish in the Lower Hudson will recover at a much slower rate than predicted in the ROD. As detailed in these comments, the NOAA model is demonstrably invalid for a number of reasons, including the fact that it fails to mimic actual data, a critical test for determining any model's validity and reliability. As further discussed in these comments, the other arguments raised by the advocates in an effort to show that recovery rates are slower than predicted by EPA's model are likewise unsupported.

The conclusion that additional dredging is not necessary at this time is supported by the independent HRF report, discussed above. That report stated that many additional years of MNA will be necessary to determine "if additional remedial action will be required" (Farley *et al*. 2017, p. 17).

1 INTRODUCTION

1.1 Purpose of Comments

The General Electric Company (GE) is submitting these comments on the U.S. Environmental Protection Agency's (EPA's) *Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site* (Second FYR; EPA 2017). Consistent with EPA guidance (EPA 2001), that report provides an evaluation of whether the remedies previously selected by EPA for the Hudson River PCBs Superfund Site (the Site) are functioning as intended by the decision documents and are protective of human health and the environment.

The Second FYR addresses two operable units (OUs) at the Site – OU1, the Remnant Deposits, and OU2, the sediments in the river. The remedy for OU1, set forth in a 1984 Record of Decision (ROD) (EPA 1984), consisted of the construction of caps on the Remnant Deposits. The remedy for OU2, set forth in a 2002 ROD (EPA 2002a), consisted of dredging of portions of the Upper Hudson River with mass or concentrations of polychlorinated biphenyls (PCBs) exceeding certain criteria, along with monitored natural attenuation (MNA) for the PCBs that remain in the river after dredging. Those remedial activities have been completed. The Second FYR concludes that the remedies for both OUs are functioning as intended. It concludes further that the remedy for OU1 is currently protective and will be protective in the long term if an institutional control is implemented to protect the cap system, and that the remedy for OU2 will be protective when the MNA component of the remedy is complete, and in the meantime institutional controls are in place to control human exposure pathways that could result in unacceptable risks.

These comments focus on OU2. Their purpose is to present GE's perspective on the issues discussed in the Second FYR relating to OU2. They provide some important background regarding EPA's selection and GE's implementation of the remedy for OU2. They demonstrate that the existing data and other information support EPA's determination that the remedy is functioning as expected in the 2002 ROD. They show further that the ROD's conclusion that the remedy is protective of human health and the environment remains valid at this time, and that long-term monitoring will be necessary to determine the long-term protectiveness of the remedy. These comments also demonstrate that there is no basis at the present time for the additional dredging of the river that some have called for.

1.2 Structure of Comments

Following this Introduction, the remainder of these comments are organized as follows:

Section 2, Background, describes EPA's evaluation and selection of a remedy for the Hudson River sediments. It outlines the key expectations and conclusions underlying EPA's selected remedy, which are important to understand in evaluating whether the remedy is functioning as expected in the ROD. This section also summarizes GE's design and implementation of the dredging portion of the remedy, which was completed in 2015, with the remaining restoration completed in 2016. Further, it describes EPA' first Five-Year Review of the remedy, which was conducted in 2012 while the construction portion of the remedy was ongoing.

Section 3, EPA's Second Five-Year Review, explains the purpose of a five-year review under EPA guidance, and presents EPA's key determinations in the Second FYR.

Section 4, Support for EPA Determinations, demonstrates that the available data support EPA's determination that the remedy is currently functioning as expected, but that additional data are necessary to fully assess the post-construction recovery of the river. It shows further that, as EPA has also concluded, the institutional controls in the form of fish consumption advisories and fishing restrictions are functioning as anticipated in the ROD. Finally, it demonstrates that, because the ROD concluded that the selected remedy is protective of human health and the environment, and because the remedy is functioning as expected to date, the ROD's conclusion on protectiveness remains valid. It notes that continued monitoring of fish, water, and sediment will be necessary to evaluate the river's rate of recovery and thus to determine the long-term protectiveness of the remedy.

Section 5, Lack of Justification for Additional Dredging, shows that the claims of some advocates that additional dredging of the river is necessary are not only inconsistent with the purpose of the five-year review, but are not supported by the materials that have been cited in support of such claims. Specifically, this section explains that the analyses conducted by the National Oceanic and Atmospheric Administration (NOAA) to show that the recovery rates are much slower than predicted in the ROD are significantly flawed and not supported by the data. This explanation is supported by a detailed critique of a NOAA paper in Attachment A. In addition, this section shows that the claims by the New York State Department of Environmental Conservation (NYSDEC) for more dredging are misguided. The main themes in NYSDEC's August 30, 2017 comments on the Second FYR are addressed in Attachment B, which demonstrates that NYSDEC has mischaracterized the ROD's expectations and prejudged the results of the long-term monitoring.

Section 6, Other Significant Comments, presents GE's comments on several analyses or statements contained in the Second FYR. This section covers some of the more significant issues. For example, it demonstrates that, by GE's estimate (described further in Attachment C). the PCB mass left in the river in non-dredge areas is considerably lower than EPA's estimate and comparable to the estimate in the ROD.

Section 7, References, lists the references cited in these comments.

2 BACKGROUND

2.1 Selection of Remedy

In December 2000, following a decade of study, EPA released a Feasibility Study (FS) and a Proposed Plan. The FS evaluated multiple alternatives to address the PCBs in the Hudson River sediments (EPA 2000). The alternatives evaluated included some larger and some smaller than the one ultimately selected. These alternatives included no action and MNA as well as active remediation alternatives involving capping of target areas with dredging of hot spots (capping of 207 acres and removal of 1.73 million cubic yards [cy] of sediment), dredging of target areas exceeding certain criteria (targeting 493 acres, with removal of 2.65 million cy of sediment), and extensive dredging of most PCB-containing sediments in the Upper River (targeting 964 acres, with removal of 3.82 million cy of sediment). These alternatives were evaluated based on the remedy selection criteria set forth in the National Contingency Plan (NCP) under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA).[2] The evaluation utilized a computer PCB fate and transport model developed by EPA to simulate and compare the results of the various alternatives in terms of the time necessary to reach certain remediation goals established by EPA. All of the alternatives evaluated, even the most extensive, required reliance on fish consumption advisories and fishing restrictions for a considerable period of time to control exposure to PCBs via fish consumption until the remediation goals were reached.

The conceptual site model underlying the remedy ties PCBs in fish and water to fine-grained sediments, loads coming into the river from GE's Hudson Falls and Fort Edward plant sites, and the remnant deposits (FS, p. 1-41). Once the loadings from the plant sites and the remnant deposits were controlled, the fine-grained sediments were deemed to be the principal source of PCBs to the water column and the food web, in large part because of the biological activity in these sediments. Coarse-grained sediments were found to be much less important in driving flux and fish PCBs (*id*., pp. 3-13 to 3-20).

In 2002, the ROD selected a remedy from the several alternatives evaluated, based on a careful analysis of the NCP remedy selection criteria and tailored to the specific conditions of the Site. The selected remedy involved dredging in the Upper Hudson River, with removal of sediments exceeding certain numerical criteria, implementation of institutional controls, and MNA of the "PCB contamination that remained in the river after dredging" (ROD, p. iii). EPA recognized the need to strike a balance between massive dredging, which could severely damage the river ecosystem and disrupt the local communities, and its view that remediation was necessary to reduce human health and ecological risks via fish consumption. The larger dredging alternative involving removal of 3.82 million cy of sediment was rejected on the ground that, according to EPA's own model, it would not result in any significant incremental reduction in human health or ecological risks, and yet would cost

[2] The NCP criteria comprise the following: two threshold criteria consisting of (i) overall protection of human health and the environment and (ii) compliance with Applicable or Relevant and Appropriate Requirements (ARARs); five primary balancing criteria consisting of (i) long-term effectiveness and permanence, (ii) reduction of toxicity mobility, or volume through treatment, (iii) short-term effectiveness, (iv) implementability, and (v) cost; and two modifying criteria consisting of (i) state acceptance and (ii) community acceptance, which are applied after a public comment period.

much more, and thus would not be cost-effective as required by Section 121(b) of CERCLA. See ROD p. 104, stating that "[t]he selected remedy . . . is $110 million less expensive than [the larger removal alternative], without substantially greater reductions in ecological and human health risks"; and EPA's Responsiveness Summary p. 11-4, stating that "the incremental improvements in risk reduction under the more aggressive remedy do not justify the additional $110 million in projected costs."

The selected remedy in the ROD was based on a number of expectations and conclusions. These included the following:

- The then-existing sediment dataset was limited. As a result, rather than setting a simple requirement for removal of a set volume of sediments, the remedy was developed to require the collection of substantial additional data and to use numerical removal criteria so that it could be adapted to the new data collected after the ROD and before design and scaled to those results if more or fewer PCBs were found. These criteria applied to PCBs with three or more chlorine atoms (Tri+ PCBs). The criteria specified in the ROD were mass per unit area (MPA) of 3 grams per square meter (g/m^2) in River Section (RS) 1, 10 g/m^2 in RS 2, and select sediments with high concentration and high erosion potential in RS 3 (ROD, pp. ii-iii, 94-95).[3] In a subsequent decision in a dispute on GE's initial Phase 1 Dredge Area Delineation Report, EPA added surface sediment concentration criteria of 10 milligrams per kilogram (mg/kg) of Tri+ PCBs in RS 1 and 30 mg/kg of Tri+ PCBs in RS 2 and RS 3, all applicable to the top 12 inches of sediment (EPA 2004). The application of both the MPA and the surface sediment criteria was to be based on sampling designed to identify areas of sufficient size exceeding the criteria to warrant removal from an engineering perspective (not to identify or designate for removal every discrete location exceeding the criteria).[4]
- The remedy would involve remediation of 493 acres and removal of 2.65 million cy of sediment, estimated to contain approximately 70,000 kilograms (kg) of Total PCBs, from approximately 40 miles of river, with the majority occurring in RS 1 (ROD, pp. i, ii, 60, 94).
- The dredging would be performed in two phases, with Phase 1 to constitute the first year of the dredging project, to be performed at a reduced rate for evaluation purposes, and Phase 2 to constitute the remainder of the dredging project (ROD, pp. iii, 95).
- Although the remedy required dredging only in the Upper Hudson River, it included MNA for the PCB contamination remaining in the river after dredging, which includes the PCBs in the Lower

[3] EPA divided the Upper Hudson River into three sections: River Section 1, extending from the former location of the former Fort Edward Dam to the Thompson Island Dam (approximately 6.3 river miles) and comprising the Thompson Island Pool (TIP); River Section 2, extending from the Thompson Island Dam to the Northumberland Dam (approximately 5.1 river miles); and River Section 3, extending from the Northumberland Dam to the Federal Dam at Troy (approximately 29.5 river miles).

[4] EPA's Responsiveness Summary explained that the criteria "were applied more as guidelines rather than absolute rules," and that "it is not appropriate to apply the criteria on a strict basis because of the high degree of variability of the sediment contamination; an isolated high value in the middle of a region of low remediation does not represent an appropriate remediation target" (EPA 2002b, p. 4-20). It also explained that other factors "such as sediment type, bathymetry, and proximity to shore" are also relevant (*id.*), and further that EPA's approach "serves to yield areas of sufficient size to permit an efficient dredging operation" (*id.*, p. 4-21).

Hudson River (part of the same Site) that are attributable to releases from the GE facilities in the Upper Hudson River. Indeed, EPA explained the benefits of the remedy for the Lower Hudson as well as the Upper Hudson River (ROD, pp. 51, 75, 103-105).

- PCBs would be left behind in the river, but those PCBs would be either buried (and not available for exposure) or at acceptable levels such that the surface sediments of the non-dredge sediments would be expected to recover at acceptable rates.
- In selecting PCB inventory (i.e., MPA) as a criterion for removal, it was understood that, in most cases, the majority of the PCB inventory was found in the top 9 inches of the sediment (FS, p. 3-17). Finding PCBs more deeply buried was not a relevant criterion. In fact, deeply buried PCBs could be left in place in the downstream river sections as defined by the "select" criterion for RS 3.
- The remedy was expected to achieve certain Remedial Action Objectives (RAOs) in fish, water, and sediments over time (ROD, pp. 50-51). To achieve these RAO, numerical Remediation Goals (RGs) were established for PCBs in fish and water, but not sediment (*id*.). These included:
  - A health-based RG of 0.05 mg/kg in fish fillets, which would allow for human consumption of one fish meal per week;
  - Interim health-based RGs of 0.4 and 0.2 mg/kg in fish fillets, which would allow for consumption of one fish meal every 2 months and one fish meal every month, respectively;
  - Ecologically based RGs of 0.3 to 0.03 mg/kg based on consumption of larger fish (represented by largemouth bass) by the river otter, and 0.7 to 0.07 mg/kg based on consumption of smaller fish (represented by spottail shiner) by mink; and
  - Surface water Applicable or Relevant and Appropriate Requirements (ARARs) of 500 ng/L, the federal maximum contaminant level for drinking water; 90 nanograms per liter (ng/L), the New York standard for protection of human health and drinking water; 14 mg/L, the federal water quality criterion for freshwater (based on fish consumption by mink); and 30 ng/L, the federal water quality criterion for saltwater, in any affected saltwater.[5]

  Achievement of those RGs, particularly for fish, would take a number of years after the remedy was complete. The specific times presented in the ROD to reach the various target levels in fish were presented for the purpose of comparing the relative effectiveness of the remedial alternatives, not as absolute predictions of those time periods. However, under all alternatives, it was recognized that it would take a substantial amount of time for fish PCB concentration to reach levels that would allow unrestricted consumption. For example, under the selected remedy, achievement of the RG of 0.05 mg/kg, allowing for human consumption of one fish meal per week, would not be achieved in RS 1 and RS 2 within the model projection period (59 years from completion of dredging) and would be achieved in RS 3 in 43 years. Achievement of the interim RGs of 0.4 and 0.2 mg/kg, which would allow for lesser amounts of consumption, would still take several years after completion of the remedy, ranging from 16 to >59 years in RS 1 and

[5] The other surface water ARARs listed in the ROD (pp. 50-51) were waived by EPA as technically impracticable to attain (ROD, p. 107).

RS 2 and 2 to 6 years in RS 3.[6] For comparison, the most extensive removal alternative considered would not substantially accelerate these times. For example, under that alternative, the RG of 0.05 mg/kg would still not be achieved in the Upper Hudson River as a whole within the model projection period (see ROD, p. 103).

- In the meantime, human exposure via fish consumption would be controlled through fish consumption advisories and fishing restrictions (ROD, pp. iv, 96). As EPA stated, "the protectiveness of the selected remedy is further enhanced through continuation of institutional controls, such as the fish consumption advisories and fishing restrictions" (*id*. p. 106). EPA noted, however, that these controls depend on voluntary compliance and thus do not entirely eliminate human exposure to PCBs, and that they also do not protect piscivorous ecological receptors (*id*., pp. 79, 104; Responsiveness Summary, pp. 3-25, 11-1).
- Overall, "[t]he selected remedy is protective of human health and the environment" (ROD p. 106). EPA reached this conclusion even though it recognized it would take substantial time after dredging to achieve the RGs for fish and that the institutional controls in the meantime would not totally eliminate PCB exposure via fish consumption.
- Monitoring after dredging to determine when RGs are reached was an integral part of the remedy (ROD, pp. iv, 61, 96) and the data to be collected were critical to allow objective evaluation of the remedy. The inclusion of post-construction monitoring as a critical part of a remedy for contaminated sediments is consistent with EPA's *Contaminated Sediment Remediation Guidance* (EPA 2005), which makes clear that such monitoring should be a part of all sediment remedies to determine if the remedial actions are effective and if and when cleanup levels and RAOs are met (pp. 7-17, 8-1).

The above expectations and conclusions were well known to the parties who participated in discussion of the ROD remedy, including the same ones commenting today. Indeed, the State of New York, including NYSDEC and the NYSDOH, concurred with the remedy specified in the ROD (Crotty 2001), with the understandings described above.

2.2 Implementation of Dredging Portion of Remedy

After EPA selected the remedy, GE proceeded to conduct the necessary sampling and design work under administrative consent orders with EPA (EPA 2002c, 2003). During pre-design, an extensive sampling program was implemented. The sampling program found more PCBs than described in the ROD; but, as noted above, the remedy was designed to accommodate such findings by using numerical removal criteria that would be applied to the actual PCB mass and concentrations found so as to scale the remedy to the sampling results. Based on the extensive sediment sampling and other data collected by GE, GE completed and EPA approved Phase 1 and Phase 2 Dredge Area

[6] To achieve the 0.4 mg/kg target level, the ROD estimated that it would take the following amounts of time from the completion of dredging: 17 years in RS 1, 16 years in RS 2, and 2 years in RS 3, with an average of 5 years. To achieve the 0.2 mg/kg target level, the ROD predicts that it would take the following amounts of time from the completion of dredging: over 59 years in RS 1, 32 years in RS 2, and 6 years in RS 3, with an average of 16 years. To achieve the 0.05 mg/kg RG, the ROD predicts that it would take over 59 years (more than the model projection period) in RS 1 and RS 2 and on average, and 43 years in RS 3. See ROD, pp. 72-73, 103, 106; Responsiveness Summary, Book 3, Table 363176-5.

Delineation (DAD) Reports in 2005 and 2007, respectively (QEA 2005, 2007), delineating the horizontal and vertical extent of the dredge areas to meet the applicable removal criteria established by EPA and thereby to satisfy the requirements of the ROD for sediment removal.

In 2005, GE and EPA executed a Consent Decree (CD) to govern implementation of the remedy (EPA and GE 2005). It provided that GE would carry out Phase 1 of the dredging project and that, after a post-Phase 1 peer review and EPA's decision on any changes to the performance standards and the scope of the project for Phase 2, GE would elect whether to perform Phase 2 under the CD. The CD included a Statement of Work for Remedial Action and Operation, Maintenance, and Monitoring (SOW), which set forth general requirements and procedures for the remedial action.

GE conducted Phase 1 of the remedial action in 2009. In 2010, following a peer review and EPA's issuance of revised performance standards and a revised SOW, GE agreed to conduct Phase 2 of the remedial action. GE conducted Phase 2 of the dredging in 2011 through 2015, with final completion of the required habitat replacement/reconstruction in 2016. Both Phase 1 and Phase 2 of the active remedial action were based on design documents approved by EPA.

By GE's estimate, the remedial action removed 2,754,324 cy of sediments and 146,015 kg of Total PCBs (45,681 kg of Tri+ PCBs). Approximately 10% of this removal occurred during Phase 1, with the remainder in Phase 2. These estimates are similar to EPA's estimates, which are that the dredging removed 2,641,926 cy of sediments and 155,760 kg of Total PCBs (48,600 of Tri+ PCBs) (Second FYR, p. 20). The PCB mass removed was much greater than anticipated. The Second FYR estimates that the PCB mass removed was 2.23 times greater that the ROD estimate of 69,800 kg (*id*., p. 41) and constituted 72% of the overall PCB mass from the Upper Hudson River, compared to 65% assumed in the ROD (*id*., p. 4). By GE's estimate, as discussed further in Section 6.1 below, over 79% of the PCB mass in the river was removed, and the estimated mass remaining in non-dredge area is similar to the ROD estimates.

In addition, the EPA performance standards allowed for engineered capping of residual sediments following dredging in certain limited circumstances – e.g., where the average Tri+ PCB surface concentration after the initial dredging pass was greater than 1 mg/kg but less than 27 mg/kg and re-dredging was not required to address remaining PCB inventory (Tri+ PCB concentrations greater than or equal to 6 mg/kg in sediments deeper than 6 inches) or where inventory or surface concentrations above 1 mg/kg were still present after a second dredging pass). During Phase 1, approximately 84,000 square yards (17.3 acres) were capped out of approximately 48 acres dredged (about 36%). In Phase 2, based on a Nodal Capping Index (NCI) developed by EPA, which was designed as a surrogate for the percentage of area capped but excluded certain capped areas from that metric, the percentage of the total Phase 2 area dredged that was capped, as measured by the NCI, was approximately 7.77%, and the percentage of the total Phase 2 area dredged that was capped with inventory present, as measured by the NCI, was 0.50%. These percentages were well below the capping limits established by EPA for Phase 2 and indicate that the capped areas generally contain very low amounts of PCB mass.

GE completed the remedy specified in the ROD in accordance with the CD and all other requirements established by EPA. Upon the completion of dredging, EPA noted that the project was an "historic achievement" (EPA Statement on Hudson River Cleanup, Oct. 1, 2015). On December 23, 2016, GE

submitted a *Remedial Action Completion Report* (Parsons 2016). EPA has not to date approved that report and issued a Certification of Completion of the Remedial Action, which is defined in the CD to exclude post-construction operation, maintenance, and monitoring (OM&M). EPA is required under the CD (Paragraph 57.e) to respond to GE's request for such a Certification of Completion no later than one year of submission of the completion report.

In short, GE has respected the process that EPA followed in selecting the remedy, and it has fully implemented the construction portion of the selected remedy. All parties should likewise respect that process, which included substantial public input along the way, and allow the next step in the remedy, long-term monitoring, to proceed without prejudging the outcome.

2.3 First Five-Year Review

In June 2012, during the implementation of Phase 2 of the dredging project, EPA completed the first Five-Year Review and issued the *First Five-Year Review Report* (First FYR; EPA 2012). In that report, EPA recognized that PCB levels in surface sediments were higher than expected at the time of the ROD (*id*., p. 27). Based on the post-ROD sampling results collected prior to dredging, EPA estimated that the recovery rate of surface sediments would be greater than predicted in RS 1, comparable to predicted in RS 3, and notably lower than predicted in RS 2 (*id*., p. 33). As to the RS 2 estimate, EPA concluded that, given the "uncertainties in the model forecasts," the "long periods anticipated to achieve the remedial goals," and the favorable findings in RS 1 and RS 3, "EPA believes that the design of the dredging and MNA remedy will achieve the RAOs and specific fish remediation goals identified in the ROD and that this potential delay to achieve remedial goals in River Section 2 is not deemed a sufficient reason to modify the remedial design" (*id*.), and thus "additional dredging is not necessary to achieve the ROD objectives" (*id*., p. 32). EPA determined that the remedy under construction "will be protective of human health and the environment upon completion," and that "[i]n the interim, human exposure pathways that could result in unacceptable risks are being controlled" (*id*., p. 40).

The First FYR also included a few specific near-term recommendations – that additional sampling should be performed in an area adjacent to dredge Certification Unit (CU) 1, that additional surface sediment data should be collected from RS 2 and RS 3, that EPA would work with the State to assess whether additional and/or more effective outreach techniques are available to communicate fish consumption advisories and fishing restrictions, and that navigation dredging might be necessary as the dredging project moved south (id., p. 39). These recommendations were subsequently implemented, although, instead of additional sampling in the area adjacent to CU 1, GE conducted additional dredging in that area, as agreed with EPA.

3 EPA'S SECOND FIVE-YEAR REVIEW

3.1 Purpose of Five-Year Review

In reviewing EPA's Second FYR, it is important to recognize that the purpose of a five-year review is very different from the purpose of the original ROD which selected the remedy. Under EPA's guidance, the purpose of a five-year review is to "evaluate the implementation and performance of a [previously selected] remedy in order to determine if the remedy is or will be protective of human health and the environment" (EPA 2001, p. 1-1). It does this by assessing whether the previously selected remedy is functioning as intended, whether the risk assumptions and RAOs underlying that remedy remain valid, and whether there is any other, new information that could call into question the remedy's protectiveness (*id*., p. 4-1). This process is a technical assessment of how the already-selected and implemented remedy is performing. It is not an opportunity to restart the remedial selection process or look anew at remedial alternatives. Instead, its focus is to determine whether the data continue to support EPA's expectations when it decided on the remedy in the first instance, and if not, to develop appropriate recommendations.

3.2 EPA Determinations

Consistent with the purpose of the five-year review, EPA determined in the Second FYR that the remedy for OU2 was implemented and is functioning as intended, consistent with the expectations in the ROD, and that additional monitoring is necessary to confirm that it continues to do so (pp. 3-6). EPA determined further that the remedy will be protective of human health and the environment in time (namely, when the MNA component of the remedy is completed), and that in the interim, the state fish consumption advisories and fishing restrictions control human exposure pathways, to the extent practicable, as anticipated in the ROD (pp. 8, 71). Thus, the Second FYR did not identify the need for additional response actions other than OM&M.

4 SUPPORT FOR EPA DETERMINATIONS

4.1 The Remedy Is Functioning as Expected.

EPA's determination that the remedy is functioning as intended is fully supported by the available data. In this regard, it is important to recognize that the water column and fish data are more important than the sediment data in evaluating the recovery of the river and whether the RAOs and RGs set forth in the ROD are being achieved. The water and fish data are better indicators of recovery because they reflect the overall impact of the remediation and natural recovery on the PCB concentrations in the river and because there are consistent, long-term data sets available, whereas there is no single, consistent sediment data set available and sediment concentrations are highly variable. Moreover, the ROD establishes numerical RGs for PCB concentrations in fish and refers to numerical PCB concentrations in water as ARARs (see ROD, pp. 50-51), whereas there are no such numerical RGs or targets for sediment. Therefore, it is critical to take account of the water column and fish data in evaluating the recovery of the river and assessing achievement of the RAOs and RGs. The sediment data are not sufficient for that objective, although they are part of the overall picture. EPA has made the determination that the remedy is functioning as expected because the rates of recovery estimated from the data collected during the pre-dredging period (1995 to 2008) are internally consistent across media and are consistent with expectations presented in the ROD. EPA has also evaluated the benefit of the remedy in terms of reduction of PCB concentrations in water, fish, and sediment both before and after dredging. In terms of the remedy benefit, data collected in 2016 are consistent with ROD expectations but additional data are needed to fully assess the post-remedy recovery. Pre-dredging and post-dredging recovery are discussed below.

4.1.1 Pre-Dredge Rates of Recovery

EPA has estimated water column Tri+ PCB recovery rates from data collected during the pre-dredging period when the river was undergoing natural recovery; those estimated recovery rates are 10%, 13%, 5%, and 6% per year for Thompson Island Dam, Schuylerville, Stillwater, and Waterford, respectively (Second FYR, Appendix 1, p. 4-2). These rates are generally consistent with rates of 10%, 10%, 10%, and 11% per year that EPA estimated for these stations by the HUDTOX model results presented in the ROD (*id*., Appendix 1, Table A1-7), although declines are slightly lower than expected at Stillwater and Waterford. This may be due, in part, to variability in the data, particularly at high flows. That variability can be controlled, to some extent, by focusing on PCB water column concentrations measured during low-flow periods. Using rates based on summer low-flow data (July through September) would thus increase confidence in the rates of decline.[7] Those rates, estimated at 11%, 12%, 8%, and 9% per year for these stations, are more comparable to ROD expectations (Figure 1).

EPA has noted that pre-dredging rates of decline estimated from Upper Hudson River fish tissue data typically range from 12% to 20% per year on a wet-weight basis and have an average of 8% on

[7] Analyses in the Feasibility Study acknowledge the seasonal variability of PCB flux due to biological activity and the correlation of water concentration to river flow (FS p. 3-12,). By constraining the rate of recovery estimates to periods when it is believed that biological activity is at its highest (i.e., summer) and filtering those summer data to flow bins, some of the variability in the data can be controlled, thereby allowing more certainty in the recovery estimates.

a lipid-normalized basis for the adult sport fish species (Second FYR, Appendix 3, pp. 4-5, 4-6). These rates are generally comparable to the ROD model predictions, as the majority of the rates estimated for the different species are within a factor of two or three of the model predictions (*id.*, Appendix 3, Table A3-4). GE's own analyses of the rates of recovery for fish on a species-by-species basis and for each pool of the Upper River (Figures 2a to 2f) are generally comparable to the species- and location-specific rates presented by EPA in Table A3-3.[8] This further supports EPA's estimated rates of recovery for fish.[9]

4.1.2 Post-Dredging Remedy Benefit

The 2016 sediment data in the non-dredge areas show that recovery has occurred relative to the sediment data collected during design. Based on the analysis provided in the First FYR (and repeated in the Second FYR), the RS-wide average surface sediment concentrations were reduced due to dredging by 87%, 36%, and 5.1% in RS 1, RS 2, and RS 3, respectively (Second FYR, Appendix 4, Table A4-5). When accounting for natural recovery as well, the Second FYR reports percent reductions that range from 80% to as high as 96% (*id.*), indicating that the primary source of PCBs to the water column has been greatly reduced. These reductions are clearly reflected in the water column data, with 2016 results consistent with ROD expectations (*id.*, Appendix 1, Table A1-10) and lower than concentrations prior to and during dredging (*id.*, Appendix 1, Figures A1-1 and A1-5). Using the data collected during low-flow periods (in this case, May through December) to control for some of the data variability, comparisons of PCB water concentrations before and after dredging show 73%, 58%, and 52% declines at Thompson Island, Lock 5, and Waterford, respectively (Figure 3a). The 2016 water data also indicate that the water column ARARs that had been deemed attainable in the ROD (excluding the 14 ng/L freshwater quality criterion, discussed in Section 6.6) have been reached consistently. Finally, the 2016 fish results suggest that fish are beginning to recover (see Second FYR p. 45), but more data are needed. The Second FYR notes that median PCB concentrations in largemouth bass in 2016 were close to the interim RG of 0.4 mg/kg and those in yellow perch achieved that level (*id.*).

In short, the initial data on PCB levels in the water column, fish, and surface sediment are promising and provide preliminary indications of a positive system response to the remedy. However, as EPA recognizes (*id.*, p. 5), data from the initial year after dredging "are not sufficient to identify post-dredging trends with a high degree of confidence, and likely reflect continued impacts from dredging operations," and hence "additional monitoring is needed" to fully assess the post-remedy

[8] The 1997 data for RS 2 on Figures A3-3A and A3-10A in Appendix 3 of the Second FYR appear to be plotted in error and are not included on GE's plots; the source of these data appears to be the PTI Food Web Study (PTI 1998), which was only conducted in the Thompson Island and Stillwater Pools (located in RS 1 and RS 3, respectively). The NOAA on-line database for the Hudson River incorrectly lists the river mile for these data as 187, which falls within RS 2 and thus is the likely source of the error.

[9] As noted above, the water and fish data are better indicators of recovery than sediment data, which produce uncertain recovery rates. Nevertheless, we note that EPA's estimated rates of decline from the sediment data range from 5% to 7% (Second FYR, Appendix 4, Table A4-4), which are consistent with the HUDTOX model predictions and support the rates estimated by the water and fish data.

recovery of the River. This is consistent with the fact that, as noted above, monitoring was an integral part of the remedy.

4.1.3 Independent Support by Hudson River Foundation Report

EPA's conclusions are largely supported by the recent independent expert report for the Hudson River Foundation (HRF), entitled *An Independent Evaluation of the PCB Dredging Program On the Upper and Lower Hudson River* (Farley *et al*. 2017). That report concluded that the dredging and natural recovery in the river resulted in a reduction of PCB concentrations measured at Waterford under low-flow conditions, which results in reduced PCB loads to the Lower River (*id*., pp. ii-iii). The report also concluded that the data to date preliminarily indicate decreases in PCBs in fish tissue (*id*., p. ii).[10] Finally, the HRF report concluded that monitoring should continue for the foreseeable future to determine whether the remedy plus ongoing natural recovery will reduce PCB concentrations to acceptable levels. It stated (*id*., p. 17): "As described in the ROD (EPA 2002), additional years of MNA will be required to meet [Total PCB] target levels and remediation goals for fish. Post-dredging monitoring is therefore expected to continue into the foreseeable future to determine if MNA will be effective in reducing PCB concentrations to acceptable levels or if additional remedial action will be required." GE agrees with that conclusion and will be discussing the long-term monitoring program for the Hudson River with EPA.[11]

4.1.4 Institutional Controls Are Operating as Expected.

As noted in Section 2.1, EPA concluded in the ROD that institutional controls in the form of fish consumption advisories and fishing restrictions would control human exposures until the long-term RG of 0.05 mg/kg in fish fillets is met, but it recognized that these advisories and restrictions are based on voluntary compliance and thus would not totally eliminate human exposures and that they also would not prevent ecological exposures. Even considering those qualifications, the ROD concluded that the remedy would be protective (p. 106).

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[10] However, there are some conclusions in the HRF report that warrant further analysis – namely, the statements related to remedy benefit under high-flow conditions and the impact of the remedy on forage fish concentrations (p. ii). While a flow versus concentration relationship is an acceptable approach to compare pre- versus. post-dredging data, no conclusions can be drawn with respect to high flow. The post-dredging high-flow data available at the time of the HRF report was one event in February 2016 – the first high-flow event after the dredging was complete. One event is not enough data to draw any conclusions, especially considering the PCB concentrations for this particular event may have been impacted by redeposited sediments. With respect to the report's conclusions on forage fish, the report's use of geometric means of PCB concentrations in the various species of forage fish biases the comparison of pre- and post-dredging concentrations because the geometric means are driven by outliers and thus do not represent the average exposure to predators. Comparisons of arithmetic mean concentrations in the forage fish before and after dredging are more appropriate because they are not as affected by outlying values and represent the average exposure concentrations available to predators. Comparison of pre- and post-dredging arithmetic means indicates that the post-dredging concentrations are lower for all stations except two stations in the Northumberland Pool (ND1 and ND2) (Figure 4). For those two stations, the HRF report compares different species pre- and post-dredging, which should not be done because of differences between exposure sources, bioenergetics, etc.

[11] Those discussions will include the HRF report's suggestions for long-term monitoring.

The institutional controls are operating as expected, as EPA recognizes (Second FYR, pp. 61-62). Indeed, under the CD (¶ 72), GE provided $4 million to New York State to support the State's implementation of fish consumption advisories and fishing restrictions, and NYSDOH has taken numerous steps to improve outreach and communications (as described in Appendix 13 to Second FYR). In addition, GE has agreed, as part of OM&M, to conduct supplemental fish sampling for NYSDOH's continued evaluation of the advisories (see Phase 2 OM&M Scope, EPA 2010, pp. 2-9 to 2-10). As EPA notes, and as the ROD acknowledged, these institutional controls are not, and were not expected to be, fully effective in preventing all PCB exposures via fish consumption. However, as a result of the above-described efforts, these controls are as effective as practicable to control exposures, as the ROD contemplated.[12]

4.2 The ROD's Conclusion on Protectiveness Remains Valid.

As shown above, the ROD concluded that the selected remedy is protective of human health and the environment. As also shown above, the remedy is functioning as expected to date. As a result, the ROD's conclusion on protectiveness remains valid. EPA's current protectiveness determination in the Second FYR is phrased differently – i.e., that the remedy will be protective upon the completion of MNA – but it has the same effect: that the remedy is expected to be protective and thus no additional dredging is necessary at this time. As EPA recognized in the ROD and continues to emphasize, long-term monitoring of fish, water, and sediment will be necessary to evaluate the river's rate of recovery and thus to determine the long-term protectiveness of the remedy. As shown above, initial results are promising and consistent with expected rates of decline, and in the interim, human exposure pathways that could result in unacceptable risks are being controlled to the extent practicable, as expected in the ROD.

[12] The Second FYR sates that, in addition to these controls, "additional institutional controls may be needed in order to protect the subaqueous caps installed by GE during the dredging and to protect area in which GE conducted habitat reconstruction and replacement measures until, for example, the new plantings become established" (p. 69). EPA notes that such additional controls "may include restrictions on anchoring and other activities that may damage the caps or the new plantings" (*id*). GE is available to discuss such controls with EPA and, as necessary, with the New York State Canal Corporation and the U.S. Army Corps of Engineers.

5 LACK OF JUSTIFICATION FOR ADDITIONAL DREDGING

Some have argued that the data collected have shown that the remedy outlined in the ROD and implemented by GE is not protective and that additional dredging is necessary. Specifically, NOAA, the State of New York, and a number of environmental groups have argued that the sampling conducted following the ROD showed more PCBs and higher concentrations in the river than were known or expected at the time of the ROD and that, as a result, PCB levels in fish are not declining fast enough and more dredging is needed.

To begin with, this argument fails to recognize that, in selecting the remedy in the ROD, EPA already considered more extensive dredging remedies. EPA's analysis clearly demonstrated that additional dredging beyond the selected remedy would not deliver better results in a significantly shorter time frame. In fact, as discussed in Section 2.1, it showed that even the most extensive removal alternative would not be significantly more protective than the chosen remedy and would not appreciably reduce the number of years to achieve the same target levels.

The arguments for more dredging are also inconsistent with the purpose of the five-year review. As explained above, the purpose of a five-year review is to evaluate whether the selected remedy is functioning as intended and expected, not to begin a new evaluation of the most appropriate remedy. In this case, as also discussed above, the purpose of the Second FYR, conducted only a year or so after the remedial construction was completed, is to evaluate whether the remedy so far is operating as intended, given the expectations at the time of the ROD. The calls for additional dredging, at their core, improperly seek to have EPA go beyond that purpose and reinitiate the remedy selection process. The efforts to have EPA require more dredging at this time, before there has been anything close to sufficient time to assess whether the selected remedy will continue to function as intended, conflict with the purpose of the Second FYR and have no basis.

In addition, the advocates' claims for more dredging are not supported by the materials on which they rely, as shown in the following sections.

5.1 NOAA's Model Emulation Is Inaccurate and Misleading

One of the documents on which the advocates for more dredging place primary reliance is a publication by NOAA and its consultants (Field *et al*. 2016), reporting on a "model" which they developed, and which they call a "model emulation," to estimate future fish concentrations in the Hudson River. The authors of this article claim that their model emulation shows that fish in the Lower Hudson River will recover at a much slower rate than was predicted by the EPA model used in selecting the remedy.

EPA prepared a review and critique of this work in 2016 based on information available (EPA 2016). EPA found substantial flaws in this study that undermine its credibility, and concluded that this model "is based on analyses that did not reflect the breadth of project sediment data or the variety of fish species data across sampling stations in the Upper and Lower Hudson River, and therefore is not supported by the available evidence" (*id*., p. 3). Accordingly, EPA stated that it disagrees with NOAA's conclusions (*id*., p. 7). However, due to the lack of the necessary information, EPA was unable to obtain and run the model emulation.

Contrary to accepted scientific practice, NOAA inexplicably did not compare its model predictions to actual data. In February 2016, a request was made under the Freedom of Information Act for the model code and documentation in order to allow such a comparison to be made independently. After numerous delays, the first production of information was made in April 2017, over a year after the request was made. With the model now available, it is possible to reproduce NOAA's results, particularly during the time period prior to dredging, for which the model predictions can be compared to actual data from the river.[13] The comparison shows that the NOAA model projections for PCB levels in surface water from 2004 through 2008 and for PCB concentrations in fish from 1998 through 2008 are considerably higher than those seen in the actual data for those years, demonstrating that the NOAA model substantially overpredicts actual PCB concentrations in fish.

This comparison is described in detail, along with other flaws in the NOAA model emulation, in the detailed technical critique provided in Attachment A. The key fatal flaws may be summarized as follows:

- NOAA's estimated historical recovery rate is highly uncertain as it is based on limited sediment data and ignores water and fish data. Fish and water concentrations estimated from the 3% recovery rate that the authors estimate from the sediments, without any model emulation, result in predicted concentrations that are much higher than the actual data.
- The model emulation is based on the FS model, which NOAA claims is inaccurate. This approach to developing a model based on an allegedly inaccurate one is illogical.
- The model emulation is a flawed, unconstrained, curve-fitting exercise; slight adjustments using alternate fits of the model result in large differences in estimated water and fish concentrations.
- Validation of the model with available measured data was not conducted. Model-data comparisons were limited to data that were manipulated through an unsupportable upward adjustment. The failure to compare the model results to the available data was unjustifiable. As noted above, accepted scientific protocol requires model validation by comparing predicted results to actual data, and comparison of the NOAA model predications to actual water and fish data prior to dredging demonstrate that the model emulation is invalid and greatly overestimates the water column and fish PCB concentrations.

For these reasons, the NOAA model emulation cannot be used to reliably predict future river conditions and in fact produces highly misleading results. As such, it cannot support the claims of those who attempt to rely on it.

5.2 NOAA's Comparison of Data with EPA's Model Does Not Undercut EPA's Conclusions on Recovery Rates

NOAA also claims that the fish, surface sediment, and PCB load data do not support the recovery rates predicted by EPA's model used in the ROD, but show slower recovery (Field and Rosman 2016). These claims are likewise unwarranted.

[13] While the model emulation code has produced results close to those reflected in the published article, there are some differences, indicating that the results presented in the article were not well documented or preserved for reproducibility.

NOAA asserts that the rates of recovery for water reported by EPA are overestimated because they include a period of active source control (*id*., Slides 5 and 6). However, as noted in the Second FYR, land-side remedial efforts related to the Allen Mill event were largely completed by April 1995 (Second FYR, Appendix 1, p. 4-2). Thus, "the period from 1995 to 2008 represents a period of MNA subsequent to the Allen Mill event" and shows declines in water column PCB levels from 5% to 13% across the four routinely monitoring stations (*id*., Appendix 1, p. 4-2).

NOAA argues that rates of recovery estimated from the fish data are overestimated due to a fish processing protocol change in 2007 (a change from analyzing fish samples with the rib on to analyzing such samples with the rib off) (Field and Rosman 2016, Slides 7-8). EPA has acknowledged the possibility of some bias on a wet-weight basis based on a special study that evaluated the impact of the protocol changes (Second FYR, Appendix 3, p. 3-4), and for this reason limits the date range for the recovery calculations based on wet-weight data to 1995 to 2006 for fish processed as standard fillet, prior to any change in processing protocol. EPA included the standard fillet data through 2008 for lipid-normalized recovery estimates because the bias in PCB concentration in lipid-normalized fish tissue concentrations due to the change in protocol was determined to be less than 20% (*id.*, Appendix 3, p. 3-4), which is well within the acceptable range of measurement error.

NOAA contends that the measured loads to the Lower River were 3 times higher than that predicted by the FS model right before dredging and that the load showed "little evidence of decline" (Field and Rosman 2016, Slide 10). In fact, however, the updated FS model in the Second FYR does not support the claim of a 3-fold underprediction, indicating that most of the underprediction was related to the flows used in the original FS model. Moreover, pre-dredging PCB water concentrations during the baseline monitoring program (BMP) showed reductions at Waterford, indicating that loads to the Lower River were declining (see Figure 1, lower right panel).

NOAA maintains that that the recovery is not functioning as intended because fish concentrations are higher than predicted by the model during the BMP period (Field and Rosman 2016, Slides 11-12). However, for the Upper Hudson River species-weighted average comparison, these reviewers have limited their model-data comparisons to data that have been adjusted. The basis and method for the adjustment are not presented, but are presumably similar to the approach they used in the NOAA paper discussed in Section 5.1, which we have shown is unsupportable (see Attachment A). The Lower Hudson River model-data comparison relies on the Farley model, which was not fully calibrated (Second FYR, Section 5.1.1.3.5). Additionally, EPA has noted that the lack of correspondence between the fish decline rates in the Upper Hudson compared with those in the Lower Hudson, as well as the lack of response in Lower River fish tissue concentrations to dredging related releases of PCBs, suggest the presence of other sources of PCBs to the Lower Hudson (*id.*, Section 5.1.1.3.4). Thus, due to uncertainty in the Farley model as well as the presence of other sources to the Lower River, the Lower River fish tissue model-data comparisons presented by NOAA should not be used to evaluate the remedy.

NOAA provides a comparison of the sediment design data to historical data and uses the sediment data to assess recovery, claiming that such comparisons and assessments are relevant (Field and Rosman 2016, Slides 13-14). However, as shown in Section 5.1 and Attachment A, such analyses with the historical sediment data are highly uncertain and cannot be used to make reliable conclusions about the recovery of the river.

NOAA claims further that the underprediction of the amount of PCB mass in the targeted areas in the ROD relative to what was actually removed is an indication that a "greater mass of PCBs remain in the river post-dredging than EPA originally expected" (*id.,* Slide 20). However, the data in the non-dredge areas do not support this claim. As shown in Section 6.1 of these comments, the amount of PCB mass in the non-dredge areas is at the levels predicted in the ROD. Much of the underestimates in the targeted areas were due to the presence of woody debris; these types of conditions generally do not exist in the non-dredge areas. Therefore, it is inaccurate to assume that an underestimation of depth of contamination and PCB mass in the dredge areas results in an underestimation in the non-dredge areas, as well.

Finally, NOAA asserts that the remedy has not had the impacts on the surface sediment PCB concentrations in the Upper River as predicted by the ROD (*id*., Slides 21 and 22). However, as discussed in Section 4.1.2, recent sediment sampling indicates that PCB concentrations in RS 2 and RS 3 have declined in the non-dredge areas relative to pre-dredging levels. That, combined with the removal of the higher PCB concentrations during the dredging, has resulted in reductions in the average surface PCB concentrations of 80% to above 90% (relative to pre-dredging conditions) for the three River Sections (Second FYR, Appendix 4, Table A4-5).

5.3 NYSDEC's Arguments Do Not Support Their Claim of Unprotectiveness

NYSDEC believes that, even though it concurred in the ROD and even though GE implemented the remedy specified in the ROD, the ROD remedy is nonetheless "not protective" of human health and the environment, and that additional dredging is "likely necessary to accomplish the goals in the ROD for achieving the targeted reductions in fish PCB concentrations in the time frames set forth in the ROD" (NYSDEC 2016, pp. 41 and 40). NYSDEC contends that the remedy left greater-than-anticipated PCB concentrations in the sediments, particularly in RS 2, and that as a result the remedy will not achieve the targeted reductions in fish concentrations in the timeframes anticipated in the ROD and additional dredging is needed to do so (see *id*., pp. 28-30, 36; see also Seggos 2016). NYSDEC claims further that the dredging will result in "little additional improvement in fish PCB concentrations in the lower Hudson, particularly south of Albany" (NYSDEC 2016, pp. 37, 40).

As noted in Section 2.2, the dredging removed a much greater amount of PCBs than anticipated, and the PCBs remaining in the river are comparable to the ROD's estimate (see also Section 6.1). With respect to the point that the average surface sediment PCB concentration based on the design data in RS 2 was higher than anticipated in the ROD, EPA already addressed that issue in the First Five-Year Review, where EPA indicated that these concentrations and the associated recovery were acceptable and were "not deemed a sufficient reason to modify the remedial design" (First FYR, p. 33). EPA has not changed that conclusion. Further, EPA analyzed sediment data collected in fall 2016 and received after NYSDEC's report was prepared; and it concluded that, based on a comparison of the design data to those new data, natural recovery has occurred in of all the river sections, with an apparent decline of a 88% in the RS 2 average (Second FYR, Appendix 4, p. 5-2).

Additionally, as mentioned in Section 4.1.2, the surface water concentrations at Lock 5 have reduced by 58% relative to pre-dredging conditions, clearly indicating that the dredging has had a positive impact on RS 2. While it is too early to make any definitive conclusions about the fish, the 2016 data are promising but more data will be needed before a full assessment can be made. Further,

NYSDEC's argument that the Lower River will see "little additional improvement" is not supported by the most recent data. The 2016 water column data indicate reductions relative to pre-dredging conditions of 59% and 36% at Albany and Poughkeepsie, respectively, reflecting the positive impact of the remedy. See also Section 6.4 below.[14]

In addition to the comments discussed above, NYSDEC submitted comments on the Second FYR on August 30, 2017, attempting to show that EPA has abandoned the expectations of the ROD and that the remedy that was considered protective in the ROD is now not protective, so that additional dredging is necessary. Those arguments are addressed in Attachment B, which shows that NYSDEC has mischaracterized the ROD's expectations and prejudged the results of the long-term monitoring.

5.4 The Independent Report for the Hudson River Foundation Supports the Conclusion that Additional Dredging Is Unnecessary at This Time

The conclusion that additional dredging is not necessary at this time is supported by the independent HRF report (Farley *et al*. 2017). As discussed above, that report concluded that, as described in the ROD, additional years of MNA will be required to meet RGs for fish, and thus monitoring will "continue into the foreseeable future to determine if MNA will be effective in reducing PCB concentrations to acceptable levels *or if additional remedial action will be required*" (p. 17; emphasis added). Given this conclusion, the report did not call for additional dredging before the monitoring period is over.

[14] NYSDEC also asserts that the remedy is not protective because fish consumption advisories allow PCB exposures to anglers who do not follow those advisories and to ecological receptors (Seggos 2016, pp. 2-3; NYSDEC 2016, p. 36). However, as shown above, those considerations were fully understood by all at the time that NYSDEC concurred in the ROD.

6 OTHER SIGNIFICANT COMMENTS

In addition to the general points discussed above, GE has a number of comments on the text and appendices of EPA's Second FYR document. This section presents some of the more significant comments.[15]

6.1 PCB Mass Outside CUs

EPA's Second FYR reports the Agency's calculation that the mass of PCBs remaining in the River outside of the dredged CUs is 60,500 or 56,400 kg, depending on the method used (p. 41 and Appendix 2, p. 4-7). This appears to be an overestimate of the mass left in the river after the dredging was completed. GE has performed its own calculations, which account for the biased nature of the design data.[16] Table 1 outlines the results of those calculations, along with GE's estimates of the mass removed and the mass capped or backfilled in the targeted areas and the estimates originally provided in the ROD Responsiveness Summary (Table 363334-1). These calculations are described in more detail in Attachment C. By these estimates, GE remediated 149,800 kg of Total PCBs by removing 145,890 kg and capping or backfilling 3,910 kg.[17] GE has further calculated that there are 34,530 to 37,900 kg of PCBs remaining in the non-dredge areas of the river. This indicates that, while more mass was found in the targeted dredge areas than originally predicted by the ROD, the dredging was effective in removing the majority of the PCBs in the river (nearly 80%), and the amount of PCBs in the non-dredge areas estimated with the more robust design dataset is consistent with what was estimated by the ROD to be left behind.[18]

[15] These comments should not be considered to indicate that GE agrees with all other statements or analyses in the Second FYR. Rather, they represent GE's comments on certain selected portions of that document.

[16] The pre-dredging sampling program was spatially biased, with more samples collected in areas of suspected higher PCB concentrations (i.e., areas where finer sediments were encountered during a surface sediment type survey). Therefore, when averaging the non-dredge area data, a spatially weighted average is preferred in order to account for the biased nature of the sampling grid.

[17] Due to a difference in calculation methods, GE's estimate of the mass removed is slightly different but not significantly different from EPA's estimate (155,739 kg).

[18] The differences in the mass estimates between dredge and non-dredge areas may be related to different characteristics of the river bottom in those areas (e.g., sediment type and grain size, depositional nature of the area, extent of debris, etc.). There is more confidence in the mass estimates for non-dredge areas using the robust design dataset because they are less affected by the characteristics that resulted in missed inventory in dredge areas.

Table 1

Comparison of Total PCB Mass Estimates

River Section	ROD Estimate[1]			Post-Remedy Evaluation			
	PCB Mass Remediated (kg)	Non-dredge Mass (kg)	% PCB Mass Removed	PCB Mass Removed (kg)	Mass Capped/ Backfilled (kg)	Non-dredge Mass (kg)	% PCB Mass Removed
1	36,000	9,200	80%	84,360	2,860	1,080-1,130	95-96%
2	24,300	3,800	86%	32,380	510	4,600-4,770	86%
3	9,500	24,500	28%	29,150	540	28,850-32,000	47-50%
Total	**69,800**	**37,500**	**65%**	**145,890**	**3,910**	**34,530-37,900**	**78-79%**

Note:
[1] Per Table 36334-1 in the ROD Responsiveness Summary

6.2 Natural Resource Injury

The Second FYR asserts that "PCB levels in surface sediments outside dredged areas remain elevated and will continue to negatively impact trust resources" (p. 50). EPA has presented no definition of "elevated" levels for purposes of this statement and no support for the assertion that PCBs in the remaining sediments are negatively impacting the resources that are subject to the resource Trustees' natural resource damage (NRD) claims. The NRD process is separate from the remediation process and has been underway for over 15 years. EPA presents no evidence, nor has there been any showing, that the PCBs outside dredged areas are causing injury to the natural resources subject to the NRD process. It is inappropriate for EPA to insert conclusions about NRD into this Five-Year Review Report.

6.3 Homologue Conversion

The Second FYR states in the text that "[e]arly NYSDEC fish tissue samples were analyzed using Aroclor-based methods, while more recent NYSDEC and GE samples were analyzed using congener-based standards" (p. 56). In fact, as EPA recognizes in Appendix 3, "[f]or both NYSDEC *and GE* data, fish tissue analyses were primarily conducted using an Aroclor-based analysis, with a *subset* of the samples analyzed using a more quantitative procedure based on PCB congeners" (p. 3-2; emphases added). In any case, EPA states that, "[t]o ensure consistency and comparability across datasets, all Aroclor-based results were converted to estimates of TPCB based on homologue equivalents ($TPCB_{HE}$) through application of conversions documented in Appendix 5" (p. 56). In Appendix 3, EPA states further that the sum of Aroclors "is not always the most accurate representation of total PCB concentration in fish," and that thus "EPA developed relationships between the total PCB concentration based on PCB congener or homologue values ($TPCB_{HE}$) and the sum of Aroclors" (p. 3-3), and thereby converted all Aroclor-based results to $TPCB_{HE}$.

GE has concerns with the statement questioning accuracy of fish tissue PCB concentrations determined through Aroclor analysis. PCB concentrations measured in fish from the Hudson River have consistently been analyzed using an Aroclor method, which is an EPA-approved method and the method approved for fish tissue analysis in the *Phase 2 Remedial Action Quality Assurance Project*

Plan (RAM QAPP; Anchor QEA and ESI 2012). Comparisons of Aroclor-based Total PCB (TPCB) concentrations and congener-based TPCB concentrations for a subset of fish collected each year have been provided to EPA annually in the Data Summary Reports from 2004 through 2011 and in 2013, 2015, and 2016. These comparisons have consistently shown that Aroclor-based TPCB concentrations correspond well with the congener-based TPCB concentrations.

In addition, there are numerous issues, which EPA has not addressed, regarding the conversion of Aroclor TPCB concentrations to $TPCB_{HE}$ concentrations. These issues include the effects of species-to-species variation on the conversion, the potential relationship of the conversion ratio to PCB concentration, the possible equation to be used going forward, and how the conversion and the $TPCB_{HE}$ metric will be applied in developing fish consumption advisories and assessing the achievement of RGs. Given these issues, GE suggests that EPA indicate that the potential conversion of Aroclor TPCB concentrations to $TPCB_{HE}$ concentrations will be evaluated and discussed further following the completion of this Five-Year Review.

6.4 Lower Hudson River

The Second FYR states that the Lower Hudson River contains "other sources of PCBs . . . (although less significant than the GE sources at Hudson Falls and Fort Edward)" and has "very different" characteristics from the Upper Hudson, and that "[i]t will therefore be important to collect additional data and other information in order to better understand the PCB contamination in the Lower Hudson River" (p. 70). It also states that "[t]he effects of PCB load reduction from the Upper Hudson to the Lower Hudson are not yet fully known but are expected to benefit the recovery of the lower river," and therefore "it is important that the PCB load to the Lower Hudson continue to be monitored under OM&M for the foreseeable future and additional information be collected about other sources and PCB fate and transport to the lower river" (p. 57).

As discussed in Section 5, the advocates of additional dredging, such as NOAA and NYSDEC, disagree with these conclusions and argue that the dredging project completed will not result in a significant improvement in the Lower Hudson and that the fish in the Lower River will recover at a much slower rate than was predicted at the time of the ROD, thus leading to the need for more dredging.

The existing data suggest that the dredging project did and will benefit the Lower Hudson. As shown in Figure 3a, the comparison of low-flow conditions at Waterford indicate a 52% decline in PCB concentrations relative to pre-dredging levels, which translates to reduced loads to the Lower River. While more data is needed to assess the concentrations and loads at higher flows, the reductions that have been measured at low flow have positively impacted in the Lower River. Figure 3b shows that PCB concentrations during low flow at Albany and Poughkeepsie have declined 59% and 36%, respectively, relative to pre-dredging concentrations.

However, as with the Upper River, and as EPA recognizes, it is critical to continue to obtain monitoring data to evaluate trends in PCB levels in fish and water in the Lower River before the need for other response actions can be assessed. That has always been part of the remedy, and GE has been collecting these data and will continue to do so. That is the appropriate approach at present to addressing the Lower River. There is no need for a full Remedial Investigation/Feasibility Study for the Lower River at this time.

6.5 Revised Risk Calculations for Ecological Receptors

The Second FYR discusses changes in exposure assumptions for EPA's pre-ROD Human Health Risk Assessment and Baseline Ecological Risk Assessment (BERA) and a revised Toxicity Reference Value (TRV) for the BERA on pages 63 to 66 and in Appendix 11. Appendix 11 states that the TRV used in the BERA was 0.044 mg/kg-BW/day based on the Lowest Observed Adverse Effect Level (LOAEL) in a study by Restum *et al*. (1998), but that the authors of the appendix now use a TRV of 0.033 mg/kg-BW/day based on the LOAEL in a newer study by Bursian *et al*. (2013), with a corresponding No Observed Adverse Effect Level (NOAEL) of 0.011 mg/kg body weight per day (mg/kg-BW/day), resulting in a more conservative estimate of risk (Appendix 11, pp. 2-6 to 2-7). Based on that revised TRV, the ROD's ecological RGs of 0.3 to 0.03 mg/kg PCBs in larger fish (represented by largemouth bass) for protection of the river otter and 0.7 to 0.07 mg/kg PCBs in smaller fish (represented by spottail shiner) for protection of mink were recalculated to ranges of 0.2 to 0.07 mg/kg for fish consumed by the river otter and 0.34 to 0.11 mg/kg in fish consumed by mink (*id*., p. 2-7). These recalculated ranges, EPA states, "would be narrower than and lie wholly within the original ranges developed in the ROD," and would "not affect the protectiveness determination of the selected remedy with respect to ecological receptors" (*id*., p. 2-7; Second FYR text, pp. 65, 66).

Although, as EPA states, the revised TRV would not affect the protectiveness determination for ecological receptors, it should be pointed out that the manner in which the results of the Bursian *et al*. (2013) study were presented by the study authors has led to an incorrect interpretation of its results. In that study, mink were fed diets containing varying amounts of PCB-containing fish collected from the Hudson River. A 20% lethal concentration (LC20) for 6-week-old kits was considered to represent a LOAEL, and this concentration was divided by an uncertainty factor of 3 to estimate a NOAEL. EPA notes that the LOAEL and NOAEL from this study were 0.033 and 0.011 mg/kg-BW/day (Appendix 11, pp. 2-6, 2-7). These values were based on the authors' reported LC20 for kit mortality of 0.34 mg/kg in feed, as reported in Table 7 of that paper. That value, however, did not take into account the mortality in the control group (which had zero exposure). Indeed, kit mortality in the control group was slightly more than in the lowest PCB dose group (administered a diet containing 0.72 mg/kg). Although GE does not necessarily agree with the results of the Bursian *et al*. (2013) study, alternative values can be identified from that study to derive more appropriate TRVs. In Supplemental Data Table S2, Bursian *et al*. (2013) report an alternative LC20 for 6-week kit mortality of 1.4 mg/kg, which represents the LC20 compared to the control group. This is very similar to the LOAEL for juvenile mortality and kit body mass (the two most sensitive test endpoints) of 1.5 mg/kg PCBs in diet. That would lead to NOAELs and LOAELs about four times higher than reported by EPA (0.135 and 0.045 mg/kg-BW/day), which would correspondingly increase the TRV to a level higher than the value used in the BERA.

Because the TRV based on more appropriate values compared to the control group in Bursian *et al*. (2013) is higher than the TRV used in the BERA, use of that study would result in a corresponding increase in the ecological RGs. Alternatively, if the prior TRV continues to be used, review of this newer study would not result in a change in the ranges of the ecological RGs.

6.6 Freshwater Quality Criterion

EPA states in Appendix 1 that it is expected that the federal water quality criterion for freshwater aquatic life (14 ng/L) "will be met consistently within several decades" (p. 5-2). This statement

appears to overstate the length of time before this criterion will be achieved. The 14 ng/L criterion was already met frequently in 2016. Further, GE has assessed this statement based on the post-dredging water column data, using: (1) EPA's statement in Section 5 of Appendix 1 that post-dredging Total PCBs are expected to decline at approximately the same rate as Tri+ PCBs; and (2) Tri+ PCB decay rates of approximately 10% per year from Table A1-7. This evaluation indicates that the 14 ng/L criterion will be met *consistently* in the Lower Hudson within the next 1 to 5 years and in the Upper Hudson within the next 10 to 20 years.

6.7 Water Column Data at Poughkeepsie

The Second FYR states that the water column data at Poughkeepsie were generally higher than the Farley model predictions (which underpredicted Tri+ PCB concentrations) and do not indicate an impact from dredging (Second FYR, pp. 33, 55, and Appendix 1, pp. 4-5, 6-2). It should be noted, however, that the likely reason for the Farley model's underprediction of water column concentrations at Poughkeepsie during the pre-dredge period is that, as the report recognizes later (*id*. p. 58), the Farley model was only calibrated to sediment and fish data, not water column data. It should also be noted that, in the Farley model, Total PCB concentrations are very similar at Albany and Poughkeepsie, whereas Tri+ PCB concentrations are higher at Poughkeepsie than at Albany, suggesting the presences of local sources. Finally, the data indicate that there was a response to dredging at Poughkeepsie, as indicated by the fact that PCB concentrations in 2016 were lower than during the BMP (see Figure 3b, discussed above).

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FIGURES

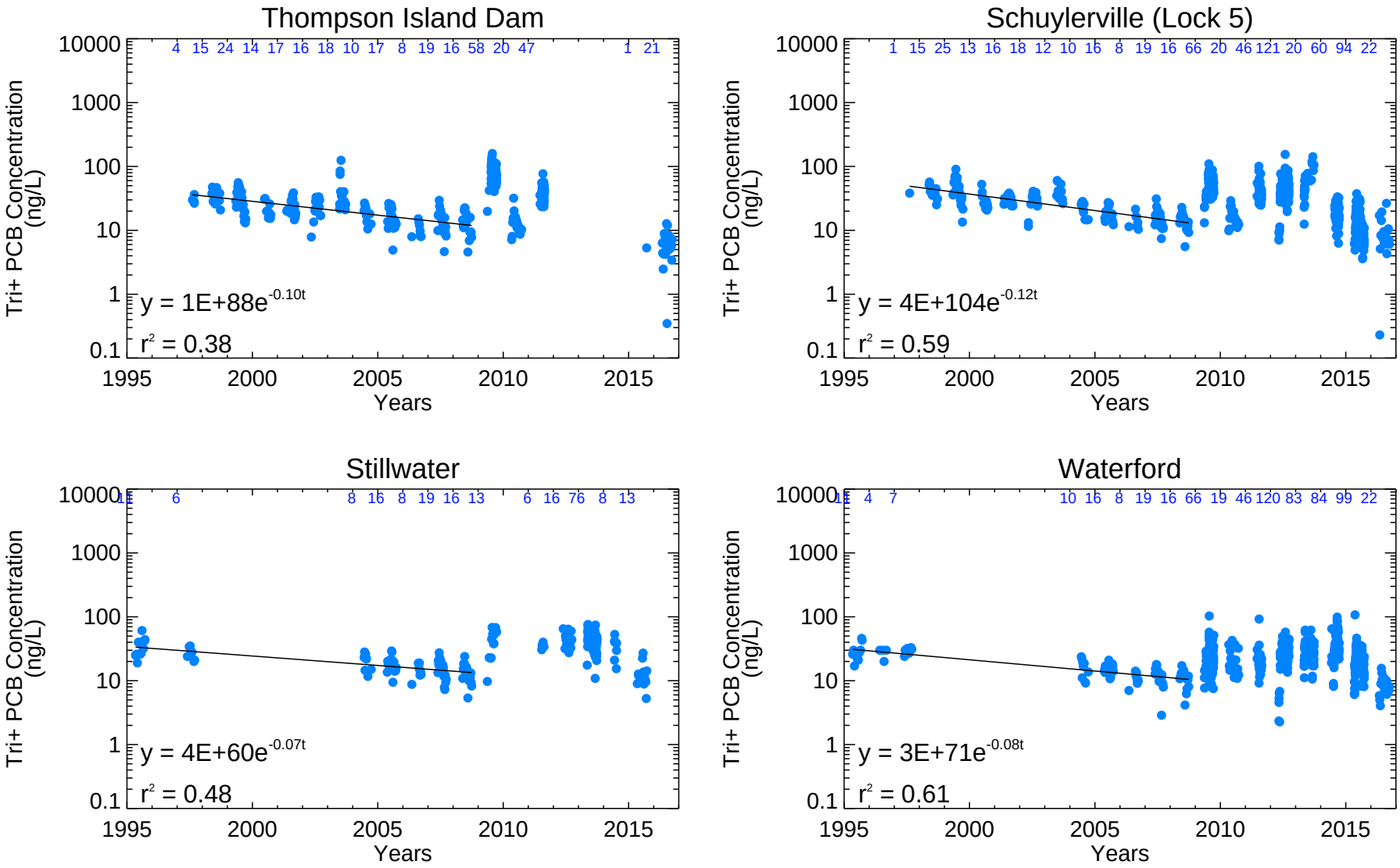

Figure 1

Time Trends in Summer Low-Flow Water Column Tri+ PCB Concentration

Data Source: USGS and GE (Post-Construction Remnant Deposit Monitoring Program, BMP, RAMP). Water data are plotted from 1995 - September 2016.
Non-detects are set to half the MDL of the maximum congener peak or Aroclor MDL concentration.
Data shown for May - September months and when FE Flow is less than or equal to 5,000 cfs.

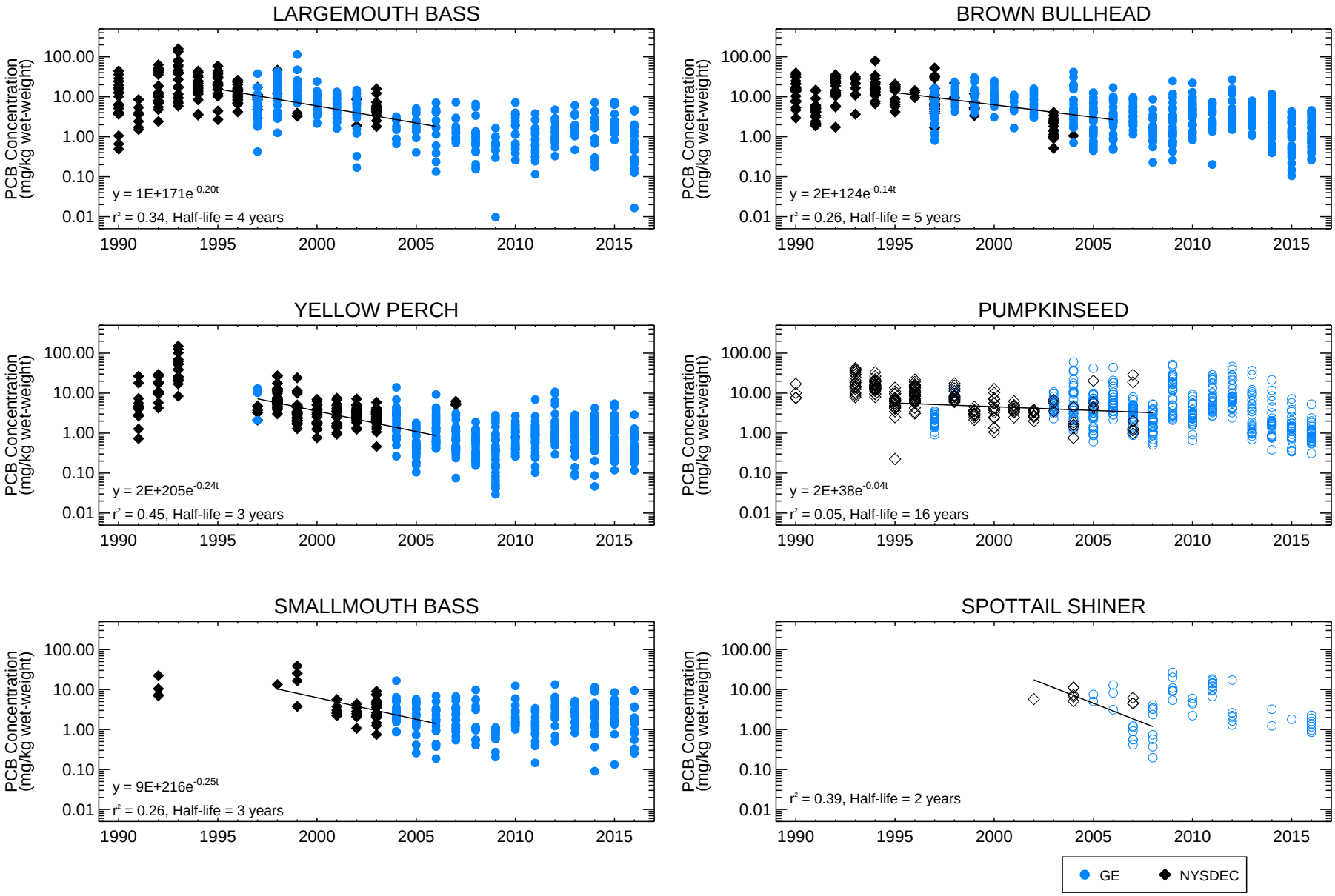

Figure 2a

Time Trends in Fish Total PCB Concentrations (Wet-weight-based) in Thompson Island Pool

NYSDEC Hudson River biota monitoring database (March, 2016) and GE RAMP database (March, 2017).
Fish data shown are from 1990 - 2016. Open symbols represent whole body and filled symbols represent fillet preparations.
Regression is based on 1995 - 2006 for Largemouth Bass, Brown Bullhead, Yellow Perch, Smallmouth Bass and 1995 - 2008 for Pumpkinseed, Spottail Shiner.

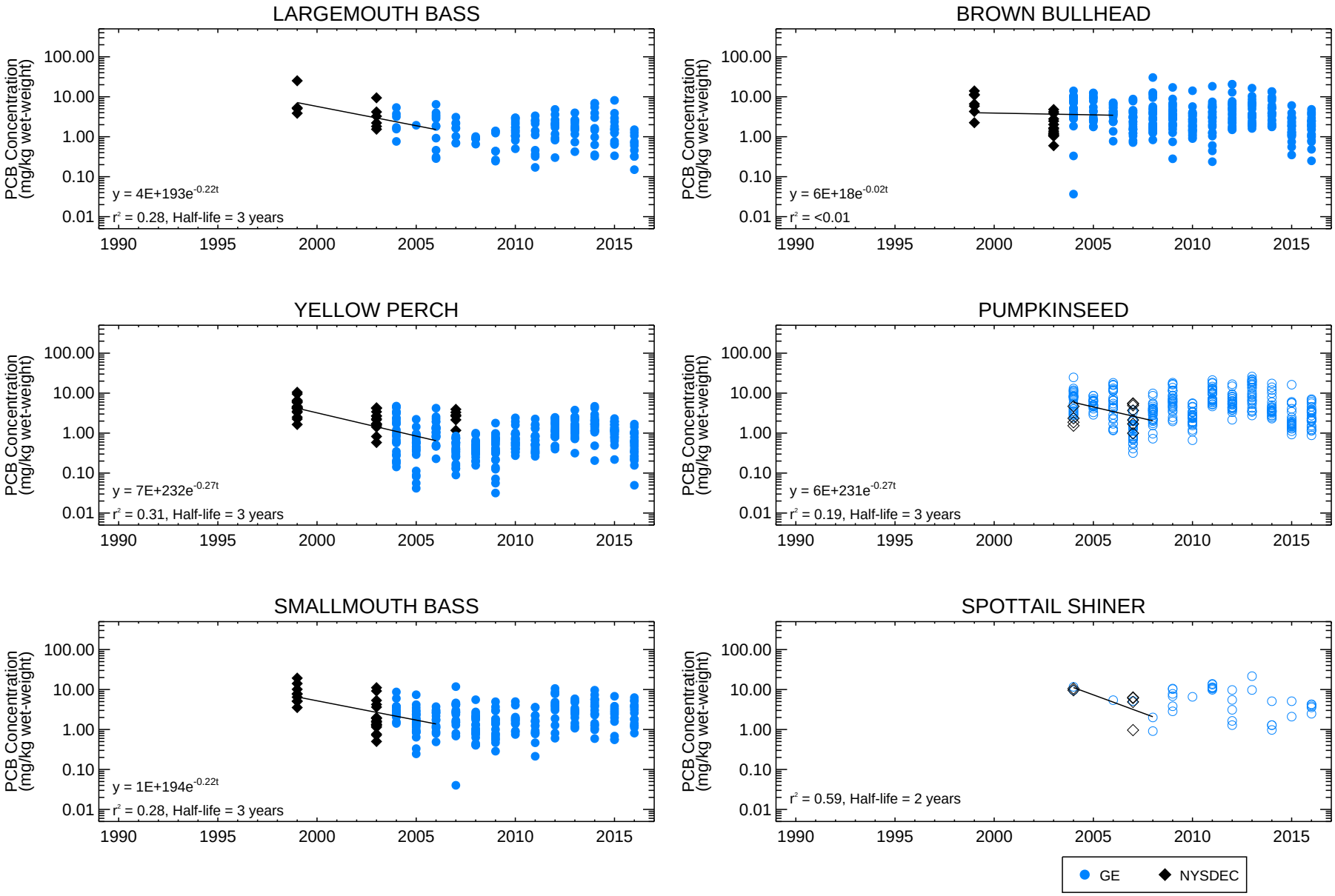

Figure 2b

Time Trends in Fish Total PCB Concentrations (Wet-weight-based) in Northumberland Pool/Fort Miller Pool

NYSDEC Hudson River biota monitoring database (March, 2016) and GE RAMP database (March, 2017).
Fish data shown are from 1990 - 2016. Open symbols represent whole body and filled symbols represent fillet preparations.
Regression is based on 1995 - 2006 for Largemouth Bass, Brown Bullhead, Yellow Perch, Smallmouth Bass and 1995 - 2008 for Pumpkinseed, Spottail Shiner.

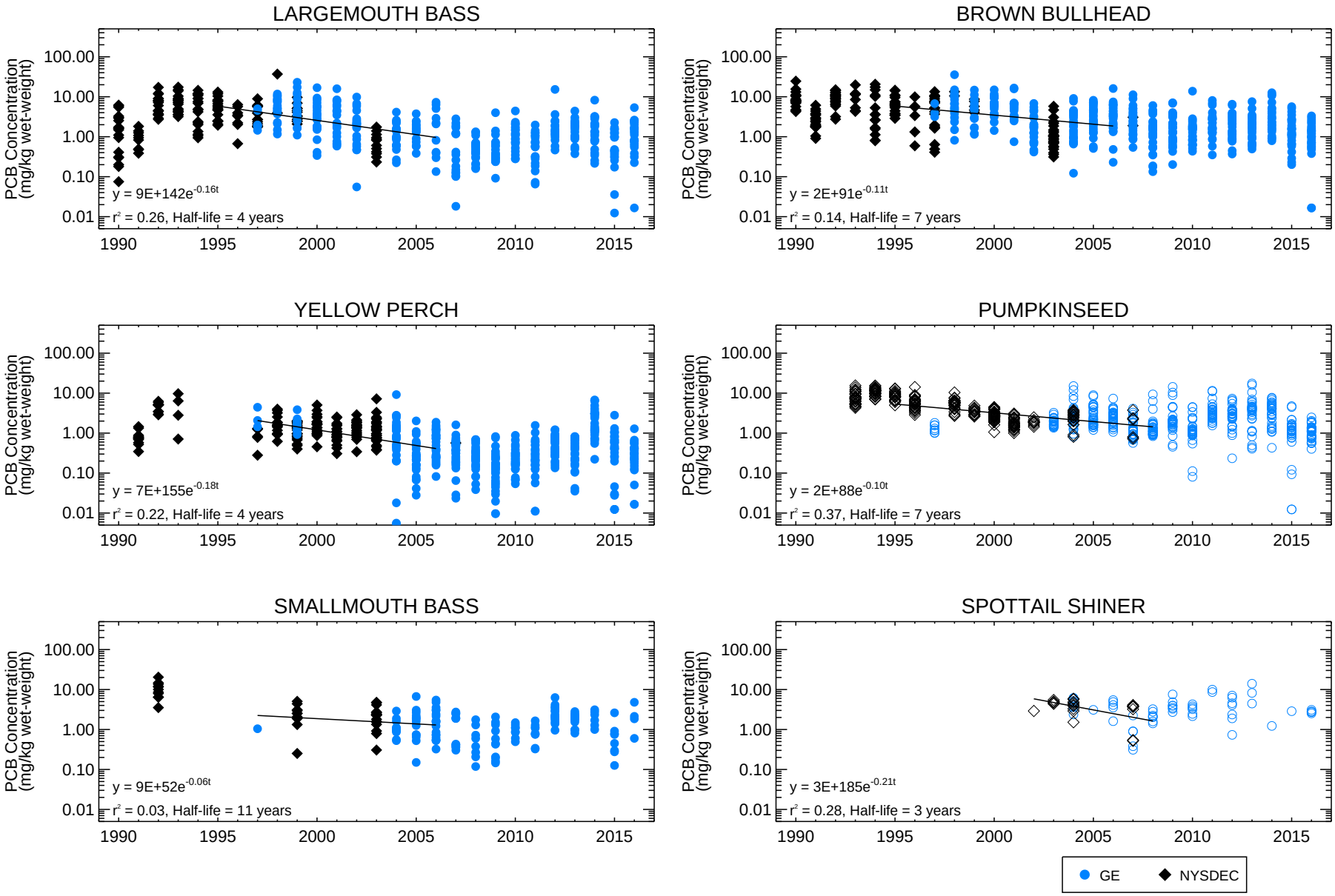

Figure 2c

Time Trends in Fish Total PCB Concentrations (Wet-weight-based) in Stillwater Pool

NYSDEC Hudson River biota monitoring database (March, 2016) and GE RAMP database (March, 2017).
Fish data shown are from 1990 - 2016. Open symbols represent whole body and filled symbols represent fillet preparations.
Regression is based on 1995 - 2006 for Largemouth Bass, Brown Bullhead, Yellow Perch, Smallmouth Bass and 1995 - 2008 for Pumpkinseed, Spottail Shiner.

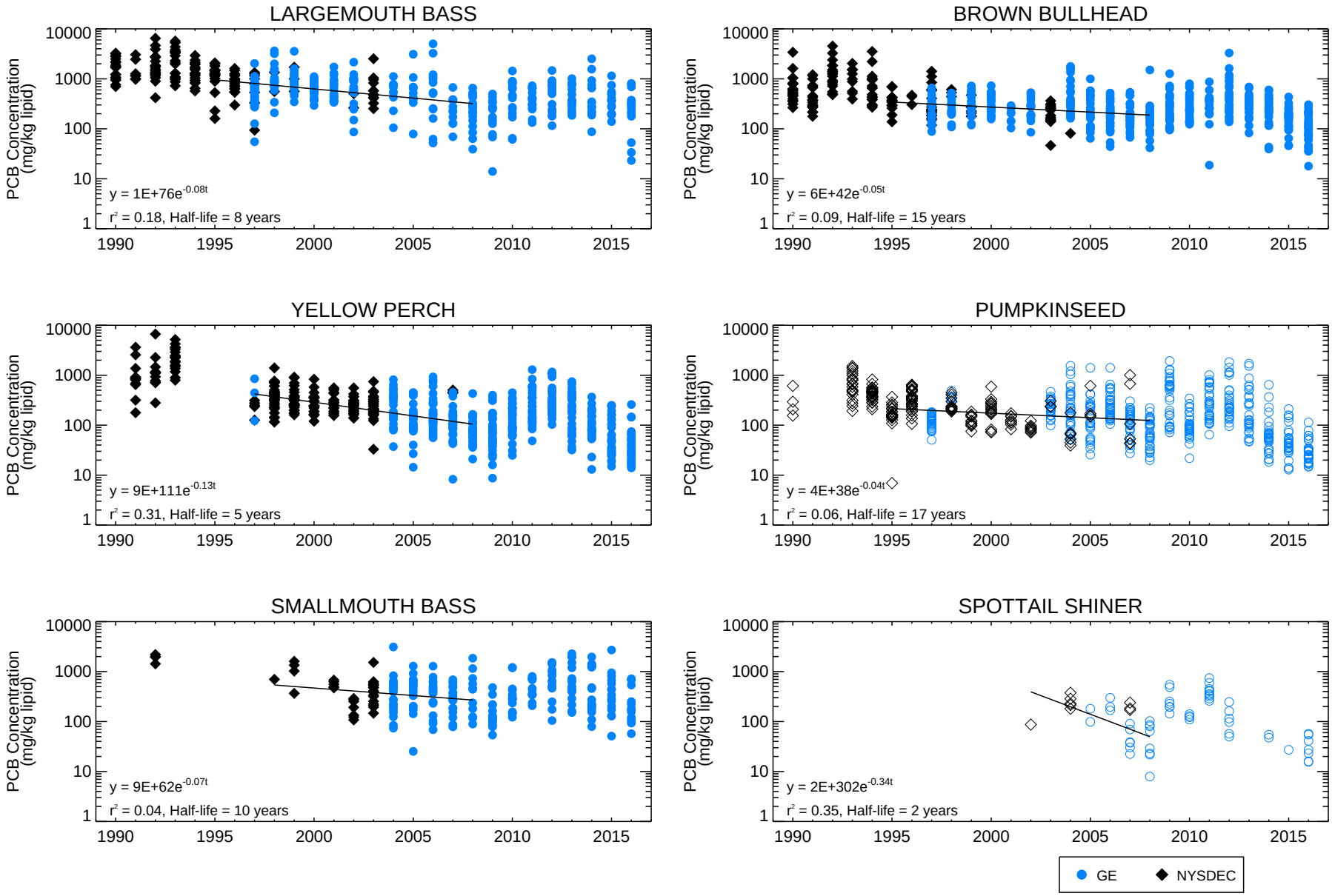

Figure 2d

Time Trends in Fish Total PCB Concentrations (Lipid-based) in Thompson Island Pool

NYSDEC Hudson River biota monitoring database (March, 2016) and GE RAMP database (March, 2017).
Fish data shown are from 1990 - 2016. Open symbols represent whole body and filled symbols represent fillet preparations.
Regression is based on 1995 - 2008.

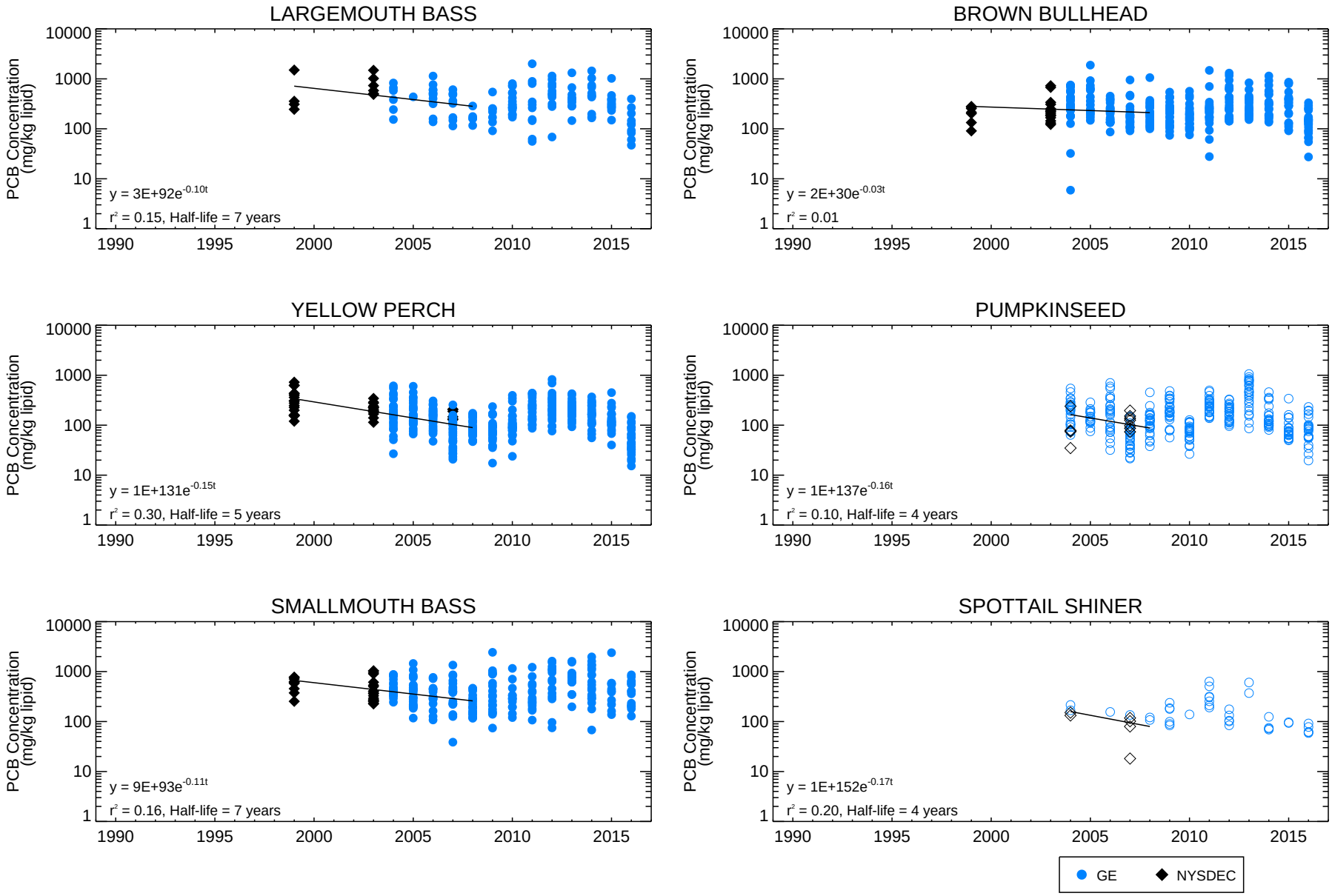

Figure 2e

Time Trends in Fish Total PCB Concentrations (Lipid-based) in Northumberland Pool/Fort Miller Pool

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Fish data shown are from 1990 - 2016. Open symbols represent whole body and filled symbols represent fillet preparations.
Regression is based on 1995 - 2008.

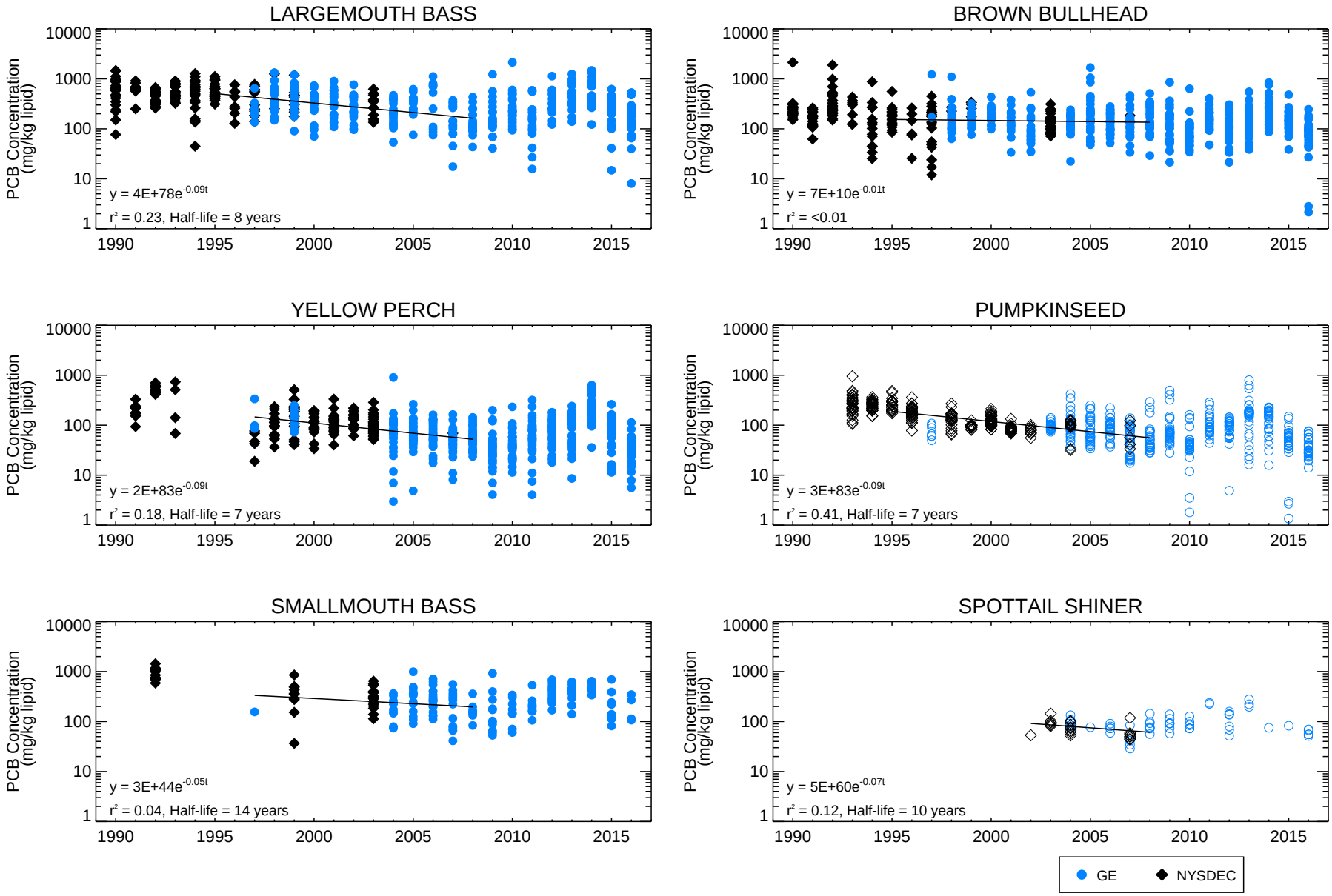

Figure 2f

Time Trends in Fish Total PCB Concentrations (Lipid-based) in Stillwater Pool

NYSDEC Hudson River biota monitoring database (March, 2016) and GE RAMP database (March, 2017).
Fish data shown are from 1990 - 2016. Open symbols represent whole body and filled symbols represent fillet preparations.
Regression is based on 1995 - 2008.

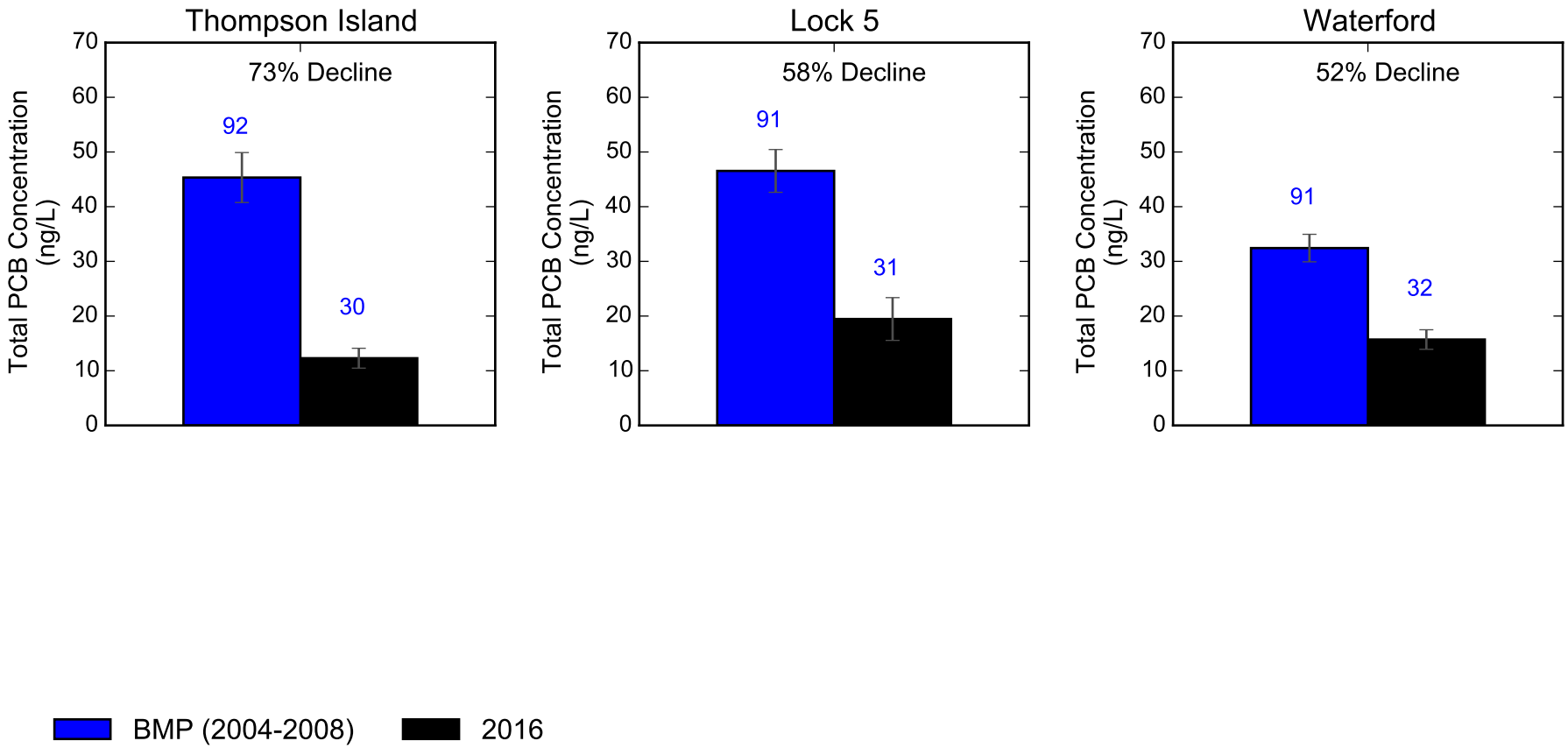

Figure 3a

Average Low-Flow Total PCB Concentrations during Baseline Monitoring and Post-dredge (2016) Years

Notes: Error bar represents +/- 2 standard errors. Non-detects set to 1/2 MDL. Duplicate data averaged.
Post-dredge and baseline data on this plot were collected from May through December where Fort Edward flow <= 5,000 cfs (for UHR stations).
Albany and Poughkeepsie stations are tidal and include all flow data.
Sample count posted above bars. Averages include Total PCB concentrations based on congener sums.
Data source: post-dredge: All_Water_Analyticals_20170411-1130.csv; baseline: Final_avg_BMP.csv.

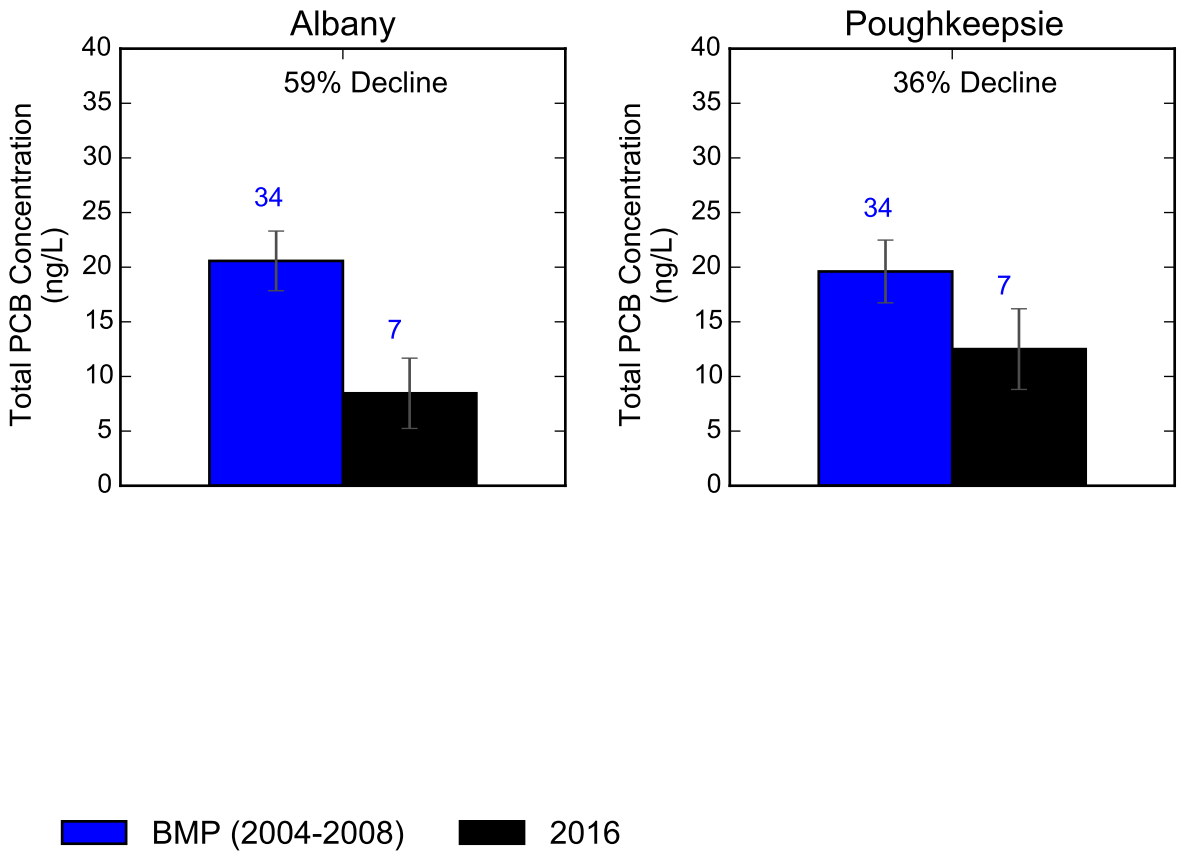

Figure 3b

Average Low-Flow Total PCB Concentrations during Baseline Monitoring and Post-dredge (2016) Years

Notes: Error bar represents +/- 2 standard errors. Non-detects set to 1/2 MDL. Duplicate data averaged.
Post-dredge and baseline data on this plot were collected from May through December where Fort Edward flow <= 5,000 cfs (for UHR stations).
Albany and Poughkeepsie stations are tidal and include all flow data.
Sample count posted above bars. Averages include Total PCB concentrations based on congener sums.
Data source: post-dredge: All_Water_Analyticals_20170411-1130.csv; baseline: Final_avg_BMP.csv.

Forage Fish

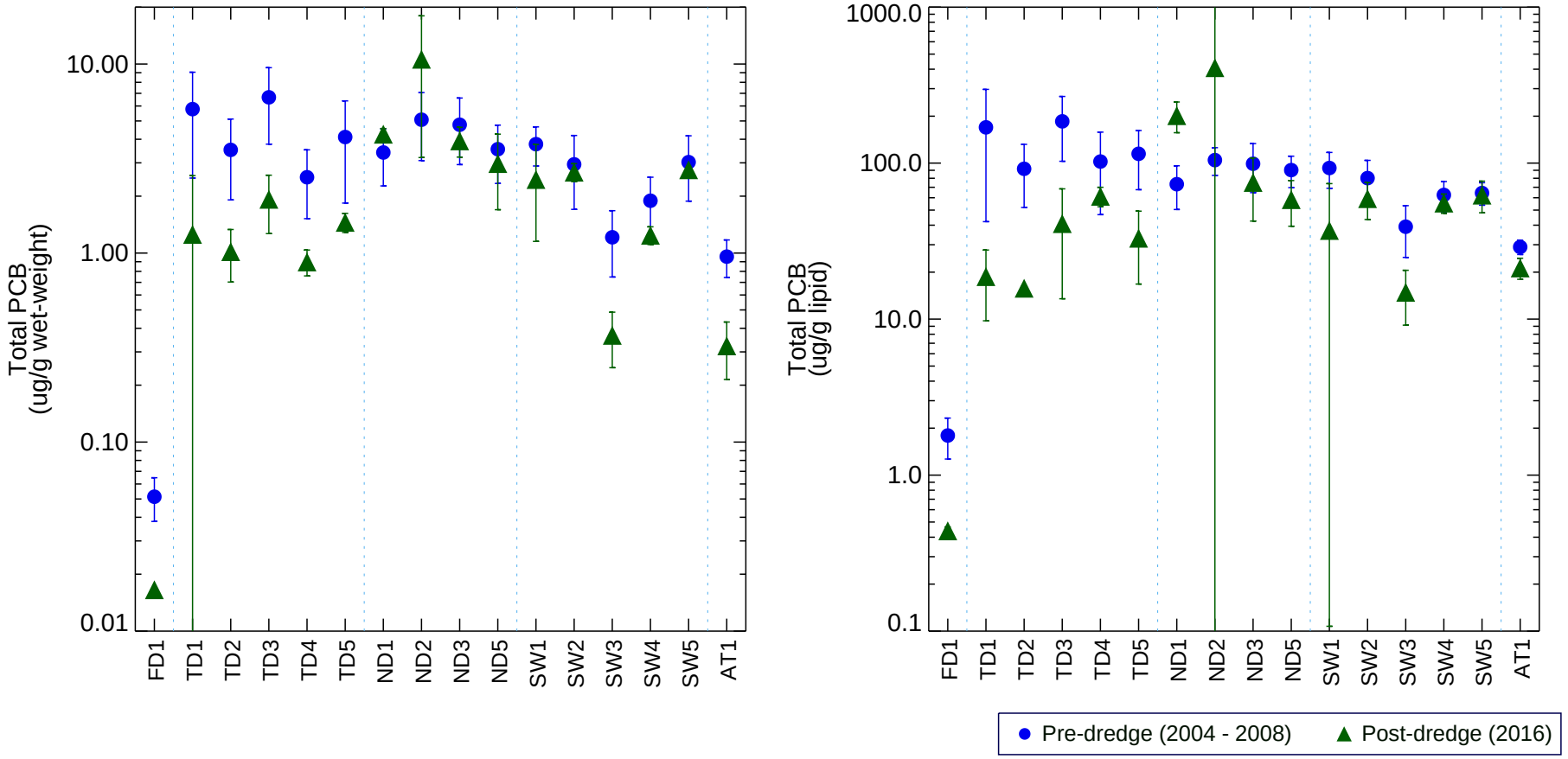

Figure 4

Spatial Trends in Forage Fish PCB Concentrations

BMP data = 2004-2008. RAMP data = 2009-2016.
Non-detects set to 1/2 method detection limit. Points are arithmetic means +/- 2 standard errors for pre-dredging (2004-2008) and post-dredging (2016) periods. Data are shown for whole body samples.
Forage fish = Fallfish, Blue gill, Tesselated Darter, various Dace, Minnow and Shiner species.

ATTACHMENT A

Attachment A

Critical Review of NOAA's Model Emulation Predicting Future PCB Fish Tissue Concentrations in the Lower Hudson River

Executive Summary

The National Oceanic and Atmospheric Administration (NOAA) and Kern Statistical Services developed an approach meant to predict future fish polychlorinated biphenyl (PCB) concentrations in the Lower Hudson River using equations developed with the results of the 2000 Feasibility Study (FS) model. Their work is presented in a paper entitled "Re-visiting projections of PCBs in Lower Hudson River fish using model emulation," published in the February 2016 issue of the journal *Science of the Total Environment*. The authors' approach used equations in which water column concentrations are calculated from sediment concentrations and other river characteristics. The water column concentrations were used to calculate fish concentrations using another equation. The authors developed this model because they claim that the sediment sampling that occurred after the 2002 Record of Decision (ROD) showed that the FS model predicted too great a recovery rate. The authors assert that based on the results of this new model (which they call a "model emulation"), the fish in the Lower Hudson River will recover post-dredging at a much slower rate than was predicted in the ROD.

The basis for the model emulation and its application are logically flawed, and as a result, the model calculates water column and fish PCB concentrations that are inconsistent with data from the Hudson River. The flaw lies in the following issues: 1) using a recovery rate that is based on spatially biased and highly variable datasets that cannot provide an accurate assessment of recovery; 2) basing the emulation model coefficients on the predictions of the FS model, which the authors claim is wrong; and 3) using the relationship between water column and sediment concentrations from the FS model as if the relationship was not dependent on concentration and would hold under altered sediment concentrations, which is incorrect. It is illogical to declare the original mechanistic model incorrect, but then use its results as the basis for developing the model emulation equations. The authors compounded these flaws by neglecting to test whether what the model predicts is sensitive to the uncertainty in its coefficients. As this attachment will show, different combinations of coefficient values that yield equally good fits to the FS model can produce widely varying fish tissue PCB concentration when the sediment PCB concentration is changed. The authors also neglected to validate the model by comparing its predictions to relevant available data—a crucial step in developing meaningful models. Model validation demonstrates that a model accurately replicates observed concentrations and provides confidence that the model can be used to predict concentrations. However, the authors did not present a validation of the NOAA model to observed

water and fish tissue Tri+ PCB concentrations.[1] As is shown in this document, when the NOAA model is compared to observed data, it becomes clear that the model does not replicate the data, and thus its predictions are not meaningful.

One of the model emulation scenarios predicts water column and fish PCB concentrations based on natural recovery starting in 2004. The baseline monitoring program on the Upper Hudson River provides pre-dredging data from 2004 to 2008, and allows for a comparison with the model emulation natural recovery scenario results. We reproduced the model emulation's predictions of water column and fish PCB concentrations and compared them to the measured PCBs in water and fish for the 2004 to 2008 period. These simple comparisons show that the model emulation overpredicts the water column and fish PCB concentrations. It is a clear indication that the model emulation does not accurately replicate the PCBs in the Upper or Lower Hudson River and cannot be used to predict the future recovery of the river. The developed and published model emulation is demonstratively inaccurate and gives misleading results as to the future condition of the Hudson River following dredging.

Overview

The following is a review of a paper authored by Jay Field of NOAA, John Kern of Kern Statistical Services, and Lisa Rosman of NOAA. The paper is entitled "Re-visiting projections of PCBs in Lower Hudson River fish using model emulation" (the NOAA paper) and was published in the February 2016 issue of the journal *Science of the Total Environment*. The paper is included as Exhibit A-1. The results from the NOAA model have been widely disseminated. They were presented to the Hudson River Foundation in May 2015 and have been used repeatedly by the Hudson River Trustees to call on the U.S. Environmental Protection Agency (EPA) to expand the remedy. As widely reported in the press, this work has also been used by many in the non-governmental organization community, New York officials, and elected officials as justification for urging the expansion of the dredging of the Hudson River.

As a result of the perceived significance of the NOAA paper to the understanding of the efficacy of the remedy and its public importance, more detailed information was requested in February 2016 through a Freedom of Information Act (FOIA) request to better understand this work and to attempt to reproduce the findings. This FOIA request had some urgency, because EPA was about to embark on a review of the remedy as part of its Five-Year Review of the selected remedy. Unfortunately, the material was not timely produced and only in May 2017, nearly 14 months after the initial review of the NOAA model, was enough material released to allow for a detailed review. We have done our best to assess how the authors developed their model and predicted fish tissue concentrations,

[1] Tri+ PCBs are PCBs with three or more chlorine atoms.

based on our knowledge and understanding of modeling, the Hudson River data, and the explanations provided in the paper.

Background

The NOAA paper details a regression model that the authors developed to predict PCB concentrations in Lower Hudson River fish after dredging is completed in the Upper Hudson River. The authors posit that this new model is needed because the mechanistic model used by EPA during the 2000 FS and the 2002 ROD overestimated the rate of natural recovery and consequently underestimated the time for fish in the Lower Hudson River to reach the targets presented in the ROD. In this attachment, we refer to this new model as the NOAA model (which they refer to as model emulation), and the EPA model used during the FS and the ROD as the FS model.

The NOAA model emulation is, in short, equations that predict water and fish tissue PCB concentration based on prescribed sediment PCB concentrations. A set of coefficients was determined by fitting the equations to the FS model concentrations. Using the fitted equations, the authors contend that they can predict water and fish tissue concentrations resulting from different assumptions about the rate of recovery of the sediments. By using different initial sediment concentrations and different assumed rates of sediment recovery in the model emulation, the authors attempt to calculate what they contend are better estimates of future fish tissue concentration than those provided by the FS model. However, the model is clearly not valid, and suffers from several fatal flaws.

Understanding the fatal flaws of NOAA's model emulation requires an explanation of how the model was developed. In simple terms, the authors based the model emulation on the results of the EPA model used in the Hudson River FS and did the following:

1. Using the EPA FS model output, they developed regression equations at different points along the river relating PCB levels in water to PCB levels in sediment.
2. Using the EPA FS model output, they developed regression equations relating PCB levels in fish to PCB levels in water.
3. Once the coefficients were established by regression, they adjusted the sediment PCB levels (i.e., increased them by approximately 2 times). However, they did not adjust any of the regression coefficients to account for this change.
4. To estimate PCB water levels into the future, they assumed that PCB levels in surface sediment would decline by 3% per year.
5. With the assumptions in numbers 3 and 4 (i.e., increased initial sediment concentration and lower sediment rate of recovery), they used the "revised" water-sediment regression from step 3 to predict PCB levels in water into the future.

The PCB levels predicted in water in the future were used with the equation developed in step 2 to predict PCB levels in fish.

This simple explanation exposes the model's flaws. Its predictions of recovery in fish are driven by the assumption that sediment PCB levels would decline at a rate of 3% per year based on an analysis of the sediment data. The historical sediment data do not provide a basis to assess rates of recovery because of differences in the nature and scope of the historical sampling programs and the inherent spatial variability of the data. The water and fish data, which are integrators of the sediment flux and upstream PCB loads, are better metrics to estimate rates of recovery. EPA's analyses of the data show higher PCB decline rates than estimated by NOAA, as presented in EPA's Proposed Five-Year Review Report (EPA 2017, pp. 5, 32, Appendix 3, Table A-3). The second flaw is calibrating a regression equation to predict water PCB levels from sediment PCB levels and then changing the sediment PCB levels without changing the regression coefficients. These coefficients reflect the relationship between sediment and water column PCB levels embodied in the FS model. Changing the sediment concentrations in the NOAA model forces changes in water column concentrations to values inconsistent with the measured water column concentrations to which the FS model was calibrated. Changing the sediment PCB levels without investigating whether the coefficients require further adjustment invalidates the model. However, even if the regression coefficients had been adjusted, NOAA's model emulation suffers from other significant flaws that make the results unrealistic. For example, NOAA established the regression equations using results from a model that they claim is inaccurate. Finally, because the regression coefficients they established have no interpretable meaning for processes on the river (regardless of what they may claim in their paper), the model emulation development is just a curve-fitting exercise, and the final equations cannot be used as a decision-making tool. Furthermore, their curve-fitting exercise is unconstrained, meaning there are multiple combinations of the regression coefficients that result in what NOAA would claim is an acceptable calibration, but yield significantly different predictions of future fish PCB levels. For all of these reasons, it is not possible to use NOAA's model emulation to predict future PCB levels in fish.

More detail on the NOAA model is provided as follows.

Assessment of Post-Dredge Surface Concentrations and Rates of Recovery

The NOAA paper focuses on the validity of the FS model developed by EPA as part of the remedy analysis, and specifically on the authors' assertion that the FS model under-predicted the pre- and post-dredge surface Tri+ PCB concentrations and over-predicted natural recovery. The authors propose the NOAA model as a means to obtain more accurate predictions of recovery without having to re-calibrate and re-run the FS model.

The authors contend that the FS model underpredicted the post-dredge average Tri+ PCB surface sediment concentrations by a range of 1.5 to 5 times, depending on the River Section. This contention is based on their comparison of the post-dredge surface sediment Tri+ PCB concentrations estimated in the FS to the average surface concentrations they estimate using the Sediment Sampling and Analysis Plan (SSAP) data in the non-dredged areas and backfill concentrations in the dredged areas.

The authors calculated a rate of recovery for the sediments of 8% per year from the FS model. They proposed a "true" rate of recovery of 3% per year, which they calculated from the 1991 O'Brien and Gere composite sampling data (O'Brien and Gere 1993) and the SSAP data collected from 2002 to approximately 2005 for the dredge design (QEA 2002) (the SSAP data were assumed by the authors to represent 2003 conditions).

The Authors' Estimated Historical Rate of Recovery is Highly Uncertain Because of Issues Matching the 1991 Data to the SSAP Data

Any estimate of trends derived from the 1991 and SSAP data would need to account for differences in the approaches to sampling. The SSAP data in the reaches below River Section 1 were collected in areas generally identified as fine sediment, based upon the side-scan sonar survey conducted in 2002 and 2003. Therefore, the SSAP sampling density is clustered in smaller pockets that are in and around dredge areas with higher PCB concentrations (see the example in Figure 1). Consequently, a straight average of the data (which is what the authors appear to have used to arrive at the numbers presented in the article) will be biased high relative to the true average of the non-dredge areas.

Additionally, the spatial coverage and availability of the 1991 and SSAP datasets are significantly different. The 1991 data are composite samples composed of between 3 and 17 discrete cores and do not have a representative sampling coverage when trying to assess River Section wide averages, whereas the SSAP data are all discrete sample points and tend to be more broadly located (although the SSAP coverage in many of the reaches below River Section 1 is still limited). The 1991 composites broadly cover each reach and each composite typically spans large distances, sometimes up to 2 miles. In contrast, the SSAP dataset has a more representative and broader coverage in many of the reaches. When looking at the non-dredge areas, only 45 out of the 132 composite samples taken in 1991 have at least two-thirds of the sample locations outside of dredge areas in the entire 40-mile stretch of the Upper Hudson River, whereas there are 4,350 SSAP cores in non-dredge areas available to calculate non-dredge surface sediment averages.[2] For these reasons, it is not appropriate

[2] The historical recovery rate in the river will be controlled by the change in the upstream loads and the recovery of all of the sediments. However, the remediation efforts at the plant sites, while ongoing, are generally believed to now be controlling the upstream loads. Also, after dredging, the areas that had the higher PCB concentrations have been replaced with backfill that has no PCBs. Therefore, after dredging is complete, the recovery rate of Upper Hudson River will be controlled only by the PCBs that are still remaining in the non-dredge areas. For these reasons, any assessment of post-dredging recovery rate should focus on the recovery observed in the non-dredge sediments.

to compare straight averages of the 1991 data to straight averages of the SSAP data to estimate historical rates of recovery.

The recent EPA Proposed Five-Year Review Report (EPA 2017) evaluates recovery rates between the available sediment datasets, as well as rates of recovery for the fish and water. EPA agreed with the conclusions noted above that the datasets are not directly comparable, as the spatial distribution and compositing schemes varied for each study. EPA developed recovery rates from water data (5% to 13% per year) and fish tissue data (typically 12% to 20% per year on a wet-weight basis, and on average, 8% per year on a lipid-normalized basis for the sport fish species). While EPA acknowledged the limitations of the sediment data, it used those data to provide rough estimates of temporal trends in the sediments. EPA's best estimates of recovery rates for cohesive and non-cohesive sediments for the period from 1976 to present ranged from 5% to 7% per year with uncertainty bounds of 3% to 10% per year using a "simple" exponential decay method which reduced potential biases by separating the data by sediment type and weighting sediment surveys by their reliability. Thus, EPA concludes that the decay rates are generally consistent with the 8% rate used during the development of the ROD. Further, the water and fish recovery rates, which have a higher level of certainty than those developed from the sediment data, are consistent with these sediment results. As such, the decay rate used by the authors of the NOAA paper is likely too low and not fully representative of the Hudson River sediments.

Model Development and Predictions

The NOAA model consists of regression equations developed to reproduce the FS model water column Tri+ PCB concentrations for each Hudson River section and for each year simulated by the FS model for Monitored Natural Attenuation (MNA) and the remedial scenarios. The first set of regression equations are a series of multiple linear equations in which the water column PCB concentration at the downstream boundary of each of 4 sections of the Upper Hudson River (the sections are the EPA-defined River Sections with River Section 3 divided in two) is calculated from two constants (River Section length and area of cohesive sediment) and two predictor variables: the water column PCB concentration at the upstream boundary of the section and the average surface sediment PCB concentration. The NOAA model-predicted water column PCB concentration in each section is then dependent on only the assumed sediment PCB concentration in each section, the PCB concentration supplied to the most upstream river section, and the coefficients in the regression equations. The sediment concentrations and water column concentrations used to determine the coefficients in the regression equations are those predicted by the FS model for two 30-year simulations of natural recovery and two 25-year simulations of the ROD remedy. The two simulations of each differ in the specified boundary condition at Fort Edward. Using predicted concentrations at

1-year intervals resulted in 440 simultaneous equations.[3] A least-squares optimization applied to these equations was used to establish the coefficients. The R-squared value reported for the relationships between the emulated water column concentrations and the FS model predicted water column concentrations was 0.98. The regression equation with the final coefficients are provided in Appendix A of the NOAA paper. Subsequent information received as part of the FOIA requests included the other independent variables necessary for us to replicate their results of model emulated water column concentrations.

The second equation used in the paper is a linear relationship between water column PCB concentrations at Waterford (independent variable) and fish tissue PCB concentrations (dependent variable) at Hudson River Mile (RM) 152. Equations were developed for four species of fish (largemouth bass [*Micropterus salmoides*], brown bullhead [*Ameiurus nebulosus*], white perch [*Morone americana*], and yellow perch [*Perca flavescens*]) using the FS model results. The R-squared values for these linear relationships were greater than 0.9 for all four species. The equations used to predict fish tissue concentrations are provided in the supplemental information with the paper.

After developing the equations and fitting the necessary coefficients, the authors estimated future fish PCB concentrations as follows: They set initial sediment concentrations in the water column/sediment regression model to values they derived from the remedial design data (i.e., the SSAP data that they use to represent 2003). They then decremented the sediment concentrations at 3% per year and calculated a 30-year time series of water column PCB concentrations (2005 through 2034). These water column concentrations were then used in the linear regression model to predict PCB fish tissue concentrations at RM 152. The results presented indicate that all four fish species modeled would take more than 25 years to reach the 0.2 milligrams per kilogram risk-based threshold used in the FS, even after the full dredging program had been implemented.

Through the FOIA, the authors provided the information necessary to recreate six figures from the paper and three figures from the supplementary information for the paper. The regression coefficients, other independent variables, and coded equations needed to run the NOAA model were also included in the FOIA information. However, additional information and the code used to develop these regression coefficients from the FS model output was not provided. The provided information allowed us to run the NOAA model and determine the water column Tri+ PCB concentrations and white perch Tri+ PCB concentrations predicted by the model emulation outside of the years highlighted in the paper. Using the equations from the paper coded by the authors, reproduction of manuscript figures indicates inconsistencies between values provided in the FOIA responses to reproduce the figures and values in the tables of the published paper, demonstrating that there are also inconsistencies between the figures in the paper and the values in the tables of

[3] 2 boundary conditions * 25 years * 4 river sections + 2 boundary conditions * 30 years * 4 river sections = 440 equations

the paper that were supposedly used to generate the figures. While the provided code allowed us to eventually reproduce the model emulation results after some effort, the inconsistencies between the information reported in the published paper and the FOIA documents indicate relatively poor documentation of the model development and approach in NOAA's files.

The Authors Based Their Model Emulation on a Modeling Framework that They Claim is Flawed

If we have correctly interpreted the authors' approach, they criticized the FS model but used its relationships between sediment and water column PCB concentrations to formulate their model. It seems illogical to use presumed incorrect model results to establish the transport of PCBs between the water column and the sediment. The FS model predictions called into question are the result of its rates of PCB transport. If the predictions are incorrect, it would have to be because the rates of PCB transport are incorrect. Using these rates cannot address the supposed fundamental problem of the FS model. The NOAA model suffers from this same problem. Inputting an alternative sediment time trend results in the model incorrectly predicting water column concentrations.

The Interdependence of the Model Coefficients Hinders Any Conclusions Regarding the Relative Importance of the Non-dredge Sediment as a Source of PCBs to the River

The equations presented in Appendix A of the NOAA paper indicate that 12 model coefficients (3 per river section times 4 sections) were set by fitting the NOAA model-predicted concentrations to the FS model-predicted concentrations. Given the explanation of these coefficients, they are interdependent. Therefore, it is likely that multiple sets of these coefficients would have resulted in acceptable fit to the FS model-predicted concentrations. We set up a test using the MNA case to determine whether different values for the coefficients could produce equally reasonable fits to the FS model-predicted concentrations and yet produce different results when the assumed rate of sediment recovery is changed from 8% to 3% per year. For this test, we varied the NOAA model coefficients by up to a factor of 5. The test showed that the values of the coefficients could range by at least a factor of 5, and the NOAA model would still fit the FS model water and fish tissue concentrations nearly as well as the coefficients used in the paper (Figure 2). However, when the natural recovery of the sediments is changed to the 3% per year rate that the authors believe is correct, the NOAA model predicts widely different Tri+ PCB concentrations in the fish tissue between the different sets of coefficients (Figure 3). This indicates that, even though the NOAA model fits the FS model-predicted concentrations, the NOAA model is unsuitable for predicting the Tri+ PCB concentrations that result from changes to the model assumptions, such as changing the natural rate of recovery of the sediments. In this case, it is difficult to reach any meaningful conclusion about the relative impact of the sediments on the recovery of the fish tissue PCB concentrations using the NOAA model emulation.

The Analysis Did Not Use Available Water and Fish Data to Validate the Model Predictions

Validation of model results is an integral step in developing models. Ideally, one set of data will be used for model calibration (in this case fitting the model emulation coefficients), and a separate set of data will be used to validate the model predictions and better understand the accuracy of the model. For example, NOAA operational models must undergo rigorous model validation before being released for public use.[4] The NOAA model emulation was calibrated to the FS model-predicted PCB concentrations, but, based on the published information, was not compared to the measured water and fish concentrations to validate that the model accurately predicted the observed water and fish tissue PCB concentrations.

A test of the NOAA model's ability to predict PCB concentrations is to determine how well the model performs against observed data. On this account, it is clear that the NOAA model fails. Its predictions of the water and fish PCB concentrations are at variance with measured levels. The information provided in the NOAA paper along with the code obtained through FOIA requests allow for the comparison of the model emulated water column Tri+ PCB concentrations to the data collected during the Baseline Monitoring Program.[5] These comparisons indicate an obvious disconnect between the NOAA model concentrations and what was measured during the baseline program, with the NOAA model predicting concentrations that were 3 to 4 times higher than what was measured in the river (Figure 4). This result indicates a clear error in the predictions of the model emulation, because the concentrations predicted are far above those measured in the same time period.

The same observations are true for the fish. A comparison of the model-emulated wet-weight fish tissue concentrations with the baseline data representing natural recovery for each baseline year is presented in Figure 5. As with the annual comparison of the water concentrations, Figure 5 clearly indicates the inconsistencies between the NOAA model predictions and the PCB fish tissue concentrations measured during the baseline program; concentrations predicted by the model for white perch at RM 192 are 2 to 6 times higher than the annual average concentrations of the measured data. This contrasts with the model-data comparison in the paper; the authors provide in Figure 10 what appears to be a reasonable comparison between fish PCB data and the model predictions. However, the fish PCB wet-weight-based data have been adjusted upward by

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[4] An example of NOAA model validation for tsunami modeling is http://nctr.pmel.noaa.gov/benchmark/SP_3053.pdf, for wind-wave modeling is http://polar.ncep.noaa.gov/mmab/papers/tn281/multi_hindanalysis.pdf, and for water level modeling is https://www.nauticalcharts.noaa.gov/csdl/publications/TR_NOS-CS29_FY11_04_Lyon_CBFOS2.pdf.

[5] Because dredging did not begin until 2009, the data collected from 2004 to 2008 can be viewed as representative of a 5-year natural recovery (i.e., MNA) period. The model emulation theoretically begins in 2004, 1 year after the year used to represent the SSAP sediment concentrations in the model emulation. Therefore, the first 5 years of the model emulation for MNA represent the Baseline Monitoring Program.

normalizing to a 3% lipid content. The authors maintain that this adjustment was applied for consistency with the white perch lipid content that was used in the EPA FISHRAND model. However, while the 3% lipid content used by EPA for the FS model may have been representative of the historical white perch data, the average lipid content of the white perch collected from RM 152 from 1997 to 2014 (range shown on the authors' Figure 10) is 1.6%. Adjusting the measured wet-weight concentrations upward as if the fish had about twice the lipid they actually had increases them by approximately a factor of 2. Without this adjustment, it is apparent that the model is consistently biased high (Figure 6).

The authors contend that changes in the fish processing protocol between 2004 and 2013 may have resulted in underestimated PCB concentrations.[6] However, if this were the case, pre-2003 and post-2013 concentrations should show a marked departure from the trend seen during this period. Instead, the trend continues during these periods even with the perturbations introduced by the dredging program. Further, the special study that evaluated the impacts of this protocol change concluded that its impact was minor, and would not have resulted in the greater-than-a-factor-of-2 increase suggested by the authors.[7]

The more recent water column and fish concentrations predicted by the NOAA model are also at odds with measured values. The white perch Tri+ PCB concentrations in the first year after remediation was assumed to end (2010), based on their model emulation, are approximately 1.5 and 2.2 parts per million (ppm) for the remedy and MNA, respectively. These values far exceed even pre-dredge white perch total PCB concentrations at Albany; the average of 2008 and 2009 data is 0.7 ppm. They are also higher than the average value during dredging (1.0 ppm; 2010 to 2014; Figure 7). In fact, using the linear relationship that the authors present in the supplemental Table S-1, the water column concentration at RM 152 that results in a 1.5 ppm Tri+ PCB average for white perch is approximately 30 nanograms per liter (ng/L). This is 8 ng/L higher than the average baseline Tri+ PCB water concentration observed at Waterford before the dredging began (23 ng/L for the 2004 to 2008 baseline monitoring period). These results are nonsensical. While resuspension during dredging does increase water column PCB concentrations, that phenomenon is short-lived and post-dredging concentrations will be lower than pre-dredging.

The model is also unreliable because its predicted rate of decline on Lower Hudson River fish PCB concentrations is much lower than has been occurring for the last 15 plus years. As stated above, the model emulation shown on Figure 10 matches the data only because the authors have applied a flawed adjustment to the measured fish PCB concentrations. The MNA scenario should match the

[6] The authors suggest that the protocol change would increase white perch PCB concentrations beyond that resulting from the 3% lipid adjustment.

[7] The results of this special study were reported in Section 4.6.5 of Anchor QEA (2015).

pre-dredge rates of decline in fish; however, pre-dredge PCB concentrations in white perch collected from Troy show rates of decline of approximately 15% per year (Figure 8)[8]; five times that estimated from the model emulation. Rates of decline in PCB concentrations in bass, bullhead, and yellow perch collected from this location are similar to that measured for white perch. Further, these rates of decline are seen in fish collected from Catskill and Poughkeepsie to the George Washington Bridge. Thus, the weight of evidence suggests that Lower Hudson River fish PCB concentrations are declining at rates faster than the NOAA model emulation, even without the dredge benefit.

Summary

The model emulation developed by the authors of the NOAA paper is significantly flawed. It fails to adequately replicate the observed data. The authors' disregard of the available data for fish and water to estimate post-dredging concentrations is unjustified and results in future predictions for fish concentrations that are not defensible. The model emulation relies on the comparison of non-comparable datasets (1991 and SSAP) to establish a rate of recovery. This rate of recovery results in predicted sediment PCB concentrations that are too high, and correspondingly high predicted water and fish tissue concentrations provide strong evidence that the rate is too low. The authors stated that the model emulation coefficients were fit by minimizing the difference between the model emulation water concentrations and the FS model concentrations. However, the authors did not provide any information on how sensitive the fish tissue concentrations were to the specific chosen set of coefficients. Our analysis using their model emulation showed that a wide range of coefficients can produce equally suitable fits to the FS model and yet result in widely varying fish tissue concentrations when the assumed rate of sediment recovery is changed, indicating that the predicted fish tissue concentrations are unreliable when the sediment concentrations are changed. In summary, the failed model validation, incorrect sediment rate of recovery, and excessive sensitivity of the predicted fish tissue concentration to the specific chosen model coefficients show that the model emulation is not suitable for predicting future PCB concentrations and that the conclusions from the paper are misleading and unreliable.

References

Anchor QEA, 2015. *2014 Data Summary Report*. Prepared for General Electric Company, March 2015.

EPA (U.S. Environmental Protection Agency), 2017. *Proposed Second Five-Year Review Report for Hudson River Superfund Site*. Prepared by EPA Region 2, New York, NY, May 31, 2017.

[8] As discussed earlier in this attachment, assessing possible post-dredging recovery rates using historical data is problematic because of the source control efforts at the Hudson Falls and Fort Edward plant sites that occurred in the 1990s and 2000s. The 15% cited in this discussion attempts to alleviate that issue to some extent by assessing the recovery from 1999 to 2009, which is after many of the source control efforts at the plants had been implemented.

O'Brien and Gere Engineers, Inc., 1993. 1991 *Sediment Sampling and Analysis Program*. Prepared for General Electric Company Corporate Environmental Programs, Albany, New York.

QEA (Quantitative Environmental Analysis, LLC), 2002. *Design Support Sediment Sampling and Analysis Program, Field Sampling Plan*. Prepared for the General Electric Company.

Figures

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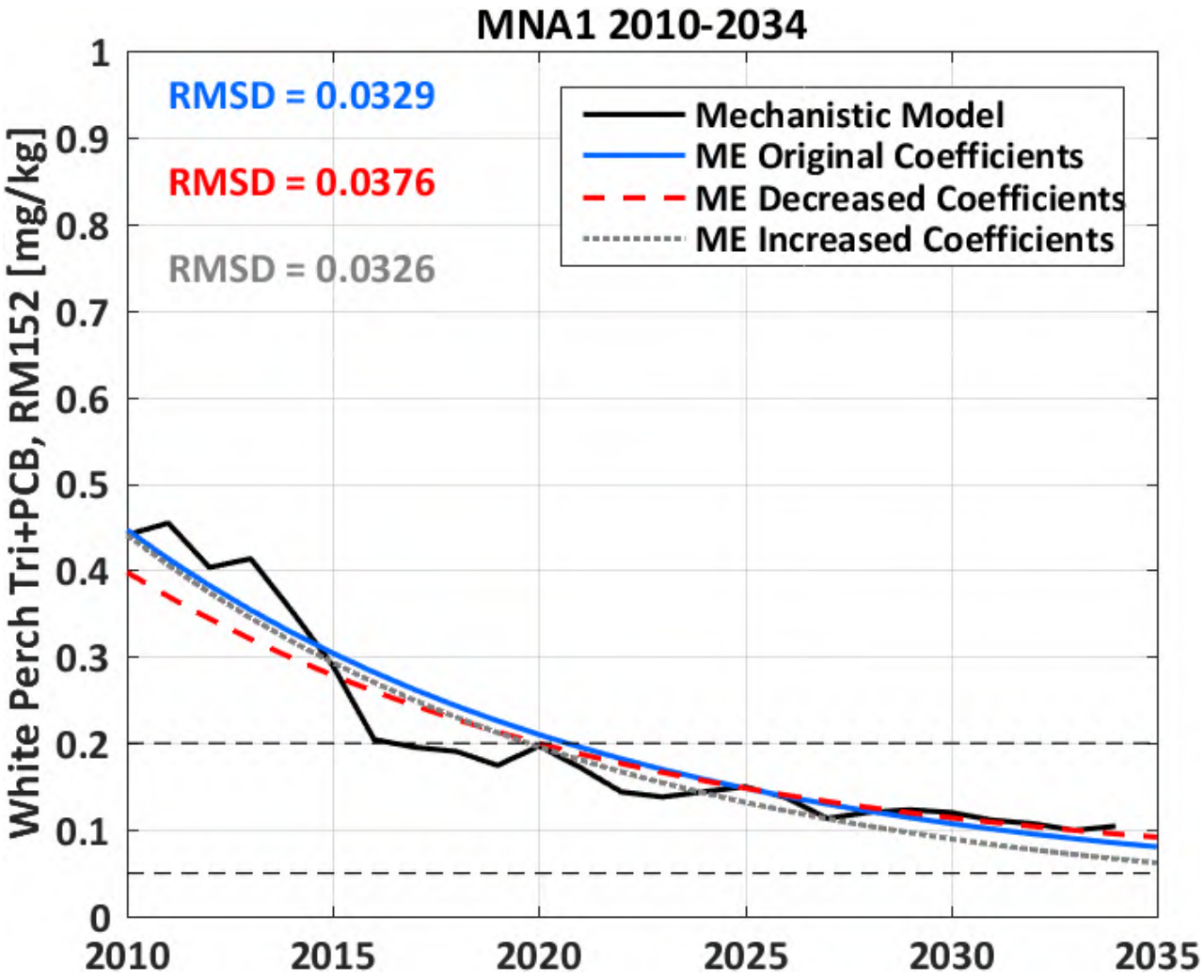
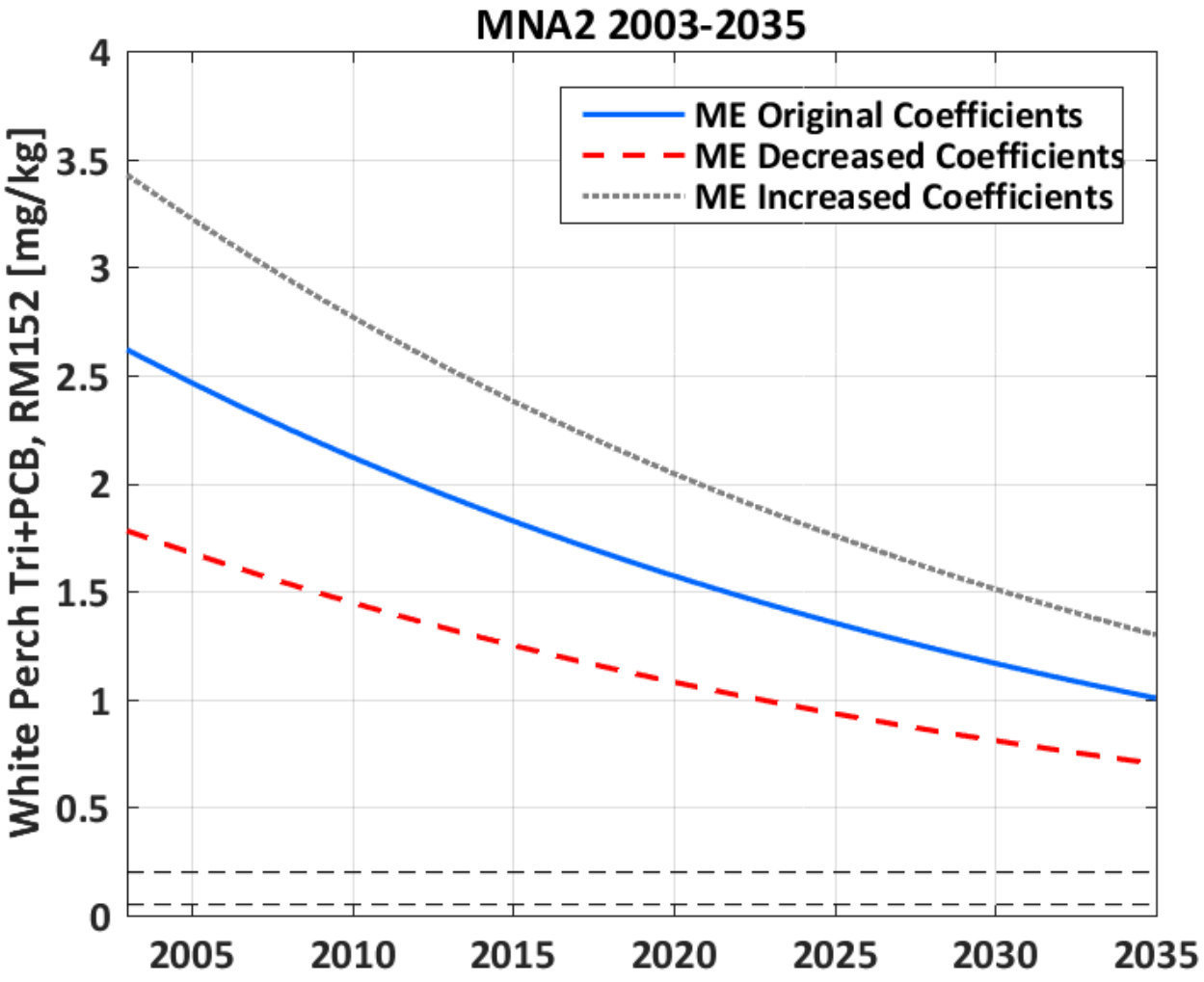
ANCHOR QEA

Figure 1
Example of Spatial Distribution of Upper Hudson River Sediment Sampling Programs
Review of NOAA Model Emulation
Hudson River PCBs Superfund Site

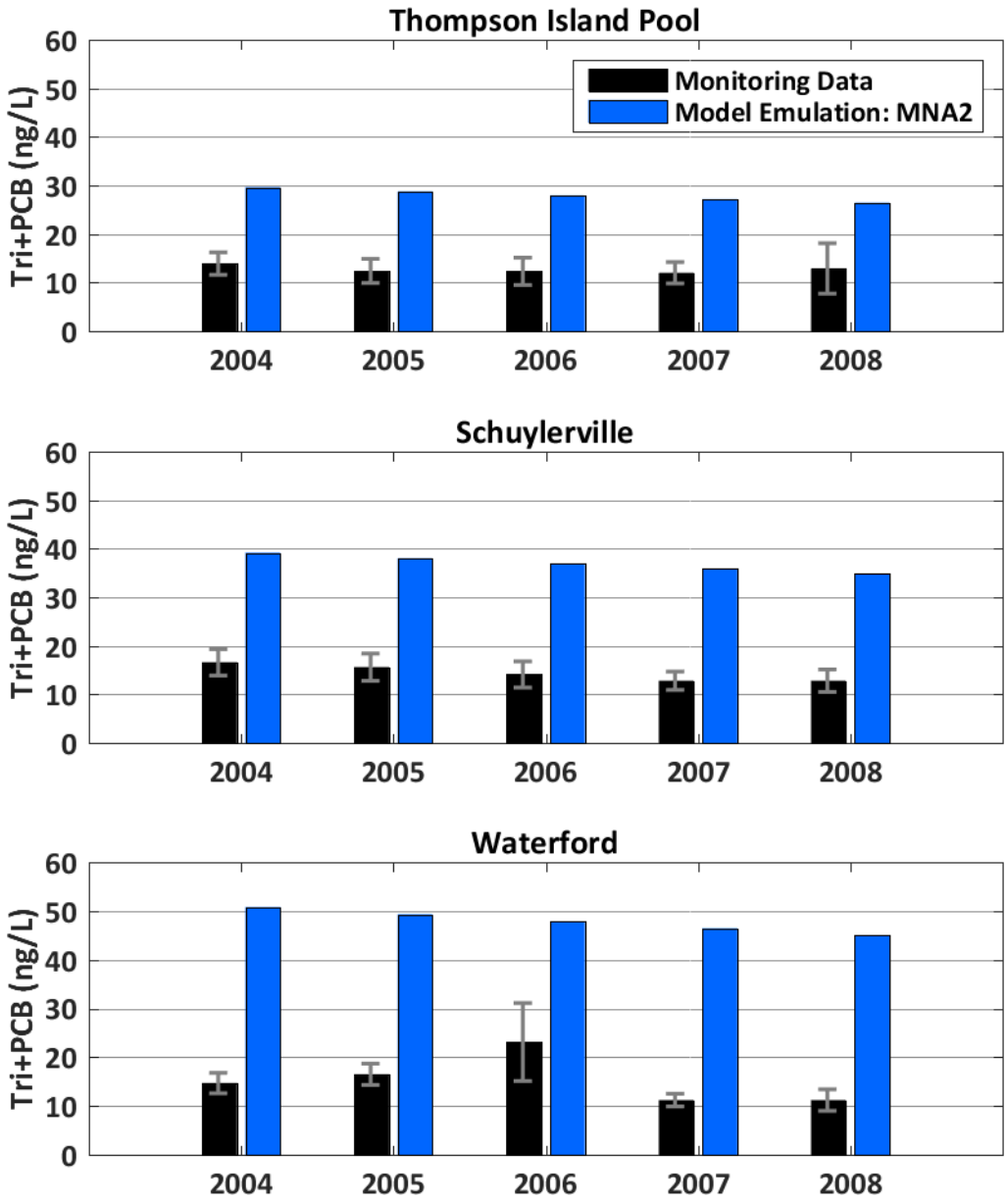


MNA1 assumes a natural rate of recovery of the sediments of 8% per year. RMSD is the root mean square difference.

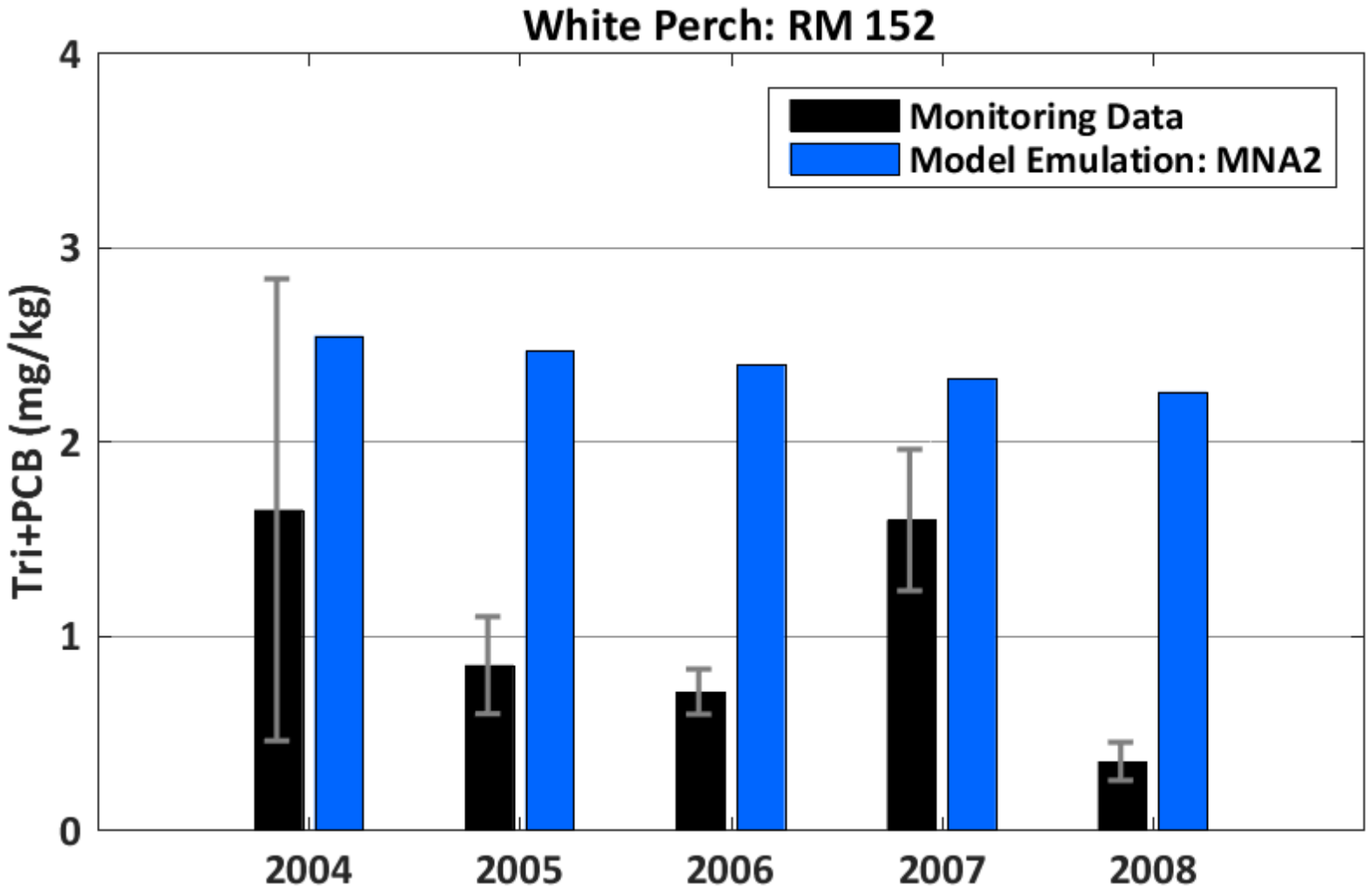
Figure 2
White Perch Tri+ PCB Concentrations (Wet-weight) from the FS (Mechanistic) Model and from the Model Emulation (ME)



MNA2 assumes a natural rate of recovery of the sediments of 3% per year.

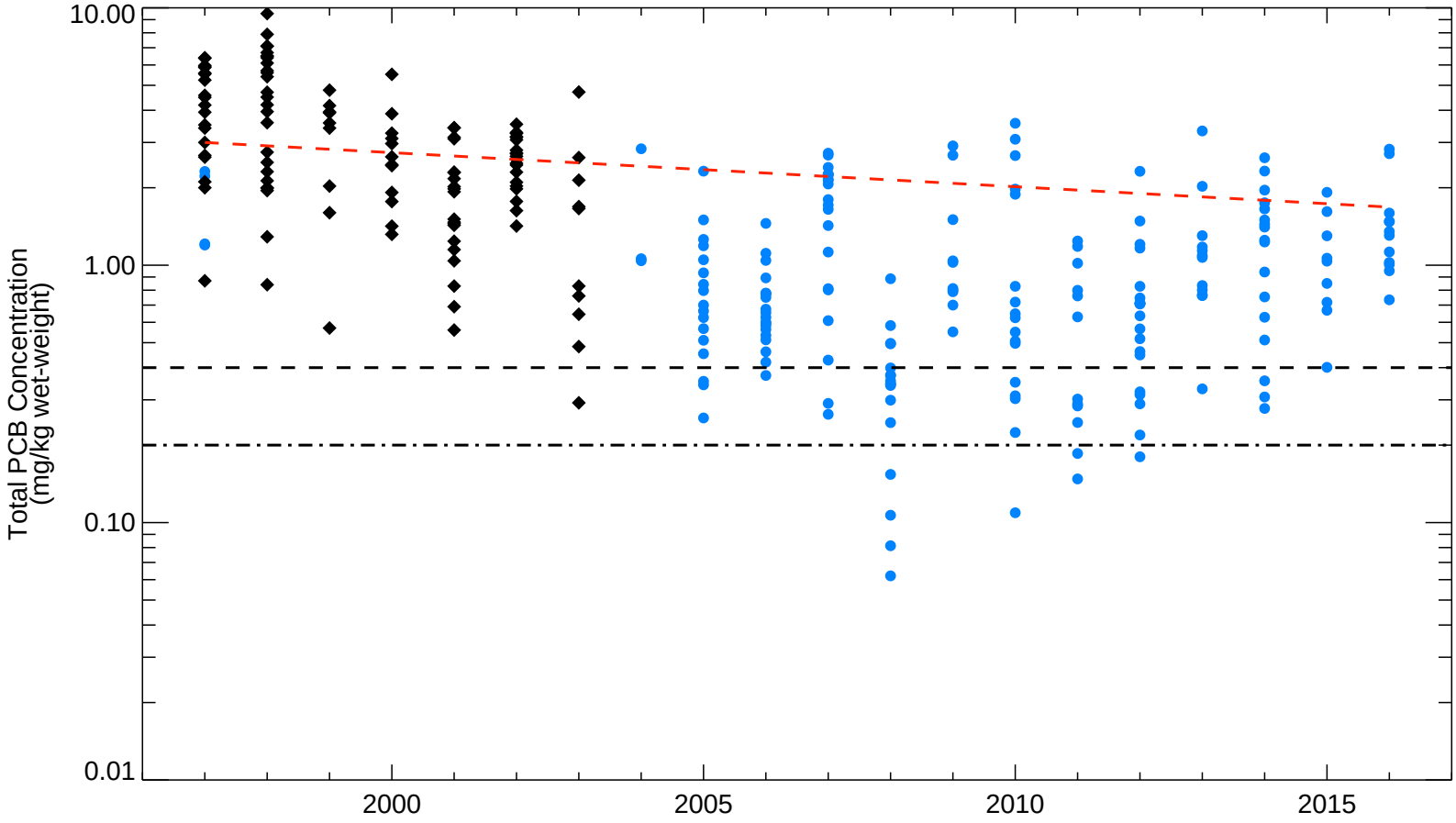


Monitoring data is the arithmetic mean +/- 2 standard errors. MNA2 assumes a natural rate of recovery of the sediments of 3% per year.



Monitoring data is the arithmetic mean +/- 2 standard errors. MNA2 assumes a natural rate of recovery of the sediments of 3% per year.

Figure 5
White Perch Tri+ PCB Concentrations (Wet-weight) from Albany/Troy
Review of NOAA Emulation
Hudson River PCBs Superfund Site



Data Sources: NYSDEC Hudson River Biota Monitoring database, 2010; GE BMP and RAMP database, 2017.
Only standard fillet preparation plotted. Model Emulation shown as red line is a 3% decline starting at 3 ppm.

● GE Data ◆ NYSDEC Data – – Model Emulation – – 0.4 mg/kg Threshold –·–· 0.2 mg/kg Threshold

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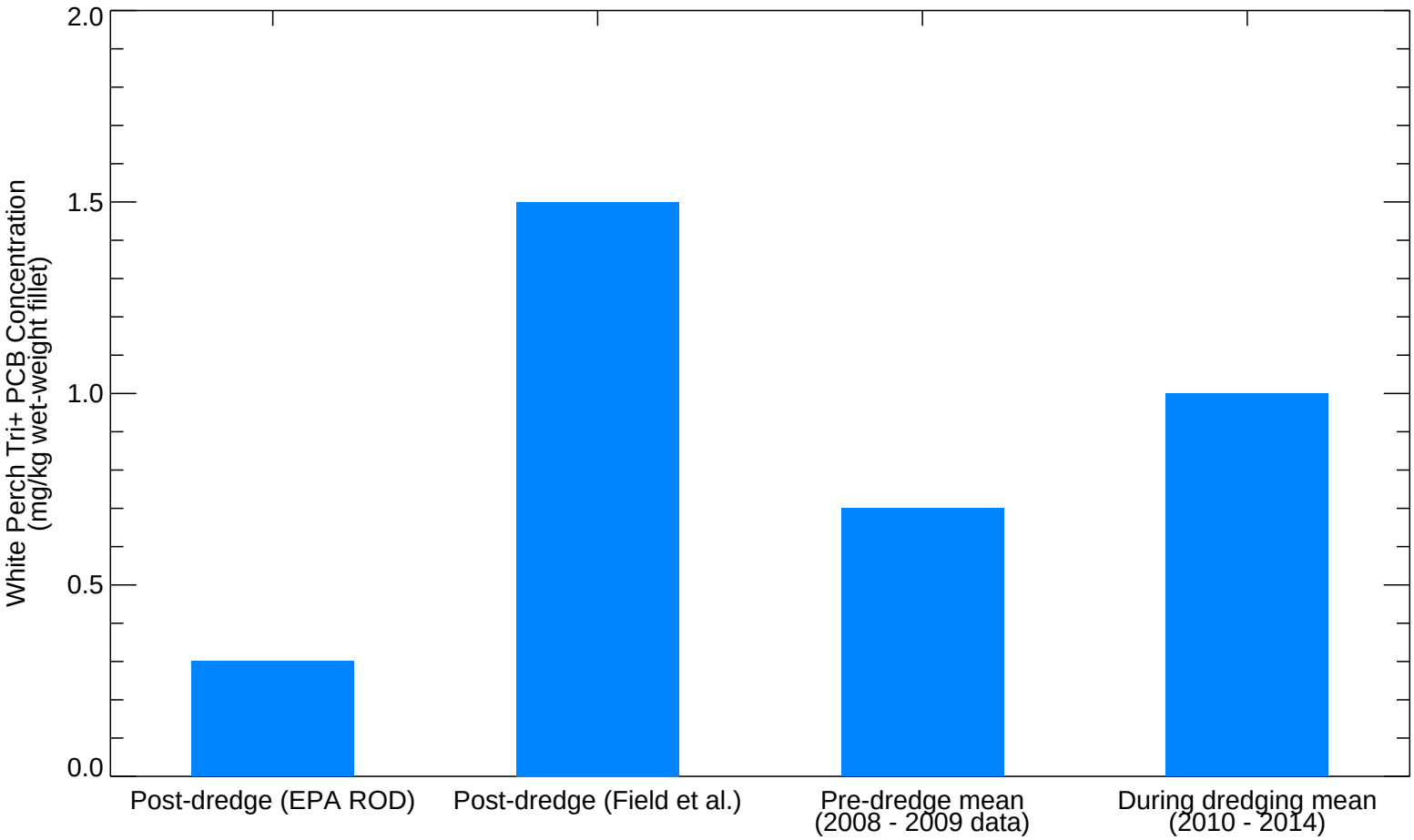
Figure 6

White Perch Total PCB Concentrations (Wet-weight) at RM 152

Review of NOAA Emulation
Hudson River PCBs Superfund Site



Data Sources: GE BMP and RAMP database, 2017.

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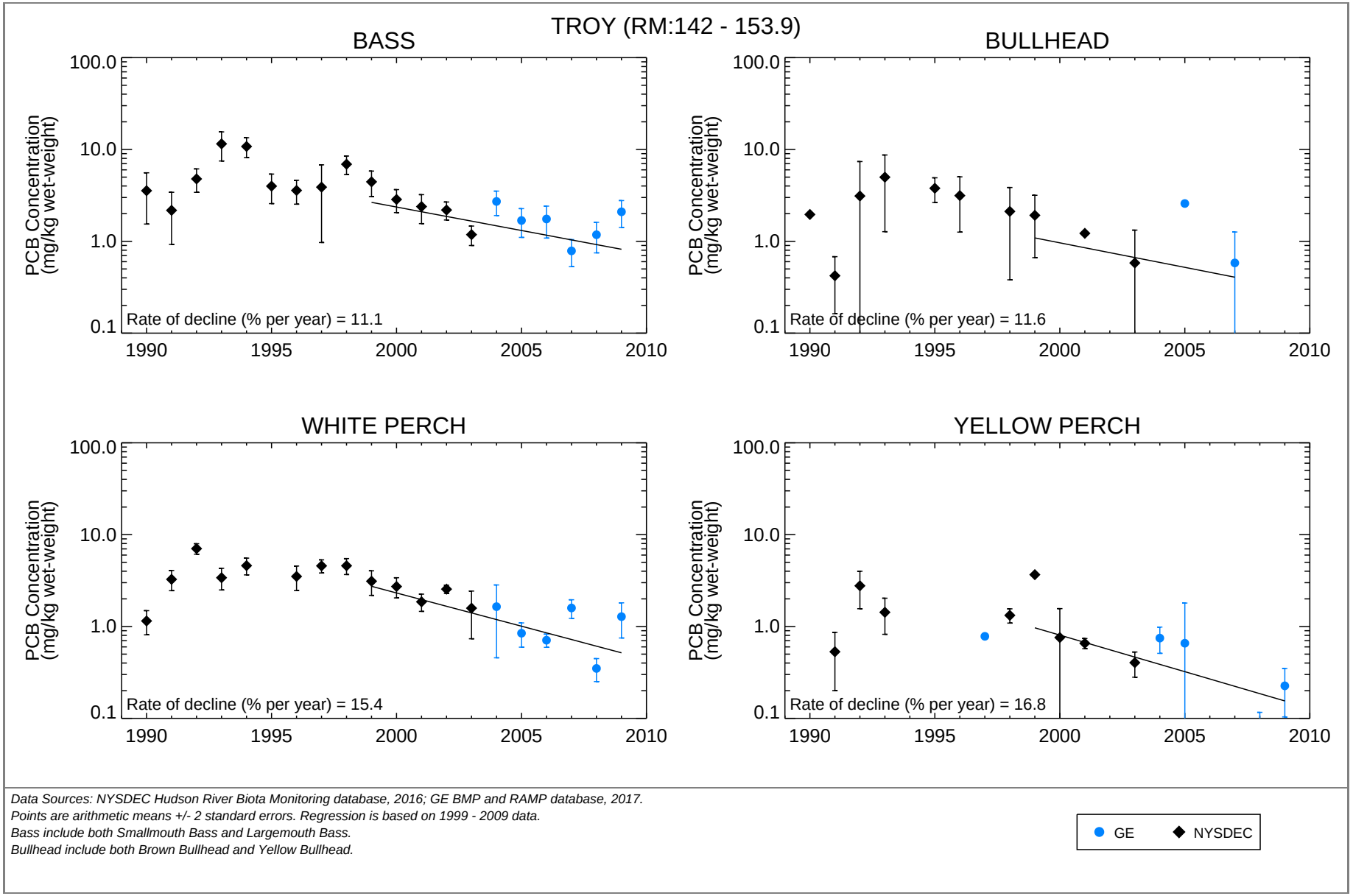
Figure 7

Comparison of Initial Post-Remedy White Perch Concentrations in the FS Model and Estimated by Field et al., with Average Pre-Dredge and During Dredging Concentrations in White Perch Collected from Albany/Troy



Data Sources: NYSDEC Hudson River Biota Monitoring database, 2016; GE BMP and RAMP database, 2017.
Points are arithmetic means +/- 2 standard errors. Regression is based on 1999 - 2009 data.
Bass include both Smallmouth Bass and Largemouth Bass.
Bullhead include both Brown Bullhead and Yellow Bullhead.

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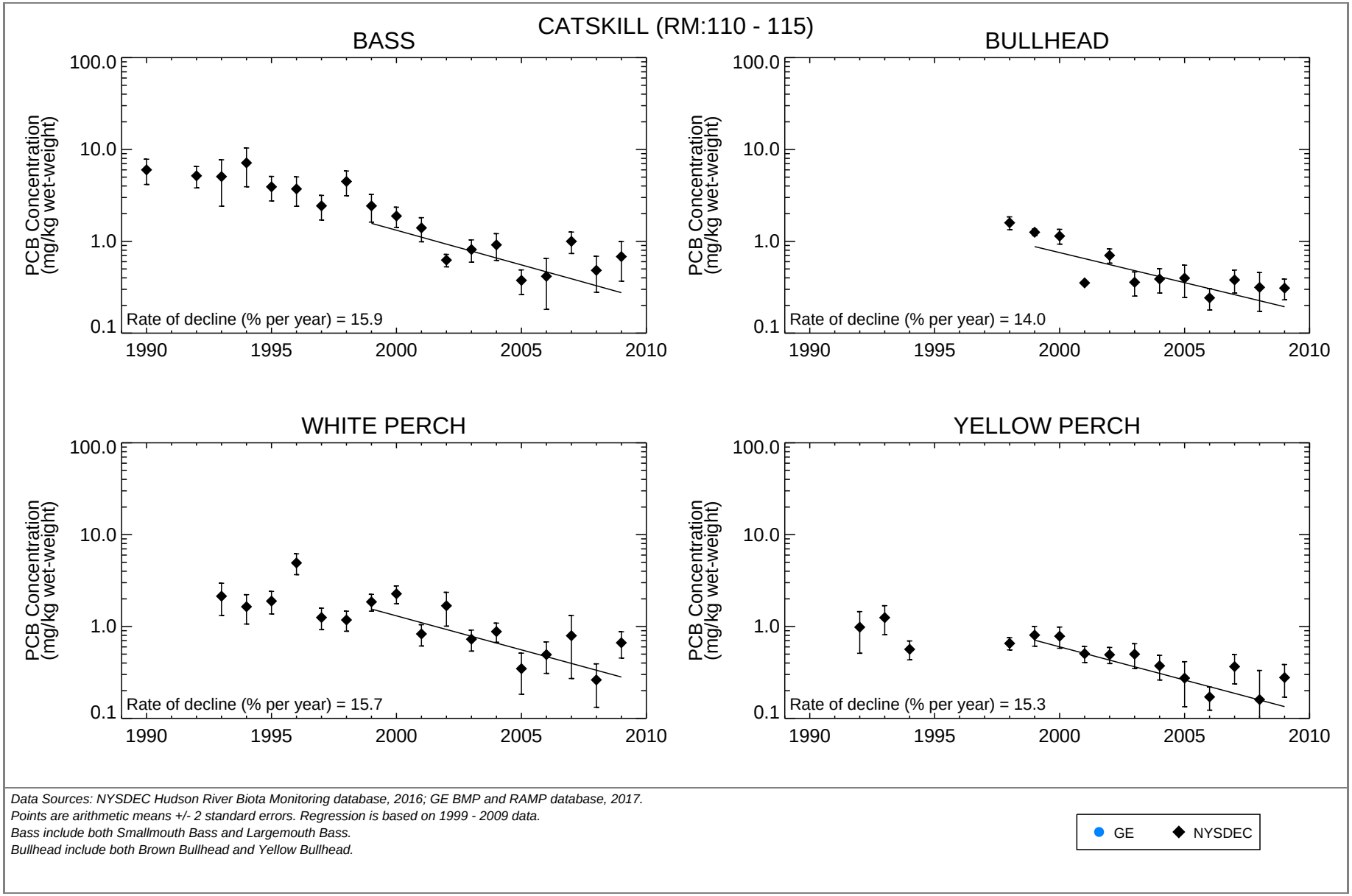
Figure 8a

Average Fish PCB Concentrations (Wet-weight-based) in Lower Hudson River

Review of NOAA Emulation
Hudson River PCBs Superfund Site



Data Sources: NYSDEC Hudson River Biota Monitoring database, 2016; GE BMP and RAMP database, 2017.
Points are arithmetic means +/- 2 standard errors. Regression is based on 1999 - 2009 data.
Bass include both Smallmouth Bass and Largemouth Bass.
Bullhead include both Brown Bullhead and Yellow Bullhead.

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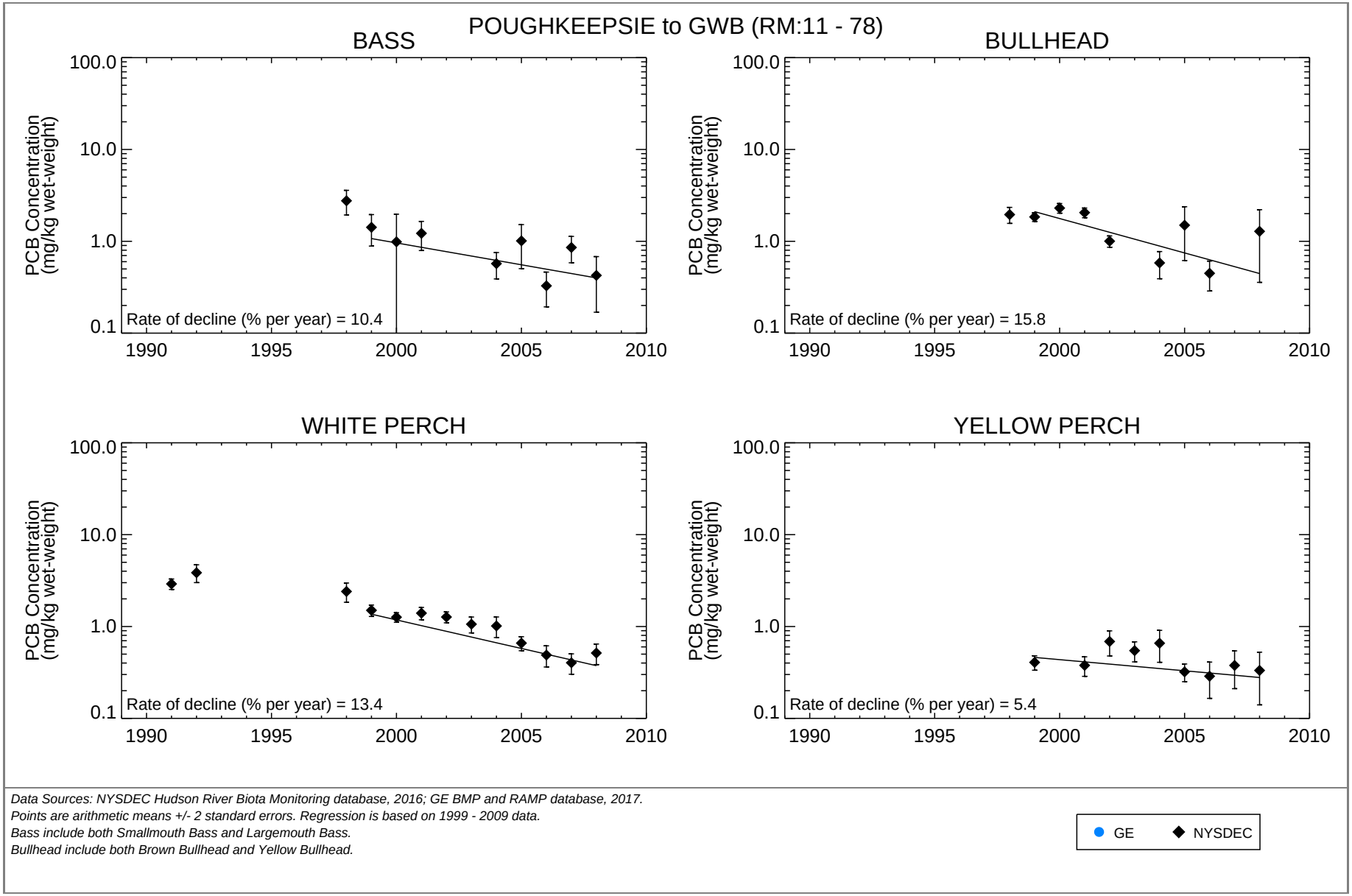
Figure 8b
Average Fish PCB Concentrations (Wet-weight-based) in Lower Hudson River

Review of NOAA Emulation
Hudson River PCBs Superfund Site



Data Sources: NYSDEC Hudson River Biota Monitoring database, 2016; GE BMP and RAMP database, 2017.
Points are arithmetic means +/- 2 standard errors. Regression is based on 1999 - 2009 data.
Bass include both Smallmouth Bass and Largemouth Bass.
Bullhead include both Brown Bullhead and Yellow Bullhead.

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File Path: \\iris\woodcliff\Projects\GE_General\Analysis\NOAA_Eval\Biota\Fish\LHR_fish_group_temporals_memo.pro

Figure 8c

Average Fish PCB Concentrations (Wet-weight-based) in Lower Hudson River

Review of NOAA Emulation
Hudson River PCBs Superfund Site

Exhibit A-1
Paper

Science of the Total Environment 557–558 (2016) 489–501

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Re-visiting projections of PCBs in Lower Hudson River fish using model emulation

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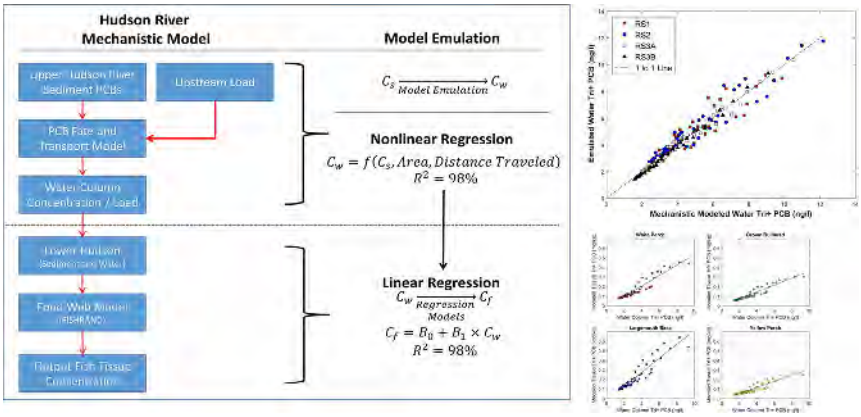
HIGHLIGHTS

- We emulated mechanistic model projections of fish PCBs in the lower Hudson River.
- Emulated models used updated sediment PCBs and recovery rate to revisit original predictions.
- Revised forecasts imply much longer time to recovery in lower Hudson River fish PCBs.
- Overestimating sediment recovery rates minimizes differences in remedial scenarios.
- Model emulation provides a mechanism to evaluate both bias and precision of models.

GRAPHICAL ABSTRACT



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ABSTRACT

Remedial decision making at large contaminated sediment sites with bioaccumulative contaminants often relies on complex mechanistic models to forecast future concentrations and compare remedial alternatives. Remedial decision-making for the Hudson River PCBs Superfund site involved predictions of future levels of PCBs in Upper Hudson River (UHR) and Lower Hudson River (LHR) fish. This study applied model emulation to evaluate the impact of updated sediment concentrations on the original mechanistic model projections of time to reach risk-based target thresholds in fish in the LHR under Monitored Natural Attenuation (MNA) and the selected dredging remedy.

The model emulation approach used a combination of nonlinear and linear regression models to estimate UHR water PCBs as a function of UHR sediment PCBs and to estimate fish concentrations in the LHR as a function of UHR water PCBs, respectively. Model emulation captured temporal changes in sediment, water, and fish PCBs predicted by the mechanistic model over the emulation period. The emulated model, using updated sediment concentrations and a revised estimate of recovery rate, matched the trend in annual monitoring data for white perch and largemouth bass in the LHR between 1997 and 2014.

Our best predictions based on the emulated model indicate that the projected time to reach fish tissue risk-based thresholds in the LHR will take decades longer than the original mechanistic model projections.

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1. Introduction

Remediation decisions at large contaminated sediment sites with bioaccumulative contaminants often rely on highly parameterized mechanistic models to make long-range temporal projections comparing natural recovery and active remedial alternatives. At the Hudson River PCBs Superfund site in New York (Fig. 1), the U.S. Environmental Protection Agency (USEPA) used mechanistic contaminant fate and transport models linked to bioaccumulation models to predict future concentrations in fish (USEPA, 2000a, 2002). Model projections of temporal changes in fish concentrations played an important role in the comparative evaluation of remedial alternatives (USEPA, 2000b).

After USEPA's Record of Decision (ROD) (USEPA, 2002), extensive remedial design sediment sampling revealed that concentrations of PCBs in surface sediments were higher and more widespread than the models had predicted (Field et al., 2009; USEPA, 2010, 2012). Additionally, USEPA observed that PCB loads from the Upper Hudson River (UHR) to the Lower Hudson River (LHR) prior to the start of dredging in 2009 were substantially greater than predicted by the models and showed little evidence of decline (USEPA, 2010). Because modeled fish tissue PCB concentrations in the LHR are a function of PCB loads from the UHR, these findings imply that time to reach target thresholds for human consumption in fish in the LHR was underestimated by the original mechanistic model projections.

In this study, we used statistical model emulation to condense relationships between inputs and outputs of USEPA's linked mechanistic models to investigate sensitivity of model predictions to this new information. Model emulation reduces complex mechanistic models into computationally-efficient equations, dramatically reducing computational demands and time and effort to recalibrate and rerun the mechanistic models, while also maintaining a relevant and consistent representation of the underlying relationships within them (Logemann et al., 2004). The model emulator developed in this study was used to estimate new outputs associated with modified and updated inputs defining a range of remedial scenarios. The model emulator was also used to evaluate the sensitivity of model predictions to variation and uncertainty in initial sediment concentrations and different rates of natural recovery of surface sediment concentrations.

2. Methods

2.1. Study area

The Hudson River PCBs Superfund site extends approximately 321 km (200 miles) downstream from two General Electric (GE) capacitor manufacturing plants adjacent to the UHR to New York Harbor (Fig. 1). USEPA's ROD in 2002 (USEPA, 2002) called for dredging and monitored natural recovery (MNA) of PCB contaminated UHR sediments extending 64 km upstream from the Federal Dam at Troy. This area was divided into three main sections, River Sections (RS) 1 (Thompson Island Pool), RS2 (Schuylerville), and RS3. Because of its overall length, RS3 was subdivided into three modeling subsections RS3A (Stillwater), RS3B (Waterford) and RS3C (Troy). USEPA did not evaluate or select a remedy for the LHR tidal estuary (245 km between the Federal Dam and the Battery in New York City).

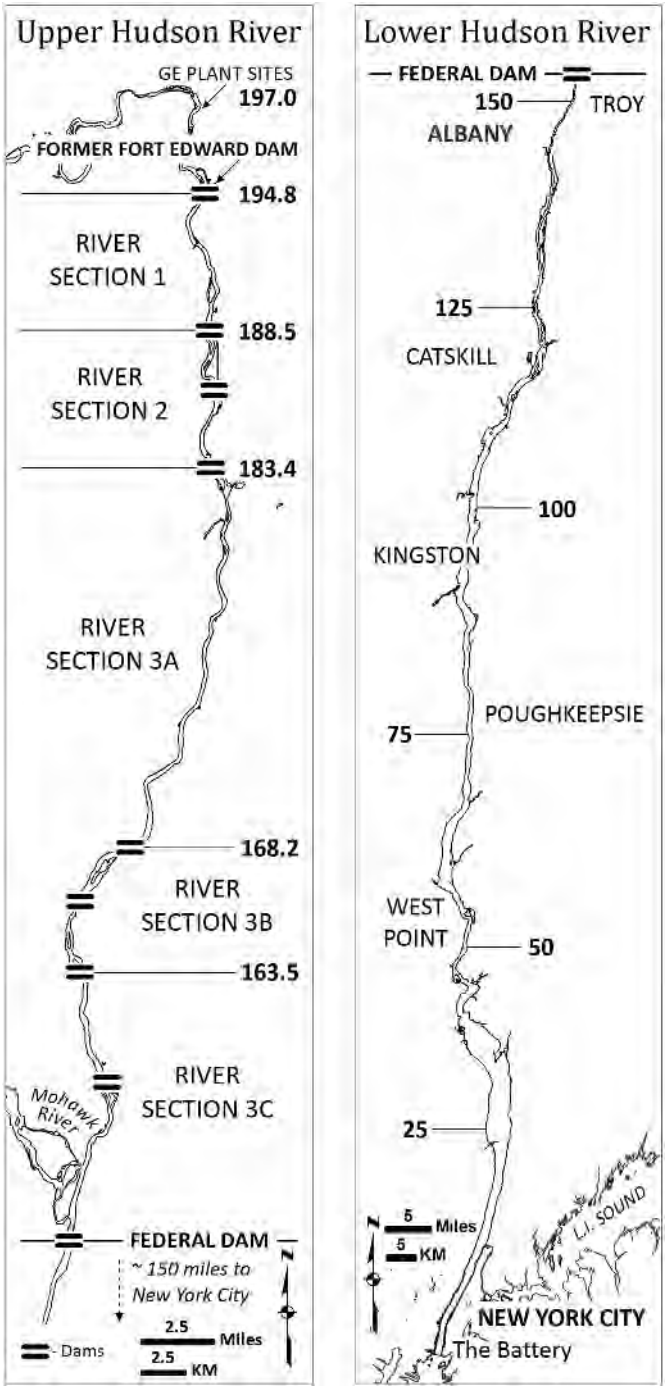

Fig. 1. Map showing the 321 km (200 mile) extent of the Hudson River PCBs Superfund site from Hudson Falls (above the GE plant sites) to The Battery in New York City. The left panel for the Upper Hudson shows the River Sections (RS) for the approximate 64 km (40 mile) remedial action area. The right panel for the Lower Hudson shows the 241 km (150 mile) tidal estuary with the fish model locations.

2.2. Sample sediment data

Sediment samples collected for PCB analysis between 1976 and 1999 by USEPA, GE and New York State were used during the Remedial Investigation and Feasibility Study (RI/FS) to assess risk and to predict future concentrations under various remedial scenarios (USEPA, 2000b, 2000c). Surface sediments were generally collected from the top 5 cm, although some penetrated as deep as 15 cm. Tri+ PCBs in water (average annual whole water concentrations), sediment and fish, the sum of trichlorobiphenyl and higher chlorinated homologues, were used for modeling because historic total PCB data did not effectively quantify mono- and di-chlorobiphenyl PCBs (USEPA, 2000a; Connolly et al., 2000). PCBs in fish tissue are primarily composed of Tri+ PCBs (USEPA, 2000a, 2002).

Subsequent to USEPA's ROD, GE collected sediment samples (mostly cores with some grab samples) from over 8000 locations throughout the UHR supporting design and implementation of the selected remedy. In RS1, most cores were collected on a triangular 24-meter (80-foot) grid from the entire pool. In RS2 and RS3, cores were collected almost exclusively within fine-grained sediments on triangular 24- or 50-

meter grids (QEA, 2002, 2005, 2007). This sampling design is considered approximately unbiased in RS1 and unbiased to fine-grained sediments in RS2 and RS3.

2.2.1. Estimated pre-dredge concentrations

We averaged surface Tri+ PCB concentrations from design sampling conducted from 2002 through 2005 representing pre-dredge surface sediment concentrations in 2003. We used these averages for initial conditions comparing updated MNA and remedial (REM) scenarios.

Most (94%) of the samples represented the top 5 cm and the remainder were from the top 15 cm or less. Average concentrations from samples including intervals up to 15 cm in depth differed inconsequentially from samples composed of the 0–5 cm interval. The USEPA mechanistic model simulated PCB fate and transport in the top 4 cm.

2.2.2. Estimated post-dredge surface sediment concentrations

Evaluating the change in surface sediment concentration following remediation required an estimate of expected post-dredging Tri+ PCB concentrations in sediment. Samples within the remedial design dredge footprints (Arcadis, 2013) were assigned a post-dredge surface sediment Tri+ PCB concentration of 0.25 mg/kg (USEPA, 2002) and arithmetic averages for each river subsection were recalculated to represent the concentration in 2003, the year USEPA expected dredging to commence.

2.2.3. Estimated surface sediment concentration decay rate

Field et al. (2009) found that the exponential temporal decrease in sediment PCBs (exponential decay rates) estimated from USEPA's mechanistic models overstated the rate of natural recovery of surface sediments. GE conducted large-scale sediment surveys throughout the UHR in 1991 (O'Brien and Gere Engineers, Inc., 1993) and in 2002 through 2005 as part of remedial design (QEA, 2005, 2007). We compared average surface concentrations from these two surveys and calculated an exponential decay rate for each river section (Table 1). The average surface sediment Tri+ PCB concentration representing 2003 in each modeled river subsection was calculated, using only samples from the top 5 cm matching the top 5 cm sampling interval collected in 1991. In RS2 and RS3, these samples from 2003 can be considered representative of cohesive sediment deposits and directly comparable to samples from the cohesive sediment transects from 1991. By necessity, decay rate estimates for RS1 were based on comparison of remedial design samples, representing both cohesive and non-cohesive sediments, with samples representing cohesive sediments collected in 1991. Because cohesive sediments tended to have higher than average Tri+ PCB concentrations, the estimated decay rate is likely to overstate the actual rate. The overall average decay rate and confidence interval (CI) was used to guide selection of model emulation scenarios.

Table 1
Average surface (top 5 cm) sediment Tri+ PCB concentration (mg/kg) in 1991 and 2003 and estimated exponential decay rate.

Model subsection	Cohesive sediment 1991[a]	Updated sediment 2003[b]	Exponential decay
1	20 (227)[c]	16.9 (3414)[c]	1.4%
2	18 (33)	14.7 (1539)	1.7%
3A	4.3 (103)	3.4 (2129)	2.0%
3B	5.7 (30)	5.6 (682)	0.1%
Average 95% confidence interval (CI)			1.3% (−0.1% to 2.6%)

[a] O'Brien and Gere Engineers, Inc. (1993).
[b] Includes cohesive and non-cohesive sediments in River Section 1 and cohesive only in River Sections 2 and 3.
[c] Number of samples.

2.3. Selected remedy

The selected remedy, initiated in 2009, included both MNA and active remediation (dredging and backfill or capping followed by MNA) in the UHR. Sediment remediation areas were defined primarily on two criteria: surface concentrations (defined by USEPA as the top 30 cm) and mass-per-unit area (MPA), a measure of PCB inventory. Remediation areas were defined as follows: for RS1, a surface concentration of 10 mg/kg Tri+ PCBs in the surface or an MPA of 3 g/m^2 Tri+ PCBs; for RS2 and RS3, a surface concentration of 30 mg/kg Tri+ PCBs or an MPA of 10 g/m^2 Tri+ PCBs. Source control near GE plant sites, approximately 3 km upstream of the modeled area, was assumed under both MNA and active remediation scenarios.

2.4. Mechanistic model framework

The mechanistic numerical models developed by USEPA predicted sediment, water and fish Tri+ PCB concentrations in the RS1, RS2, RS3A, and RS3B reaches of the UHR (USEPA, 2000a). GE also developed similar mechanistic models that were generally consistent with those developed by USEPA (QEA, 1999a). USEPA used the projections of PCB load from the UHR (RS3B) to the LHR from the Upper Hudson River Toxic Chemical Model (HUDTOX) as input to the Farley model (Farley, 1999; USEPA, 1999) to calculate sediment and water concentrations in the LHR. Output from the Farley model was then used as input to USEPA's FISHRAND model, a mechanistic food web model, to predict Tri+ PCB concentrations in four species of fish (white perch, brown bullhead, largemouth bass, and yellow perch) at four LHR locations downstream of the Federal Dam at Troy (RM152 (Albany/Troy) (river kilometer [RK] 245), RM113 (Catskill) (RK 182), RM90 (Kingston) (RK 145), and RM50 (West Point) (RK 80) USEPA, 2002). While PCB-contaminated sediment in the UHR was the primary focus for remedial alternatives, reduction in PCB load to the LHR was a major remedial action objective and was expected to result in a reduction of PCB concentrations in lower river fish. Because initial PCB concentrations in LHR fish were lower than UHR fish, model projections indicated that LHR fish would reach human health risk management objectives (thresholds) much sooner than UHR fish.

We captured mechanistic model output by digitizing Tri+ PCB time series from the USEPA mechanistic model output for MNA and the selected remedy, including sediment (USEPA, 2000b: Figures 6-24, 6-26, 6-28, and 6-30; USEPA, 2002: Figures 363150-1, 3, 5, and 7) and water (USEPA, 2002: Figures 363150-10, -11, -12, and -13) for four model subsections in the UHR and fish at four locations in the LHR (USEPA, 2002: Figures 313787-2, 3, 4, and 5). Digitizing was accomplished using Plot Digitizer, a shareware Java program used to digitize scanned plots. Digitized sediment, water, and fish Tri+ PCBs time series were interpolated to equally-spaced annual time steps so that modeled values for each media could be paired temporally. Interpolation was conducted using linear interpolation using MATLAB© software (MATLAB 8.6, Release 2015b, The MathWorks Inc., Natick, MA, 2000). These time series simulated scenarios assumed dredging would begin in 2003 or 2004 and end by 2010 for the selected remedy.

2.5. Model emulation

Digitized input and output from mechanistic model projections provided a basis for using nonlinear optimization to fit a simplified mathematical model of water concentrations (C_w) in each UHR subsection as a function of 1) original and updated sediment Tri+ PCBs (C_s), 2) upstream source input (2 ng/L or 0 ng/L), 3) area of subsection, and 4) distance from the downstream dam in each subsection (see Supplementary Fig. 1). The emulated model structure is a simplified parameter version of the USEPA mechanistic model including four one-dimensional model compartments representing each river subsection.

2.5.1. Model emulator

The model emulator represented each of the four river subsections with one model compartment composed of three terms representing PCB transfer to or from the water column: 1) upstream source minus deposition; 2) release/resuspension minus deposition of a fraction of these resuspended solids; and 3) post-dredge resuspension of disturbed residuals. The general form of the emulator within the *i*th subsection is:

$$\begin{aligned} \textit{Water Column Load}_i = &(\textit{Water Column Load}_{i-1} - \textit{Deposition}_i) \\ &+ (\textit{Resuspension}_i - \textit{Deposition}_i) + \textit{Post Dredge Resuspension}_i. \end{aligned} \quad (1)$$

Each model compartment (i.e. river subsection) represents an impounded pool within which flows are generally laminar. Deposition of PCBs from the water column to the sediment bed was assumed proportional to distance traveled within each subsection with constant deposition rate per unit distance (g_i, $i = 1,2,3,4$) within river segments.

Release/resuspension of sediment PCBs to the water column was assumed to be directly proportional to average PCB concentration and area of PCB-containing cohesive sediments per river subsection with net sediment to water transfer coefficients (γ_i; $i = 1,2,3,4$) assumed constant through time.

Post-dredging sediment residuals were assumed to be more susceptible to resuspension with sediment to water transfer coefficients (β_i; $i = 1,2,3,4$) proportional to pre-dredge PCB concentrations and area dredged. These lower density disturbed residuals were assumed to decline with time at an 8% rate as they either flushed downstream, or became more consolidated and less susceptible to erosion.

Lower Hudson River fish Tri+ PCBs (C_f) were predicted from modeled water column Tri+ PCB concentrations (C_w) from the mechanistic model output for RS3B using linear regression.

2.5.2. Emulator calibration

Net contaminant transfer coefficients were estimated by minimizing root mean squared error between temporally paired emulated and mechanistic modeled Tri+ PCB concentrations in water. The paired sediment and water time series for each of the 4 river sections spanned 30 years (2005–2034) for MNA and 25 years (2010–2034) for REM1 (the selected remedy) and each remedial scenario was modeled assuming: 1) partial source control with Tri+ PCB load decreasing from 0.16 kg/d to 0.0256 kg/d by the year 2005; and 2) complete source control, assuming upstream Tri+ PCB load would decrease from 0.16 kg/d to 0.0 kg/d (USEPA, 2000b). These 55 time steps and 4 river sections and 2 upstream load scenarios resulted in a system of 440 simultaneous nonlinear equations with 12 unknown net transfer coefficients which were solved using nonlinear optimization using MATLAB© scientific software (The MathWorks 2015). Full mathematical detail is provided in Appendix A. The estimated coefficients are summarized in Table S-1. Mechanistic water column Tri+ PCB concentrations from RS3B were treated as predictors of LHR fish Tri+ PCB concentrations and were calibrated by linear regression. Projections of LHR fish tissue Tri+ PCBs were calculated by applying this regression model to emulated water Tri+ PCB concentrations at the downstream end of RS3B.

Although we calibrated the model emulation to both upstream load scenarios, we found only small differences in future model projections of primary interest, so we focused on scenarios with average upstream source concentrations of 0.0256 kg/d (approximately 2 ng/L Tri+ PCB). This is reasonable because measured water column Tri+ PCB concentrations upstream of RS1 have been approximately 2 ng/L Tri+ PCB since 2004 (Farrar, 2011; USEPA, 2010). For the calibration step, we selected 2005 as the initial year for MNA because mechanistic model projections reached baseline concentrations of 2 ng/L Tri+ PCBs in that year. Initial year 2010 was selected for REM1 because dredging was anticipated to be completed by that time.

2.5.3. Uncertainty

Analytical statistical theory for mechanistic simulation models is generally intractable due to their complexity, so statistical inference to model predictions is often limited. In situations where computer runtime for simulation models is relatively short, statistical inference may be available through Monte Carlo simulation or Bayesian Markov Chain Monte Carlo Methods (Raftery et al., 1995; Smith, 1994; USEPA, 1994). These examples have the commonality that mechanistic model equations are relatively simple and can be run repeatedly, a necessity for both Bayesian and Monte Carlo methods. Because linked fate and transport models often require extremely long run-times (Glaser and Bridges, 2007), Monte Carlo or Bayesian simulation is not directly applicable. Model emulation provides a solution to this computational problem by providing a surrogate model that can be run repeatedly within a reasonable period of time, while maintaining essential elements of the physical processes embodied in the mechanistic model. This advancement provides a mechanism to evaluate both bias and precision of models, providing risk managers with a more complete description of the reliability of predictions.

2.5.3.1. Bias. Our primary objective was to apply model emulation deterministically to evaluate bias in modeled forecasts associated with change in initial sediment bed Tri+ PCB concentrations. Future Tri+ PCB concentrations in sediment, water, and fish tissue were estimated using updated sediment Tri+ PCB concentrations reflecting averages from comprehensive remedial design sampling. Changes in these values associated with updated estimates of temporal decay rates in sediments were also considered. Using these modified model inputs, future Tri+ PCB concentrations in LHR fish were re-calculated and compared to human health total PCB risk thresholds of 0.05 mg/kg, 0.2 mg/kg and 0.4 mg/kg, representing levels protective of fish consumers eating one meal per week, one meal per month, and one meal every two months respectively (USEPA, 2002). USEPA considered Tri+ PCB and total PCB concentrations interchangeable in fish (USEPA, 2002).

These estimates representing central tendency or best estimates updated for new sediment surface and decay rates were compared with the original mechanistic model estimates.

2.5.3.2. Precision. We also estimated precision of model forecasts using parametric Monte Carlo simulation for auto-correlated time series of sediment Tri+ PCB concentrations. Synthetic sediment time series were generated that reproduced temporal autocorrelation patterns and between river section cross correlations similar to those in original EPA mechanistic modeled sediment time series. Each sediment Tri+ PCB concentration time series was simulated from a lognormal distribution with mean concentration

$$C_i(t) = C_{0i} e^{-kt + \varepsilon_i(t)}$$

where C_{0i} is the initial sediment Tri+ PCB concentration in the *i*th subsection, and *k* is the PCB concentration decay rate. Because the sediment decay rate was estimated from just two points in time (1991 and 2003), we viewed this as a relatively uncertain parameter and as such investigated a relatively wide range of plausible decay rates uniformly distributed on the interval from 0.02 to 0.05. The residual time series $\varepsilon_i(t)$ was simulated as a normally distributed mean zero correlated random variable with autocorrelation and variance estimated from the residuals of an exponential fit to the mechanistic model time series. [The mathematical details of this probability model are summarized in Appendix B.]

This Monte Carlo simulation procedure involved four steps; 1) simulating four normally distributed auto-correlated sediment time series ($\varepsilon_i(t)$, $i = 1,2,3,4$), 2) randomly selecting a uniformly distributed decay coefficient between 0.02 and 0.05, 3) calculating $C_i(t)$ and 4) applying the model emulator, to these four sediment time series, producing four corresponding Tri+ PCB time series for water and finally a synthetic fish tissue Tri+ PCB time series. These four steps were

repeated 1000 times, and the fish Tri+ PCB time series were plotted, and the time to reach risk thresholds was calculated for each of the 1000 synthetic time series.

2.6. Remedial scenarios evaluated

Model emulation was used to evaluate the following remedial scenarios: (1) Mechanistic model projections for sediment PCB concentrations under Monitored Natural Attenuation (MNA1) and the selected remedy (REM1); (2) MNA (MNA2) and the selected remedy with updated sediment PCBs (REM2); and (3) An alternative remedial scenario (REM3), not considered in the ROD, that applies the RS1 cleanup target levels to RS2 and RS3 with updated sediment PCBs. For each of these scenarios, we applied both the original (8%) and the updated (3%) rate of exponential decrease in surface sediment PCBs.

3. Results

3.1. Model emulator

3.1.1. UHR sediment to water

Fitting a set of nonlinear and linear regression models using inputs and outputs from the original mechanistic models provided a computationally simple means to reproduce the USEPA water column model Tri+ PCB results under MNA and selected remedy scenarios. The mechanistic model developed by USEPA predicted sediment and water Tri+ PCB concentrations in RS1, RS2, RS3A and RS3B that were used to compare remedial alternatives.

The four-compartment nonlinear model emulator with twelve parameters linking PCB transfer from sediment to water explained 98% ($R^2 = 0.98$) of the variation in mechanistic modeled water column concentration over the 30 year projection for MNA and the 25 year projection for REM1 (Fig. 2). This demonstrates that the model emulator successfully captures the changes in sediment and water concentrations predicted by the mechanistic model for MNA and for the selected remedy in the UHR model sections over the emulation period.

3.1.2. UHR water to LHR fish

The mechanistic model predicted Tri+ PCB concentrations in four species of fish (white perch, brown bullhead, largemouth bass, and yellow perch) at four locations in the LHR (USEPA, 2002). Fish tissue Tri+ PCB concentrations in the LHR below the Federal Dam (RM152) had a strong linear relationship to water column Tri+ PCB at Waterford (RS3B) in the UHR for all four modeled species ($R^2 \geq 0.90$, Fig. 3). This linear relationship between water Tri+ PCB at RS3B and LHR fish concentrations in the mechanistic model output provided the basis for the model emulation of fish PCBs.

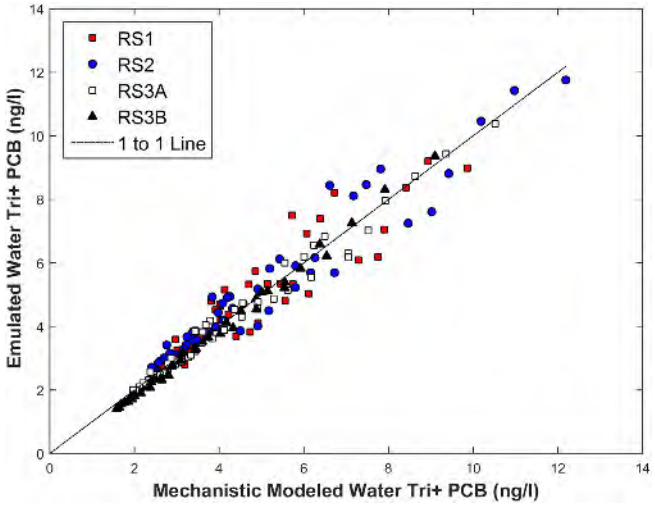

Fig. 2. Emulated vs original mechanistic model projected Tri+ PCB (ng/l) water concentrations by river subsections on the Upper Hudson River for MNA and the selected remedy.

Modeled fish tissue Tri+ PCBs for all four species at the other three LHR locations (RM113, RM90 and RM50) were also strongly linearly related to Tri+ PCB concentrations at Waterford, showing that the mechanistic model linking water to fish was effectively linear [Supplementary Table S-1 lists the regression coefficients and standard errors for white perch, brown bullhead, largemouth bass, and yellow perch at all four LHR locations].

Mechanistic food web model predictions of fish tissue concentrations for all four species at RM152 are strongly linearly related ($R^2 > 0.99$; Supplementary Fig. 2). Largemouth bass are predicted to have higher PCB concentrations than white perch, while brown bullhead and yellow perch are predicted to have lower concentrations.

Mechanistic model projections of white perch Tri+ PCB concentrations at RM113, 90, and 50 are also proportional to white perch Tri+ PCB concentrations at RM152 ($R^2 > 0.96$) and decrease with distance from the Federal Dam (Supplementary Fig. 3). The other three species had similar proportional relationships (not shown).

Emulation equations, with estimated coefficients, were applied to new model inputs such as new average PCB concentrations and decay rates in sediment (see Table A.2 for nonlinear regression coefficients).

The model emulation combined the nonlinear regression model between sediment and water with these linear regressions linking fish tissue and water column Tri+ PCBs to predict fish tissue Tri+ PCBs in the LHR from sediment Tri+ PCB concentrations in the four upper river sections. A comparison between the mechanistic model projections of Tri+ PCBs for all four species at RM152 and the emulation results are shown in Fig. 4 ($R^2 = 0.92$). Emulated model concentrations for largemouth bass and white perch tended to underestimate the mechanistic model at the higher concentrations (early in the time period).

3.1.3. Updated surface sediment concentrations

The average Tri+ PCB concentration in sediment samples from the top 5 cm in 2003, exceeded the upper bound of the mechanistic model predictions (representing the top 4 cm) under MNA (MNA1) and were more than twice the mean concentration predicted for cohesive sediments in all four model subsections of the UHR (Table 2; Fig. 5). The GE mechanistic model for RS1 similarly understated average measured sediment PCBs in 2003 (QEA, 1999a).

The projected Tri+ PCBs concentrations in surface sediment under USEPA's natural recovery scenarios declined with an approximate 8% annualized exponential decay rate (USEPA, 2000a). Using the cohesive sediment data from the 1991 transect survey and the sediment data collected in 2003, we estimated the decay rate over the twelve year period to be 2% or lower in all four model sections (Table 1) with an average decay rate of 1.3% (95% CI = −0.1% to 2.6%). The 3% rate selected for simulated scenarios was a round number representing a reasonable upper bound for calculated decay rates shown in Table 1.

Dredging was expected to begin in 2003 and require 6 years to complete (USEPA, 2000b). In the emulation, we treated 2010 as the first post-dredging year. We assumed that natural recovery would continue outside the dredging footprint while dredging occurred. To estimate surface sediment concentrations in the initial post-dredging year, needed for simulating post-dredging scenarios, exponential decay rates of 8% and 3% were applied to the average surface concentration estimated from pre-design sampling in 2003. Post-dredging river-subsection averages were then calculated accounting for reduced concentrations due to dredging and backfilling (Table 2).

The post-dredging surface Tri+ PCB concentrations estimated for 2010 were also considerably higher than predicted by the USEPA models. In RS2 and RS3, where the target cleanup levels were at least a factor of 3 higher than for RS1, estimated post-dredging surface

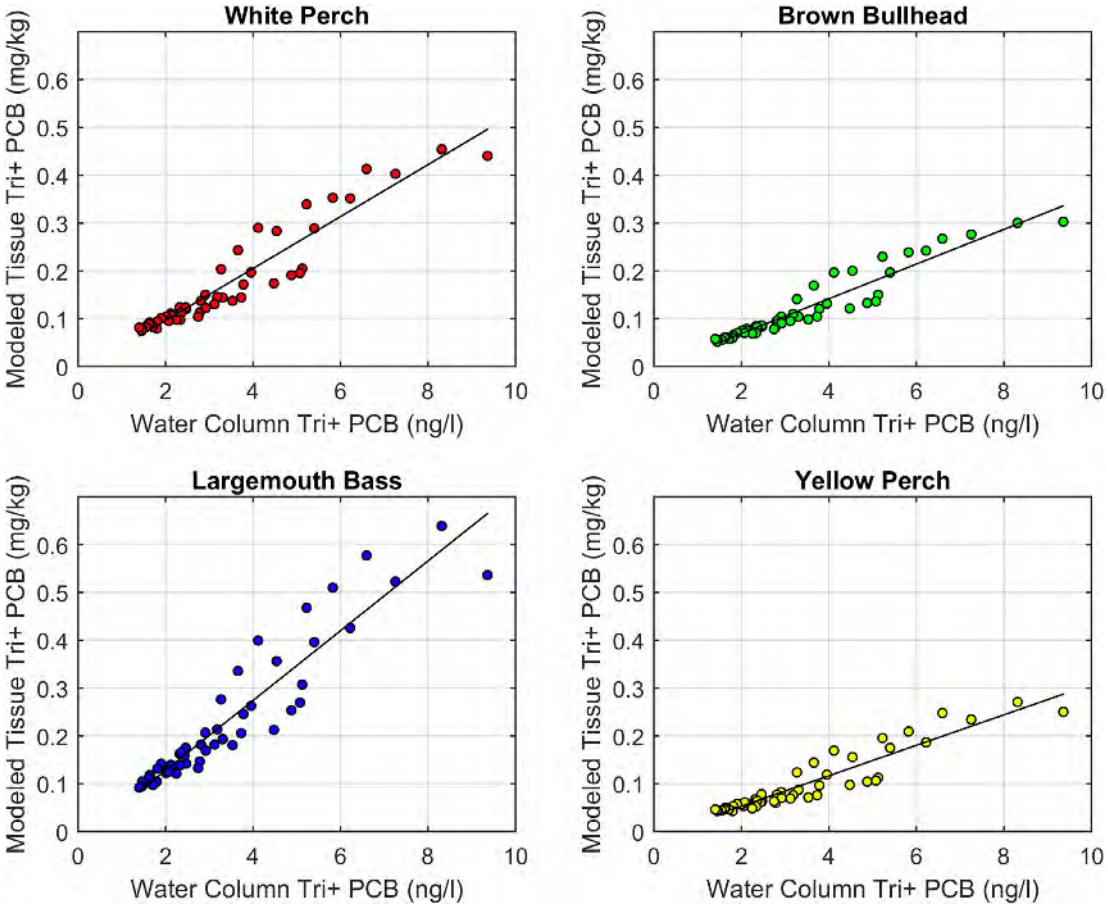

Fig. 3. Mechanistic model Tri+ PCB water concentrations (ng/l) at Waterford (RS3B) vs tissue concentrations (mg/kg) for white perch, brown bullhead, largemouth bass, and yellow perch from RM152 for MNA and the selected remedy.

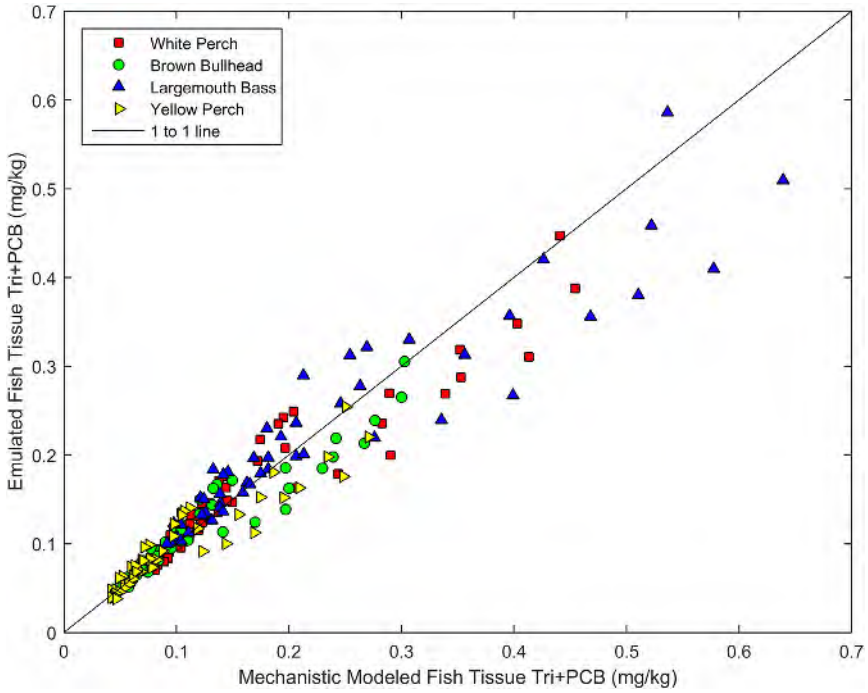

Fig. 4. Emulated vs original mechanistic model projected Tri+ PCB (mg/kg) fish concentrations for white perch, brown bullhead, largemouth bass, and yellow perch from RM152 for MNA and the selected remedy.

Table 2
Average Tri+ PCB concentrations (mg/kg) in surface sediment by river subsection under different remedial scenarios and rate of exponential decay in concentration between 2003 and 2010.

River subsection	Reach	Remedial scenario					
		MNA1[a]	MNA2[b]	REM1[c]	REM2[d]	REM2[e]	REM3[f]
	Year	2003	2003	2010	2010	2010	2010
	Decay				8%	3%	3%
RS1	Thompson Island Pool	8.5	16.9	0.5	0.8	1.1	1.1
RS2	Schuylerville	6.5	14.7	1.0	2.8	3.9	1.0
RS3A	Stillwater	1.3	3.7	0.5	1.4	2.0	1.0
RS3B	Waterford	1.0	6.0	0.4	1.9	2.7	0.9

[a] MNA1: Mechanistic model predictions for Monitored Natural Attenuation for sediment concentrations in 2003.
[b] MNA2: Measured sediment concentrations in 2003 based on updated data.
[c] REM1: Mechanistic model predictions for the selected remedy for sediment concentrations post-remediation (2010).
[d] REM2: Estimated concentrations for the selected remedy post-remediation (2010) based on updated data, assuming 8% exponential decay since 2003.
[e] REM2: Estimated concentrations for selected remedy post-remediation (2010) based on updated data, assuming 3% exponential decay since 2003.
[f] REM3: Estimated post-remediation (2010) concentrations for hypothetical remedial scenario that applies RS1 cleanup levels to RS2 and RS3, based on updated data and assuming 3% exponential decay since 2003.

concentrations, based on updated data, are about 5 times higher than previously predicted based on the mechanistic model.

3.1.4. Emulated models with updated surface sediment concentrations pre- and post-removal

The effect of a lower natural recovery rate (3%) in sediment was also evaluated in combination with updated sediment surface Tri+ PCBs concentration. This updated decay rate is more consistent with the observed changes in surface concentrations during the 12 year period between the 1991 transect survey and the remedial design data collected in 2003, while not being overly conservative with respect to anticipated decay rates. The mechanistic model profile using USEPA's original projections of sediment concentrations under MNA (MNA1) and the selected remedy (REM1) was compared to the emulated model projections using an exponential decay rate of 8%. The computed exponential decay function closely matches the original model projections (Fig. 6), supporting the use of an exponential decay model for emulated results representing other decay rates (e.g., 3%) for surface sediment concentrations under MNA2, REM2 and REM3.

The emulated models projected LHR fish Tri+ PCBs using updated surface sediment concentrations (i.e., based on the 2003 pre-design sampling) as input. Estimates of pre- and post-removal surface sediment concentrations derived from the extensive remedial design sediment dataset (Table 2) provided more accurate characterization of surface Tri+ PCB concentrations prior to initiation of remediation.

Fig. 7 illustrates the difference between USEPA's original scenarios (MNA1 and REM1 with 8% decay rates) and updated scenarios (MNA2, REM2 and REM3 with updated sediment and 3% decay rates) for Tri+ PCB concentrations in white perch at RM152. The emulated LHR fish Tri+ PCB concentrations (MNA2, REM2, REM3) were substantively higher than USEPA's original mechanistic model predictions for MNA1 and REM1 and remain elevated over a much longer period. The updated sediment surface and decay rates for MNA2, REM2, and REM3 provide greater discrimination between remedial alternatives than in the evaluation of remedial alternatives prior to remedy selection.

The model emulator was used to estimate the number of years necessary to reach USEPA risk thresholds in white perch at RM152 under original modeled scenarios (MNA1, REM1) with the number of years to reach thresholds based on updated scenarios (MNA2, REM2, REM3) using two sediment exponential decay rates: 8% (mechanistic model) and 3% (upper bound of empirical estimate). Fig. 8 displays the number of years predicted to attain the 0.4 and 0.2 mg/kg Tri+ PCB thresholds for white perch at RM152 under remedial scenarios REM1, REM2 and REM3, each with 3% and 8% exponential decay rates. For all scenarios, using the updated sediment concentrations the time for fish tissue Tri+ PCB concentrations to reach remedial action objectives of 0.4 and 0.2 mg/kg is estimated to be substantively longer than originally predicted. For the original selected remedy (REM1) under either 8% or 3% decay assumptions, white perch at RM152 were projected to reach the 0.4 mg/kg threshold before or immediately after dredging was completed. With updated sediment concentrations (REM2) and 3% decay, white perch at RM152 were estimated to reach 0.2 mg/kg more than six decades longer than the original mechanistic model projections. The REM3 scenario greatly reduced the time to thresholds compared to REM2, but still longer than the original model predictions (REM1) [see Supplementary Tables S-2 and S-3 for time to 0.2 and 0.4 mg/kg thresholds for all scenarios, species, and locations].

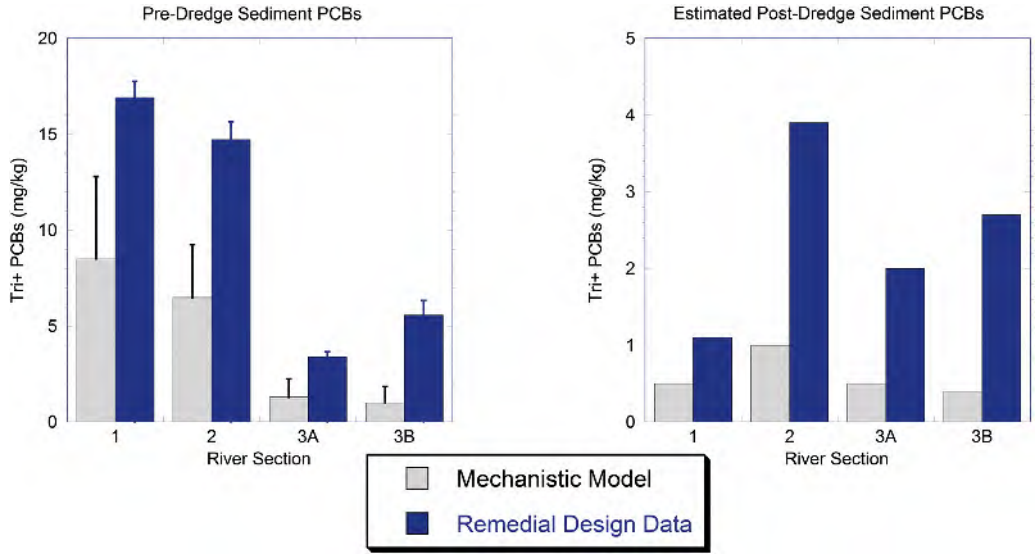

Fig. 5. Mechanistic model predictions of average and upper bound (error bars) surface sediment (top 4 cm) Tri+ PCB concentrations for 2003 pre-dredging (left panel) and post-dredge concentrations (right panel) compared to estimated river subsection average pre- and post-dredge sediment (top 5 cm) concentrations from remedial design sampling between 2002 and 2005 (approximately 2003).

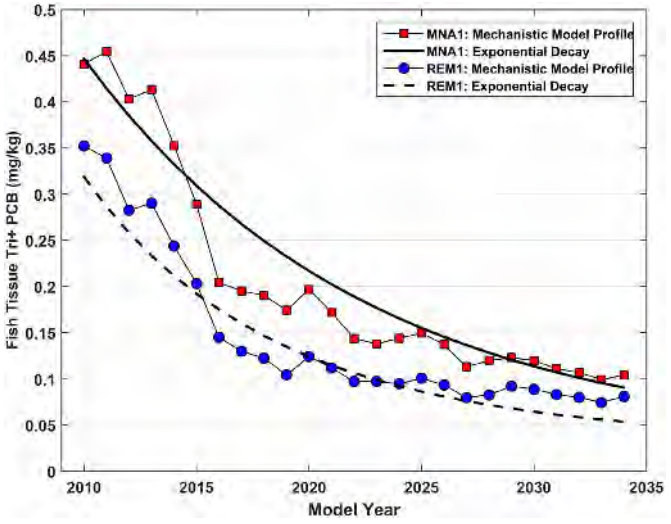

Fig. 6. Emulated model projections for white perch Tri+ PCB concentrations (mg/kg) from RM152 under MNA (MNA1) and the selected remedy (REM1) comparing the original mechanistic model (square and circle) results with simulated exponential decay rate of 8% (solid and dashed line).

3.2. Precision

Precision of emulator-based Tri+ PCB concentration in fish tissue was estimated using Monte Carlo simulation of equally likely sediment time-series with a range of decay rates (2% to 5%) and with statistical properties matching original mechanistic model sediment time series. The emulator was applied to these time-series, propagating uncertainty in sediment Tri+ PCB concentrations through to corresponding uncertainty in output Tri+ PCB concentrations in white perch at RM152. Fig. 9 shows the Monte Carlo distribution of future trajectories of fish tissue Tri+ PCB concentration, illustrating the uncertainty in estimates of the number of years needed to reach risk thresholds. The estimated number of years to thresholds were estimated to be 27 (95% CI: 19, 43), 49 (95% CI: 35, 77) and 102 (95% CI: 73, 162) for the 0.4 mg/kg, 0.2 mg/kg and 0.05 mg/kg risk based thresholds respectively.

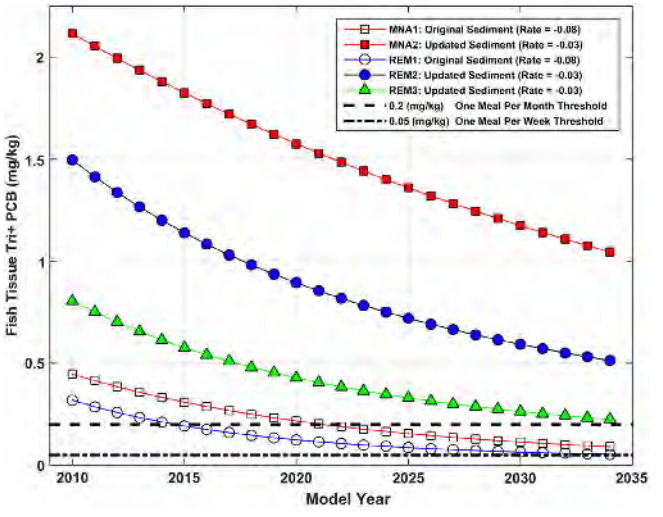

Fig. 7. Emulated model projections for white perch Tri+ PCB concentrations (mg/kg, wet weight) from RM152 for MNA (squares) and the selected remedy (REM) (circles) comparing the time to reach risk thresholds of 0.2 and 0.05 mg/kg at 8% (open symbols) and 3% (filled symbols) exponential decay rates for original mechanistic model concentrations (MNA1, REM1), updated sediment concentrations from remedial design sampling (MNA2, REM2), and hypothetical scenario that applies the RS1 target cleanup levels to RS2 and RS3 using updated sediment concentrations (REM3) (triangles).

4. Discussion

4.1. Interpretation of key findings

4.1.1. Model emulation

Model emulation provides a fast and inexpensive way to efficiently calculate outputs from inputs for complex mechanistic models, while retaining underlying physics-based properties. The method of model emulation is relatively new, with recent developments in global climate modeling stimulating the need to quantify uncertainty in complex mechanistic simulation models (Castruccio et al., 2014). An approach similar to ours was proposed by Margvelashvili et al. (2010) emulating a linked one-dimensional sediment/contaminant and three dimensional sediment transport model in the South-East Tasmanian coast of Australia.

For the Hudson River, sediment fate and transport model emulation successfully reproduced mechanistic model projections of sediment and water Tri+ PCB concentrations in the UHR and fish Tri+ PCB concentrations in the LHR. These results demonstrate that essential elements of the mechanistic mass balance model were captured by the emulator and support its validity for re-visiting temporal projections of fish tissue concentrations in the LHR with updated model inputs. Use of the emulator allowed us to update original predictions without necessitating access to computer codes that are often not readily available to third party investigators. Model emulation may also reduce the time to update complicated simulation models, because recalibration procedures may also entail re-evaluation of the physical mechanisms of the model itself. We believe these features of model emulation could enhance the transparency and accountability of the comparisons of alternative remedial scenarios.

4.1.2. Surface sediment concentrations and natural recovery

Extensive systematic remedial design sampling of surface sediment conducted to delineate dredge areas showed that the mechanistic model predictions of surface sediment concentrations underestimated surface PCBs under MNA and post-remediation scenarios and overestimated the rate of decrease in surface sediment PCBs. The higher than predicted post-remediation concentrations primarily resulted from high concentrations of PCBs in surface sediment adjacent to the planned dredge areas (Field et al., 2011).

Multiple reasons are possible for the mechanistic model underestimating surface sediment Tri+ PCBs, but processes that resulted in an overstated effective recovery rate (8%, MNA1 scenario) (as compared to our empirical estimate of <3% from data only available after the original model was developed) should be considered. Overestimated natural recovery rates are not unique to this model or this situation. For example, models developed by GE for the UHR had a similar effective decay rate (QEA, 1999a). Rates of recovery derived from data collected in the 1970s to mid-1980s have also led to overly optimistic estimates of rates of decline. Consistent with our findings, PCB concentrations in Great Lakes salmonids declined at high double digit rates in the 1970s and 1980s, but the inclusion of more recent data showed that declines have slowed to the low single digits in the 1990s and later (Rasmussen et al., 2014). Examination of PCB data from the 1970s to 2000s in several species of Great Lakes fish suggest that the estimates of contaminant decline were overly optimistic and responses to mitigation weaker than anticipated (Carlson et al., 2010; Sadraddini et al., 2011).

4.1.3. Estimated rate of recovery and fish concentrations

Monitoring data for adult white perch collected annually at RM152 in the late spring between 1997 and 2014 (NOAA, 2015) were normalized to 3% lipid for consistency with the USEPA FISHRAND model and overlaid on updated emulated model predictions for MNA (MNA2) at 3% and MNA1 at 8% decay. The original mechanistic model understates the measured tissue concentrations, whereas the updated predictions using 3% decay are more consistent with the measured data (Fig. 10).

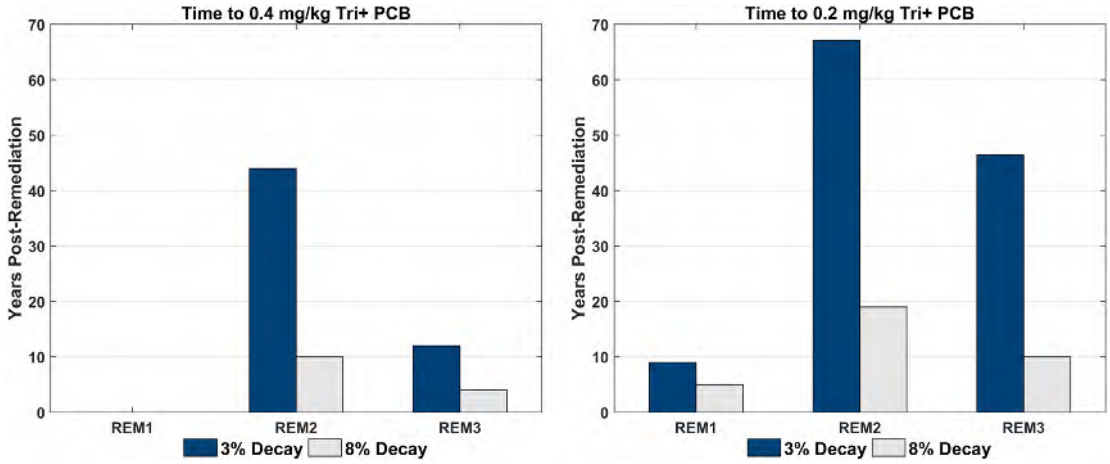

Fig. 8. Emulated model projections of the number of years to reach 0.4 and 0.2 mg/kg Tri+ PCB thresholds for white perch at RM152 under three remedial scenarios and two exponential decay rates, 3% and 8%: the selected remedy with original initial sediment concentrations (REM1), the selected remedy with updated initial sediment concentrations (REM2), and a hypothetical scenario that applies the RS1 target cleanup levels to RS2 and RS3 using updated sediment concentrations (REM3).

It could be argued that this apparently lower than expected decay rate in LHR white perch tissue concentrations is an artifact of Tri+ PCB releases from UHR dredging which began in 2009. However, the updated predictions equally describe trends in monitoring data collected between 1997 and 2009 (Fig. 10), supporting the lower than anticipated 3% recovery rate. It should also be noted that, due to a change in fish processing protocol between 2004 and 2013 (USEPA, 2015), lipid-adjusted Tri+ PCBs shown in Fig. 10 may understate actual concentrations during that time period. Adjusting these data for this change in protocol would shift Tri+ PCBs upward, suggesting even slower recovery rates, again supporting our finding that recovery rates are <8%. Similar results were observed for largemouth bass (Supplementary Fig. S-4). The monitoring data do not definitively identify the correct decay rate, but 3% is a demonstrably better fit to the data than 8%.

4.2. Use of model emulation to evaluate uncertainty

Resource managers need to account for uncertainty in modeled forecasts to avoid selecting overly optimistic, or pessimistic, remedial options. For relatively simple measurement endpoints, statistical analyses are regularly used to quantify uncertainty. For example, uncertainty in exposure estimates is generally quantified using 95% confidence limits. When more complicated functions of the data are involved, the statistical methods of bootstrapping (Efron, 1979) and Monte Carlo simulation (Manly, 1991; USEPA, 1997) are used to describe uncertainty distributions. Bootstrap and Monte Carlo methods involve selecting equation inputs from statistical distributions to which model equations are applied, producing distributions of model outputs. Traditional metrics of uncertainty, such as confidence intervals or percentiles, are calculated directly from the output distributions. The time required to run linked sediment fate and transport models precludes direct application of bootstrap and Monte Carlo methods, because the model runs must be repeated many times to develop statistical distributions of output parameters.

Our model emulation provides a novel approach to extend the utility of complex linked sediment transport, contaminant fate and transport, and bioaccumulation models for the Hudson River by creating a computational shortcut that reliably predicts mechanistic model outputs from imperfectly known model inputs. By varying inputs to the model emulator (i.e. sediment concentrations and decay rates) within reasonably constrained ranges, the uncertainty distributions of emulated outputs were developed, simulating the uncertainty distributions of the mechanistic model. Importantly, because the mechanistic model is based on linked physical processes thought to be predictive, the model emulator can also be considered to be similarly predictive. The use of model emulation allowed for the investigation of the sensitivity of model outputs to uncertainty in model inputs, including both bias and precision.

4.2.1. Bias

The emulator was used in a deterministic way by modifying model inputs. The resulting mechanistic model forecasts were highly sensitive

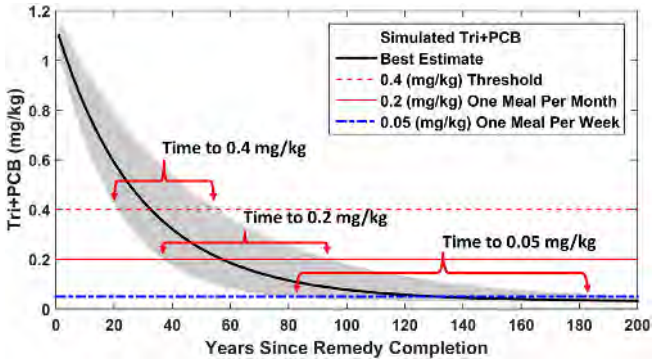

Fig. 9. Monte Carlo distribution of Tri+ PCB concentrations (mg/kg) in white perch at RM152 using the emulated model for the selected remedy with updated sediment concentrations and exponential decay rates in sediment Tri+ PCBs between 2 and 5%.

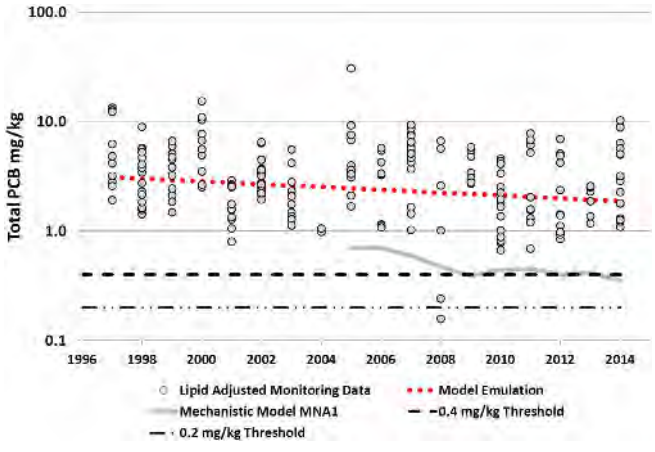

Fig. 10. Emulated model (dotted line) for white perch Tri+ PCB (mg/kg; normalized to 3.0% lipid) from RM152 with 3% exponential decay compared to monitoring data for white perch between 1997 and 2014 (circles) and risk thresholds (0.2 and 0.4 mg/kg PCBs) (horizontal dashed lines). Dredging began in 2009 and was completed in 2015.

to changes (e.g., bias) in initial sediment bed Tri+ PCB concentrations and temporal trend rates, but less so to variation in loads from upstream sources. This paper focuses on the scenario with upstream input concentration decaying to 2 ng/L Tri+ PCBs by 2005, which is consistent with recent monitoring data (USEPA, 2010). The mechanistic models indicated that recovery eventually would be limited with a 2 ng/L upstream baseline load compared to complete source control (upstream load = 0 ng/L). However, the emulated model for 0 upstream load (results not shown) did not differ much from the 2 ng/L model during the emulation period, possibly because initial higher than expected sediment concentrations and lower than expected decay rates mask relatively small differences due to upstream loads.

Updating input sediment Tri+ PCB concentrations to reflect more comprehensive, recent sampling led to the realization that concentrations observed in 2003 sample data exceeded the deterministic upper bound developed from the mechanistic model. Updating input bed sediment concentrations with this new information led to longer estimated recovery times for LHR fish, indicating reduced apparent benefit forecasted for the selected remedy.

The rate of natural recovery is more uncertain than surface sediment concentrations in 2003 because recovery estimates require comparisons with data from older sampling programs, which were based on subjective sampling designs and much smaller sample sizes. Although no completely unbiased sediment sampling program had been conducted prior to 2003, the 1991 UHR transect survey (O'Brien and Gere Engineers, Inc., 1993) was closest to an unbiased systematic sampling study with spatially extensive coverage and many sampling locations distributed throughout the UHR. Lack of unbiased estimates of mean surface concentration at multiple points in time limit the potential to accurately estimate the natural recovery rate. For our study of bias in the decay rates, we used 3% because, while we believe that our sediment decay rate estimate is the best available, the fact that it is based on just two time steps and because only one time step is based on a completely unbiased sampling design, the estimate of 1.3% exponential decay is highly uncertain. Therefore, for evaluating bias, we used 3% as a value that is meaningfully <8%, yet not overly pessimistic. Such subjectivity about sediment recovery rates, at one of the most heavily studied Superfund sites in the United States, is disconcerting and should stimulate a focus on improving the estimate of the rate of recovery at other contaminated sites where remedial alternatives are being evaluated.

4.2.2. Precision

The precision of model forecasts was estimated using a parametric Monte Carlo approach to simulate autocorrelated time series of bed sediment Tri+ PCB concentrations. Sediment concentration inputs were modeled as a first order (i.e. exponential) decay function with temporally correlated residual errors. Application of the model emulator to the 1000 sets of simulated sediment time series resulted in corresponding ensembles of water and fish tissue time series. As discussed above, temporal recovery rates at the Hudson River site are highly uncertain, so the effects of this uncertainty were incorporated into this analysis by simulating first order decay rates as a range of values uniformly distributed from 2% to 5%. This range was chosen subjectively, but nonetheless the analysis illustrated that even modest uncertainty in decay rates can translate into a wide range of estimated times to recovery (Fig. 8). This result indicates that reliable estimates of exponential decay rates in contaminated media are required for reliable remedial alternatives comparisons.

Each of the 1000 simulated time series varies through time around its selected exponential decay rate. When data are strongly correlated temporally, concentration time series may wander far from the exponential decay curve for significant periods of time, leading to greater uncertainty in estimates of time to threshold values. Although results were not shown, the Monte-Carlo procedure was used to evaluate effects of temporal autocorrelation by holding the exponential decay rate fixed across all 1000 simulations. This analysis showed that times to reach threshold concentrations were insensitive to these types of excursions of sediment concentrations due to autocorrelation.

If large linked contaminant fate and transport models are to be used for remedial alternatives evaluation, supporting sediment data appropriate for estimating temporal decay rates are necessary. Frequently, high resolution geochronology sediment cores are used to deduce sedimentation rates and indirectly extrapolate natural recovery rates that are often extrapolated over large spatial regions. However, exposures to biotic receptors are generally assumed proportional to spatial averages, which may not be adequately represented by a small number of high resolution cores. This problem is likely exacerbated by the tendency for investigators to rely on high resolution cores with interpretable geochronology, which typically are collected in low energy areas with continuous deposition and greater than average sedimentation rates that are not representative of site conditions (USEPA, 1998; QEA, 1999b). Those rates, which could be considered to represent an upper bound on sedimentation rates, are then extrapolated over large areas with varying energy regimes and less interpretable geochronologies.

The model emulation approach was useful for quantifying bias and precision of mechanistic model forecasts of fish tissue Tri+ PCB concentrations at the Hudson River. Further application of the method is recommended at contaminated sediment sites where large contaminant fate and transport models have been developed for use in remedial decision-making. Model emulation at other large sites should provide further support for utilizing this approach when additional site data become available to evaluate model projections.

4.3. Improving model calibration and validation

Following the approach used by Castruccio et al. (2014), model emulation can also improve the objectivity and efficiency of model calibration and validation by using a mechanistic model to "pre-calculate" a relatively wide range of model input and output combinations from which a model emulator can be developed. The emulator is then used to iterate on model inputs until optimal combinations of input parameters minimizing error between outputs and sample data are obtained. The emulator provides a mechanism to efficiently calculate combinations of inputs and outputs, allowing many more combinations of model parameters to be evaluated than would otherwise be possible using the mechanistic model directly.

This approach would provide an understanding of the full range of inputs calibrating to the sample data. Combinations of model parameters resulting in similar model fit to data would be considered to represent similarly likely scenarios. If only a small range of model parameters fit the data well, one would conclude that the available data are adequate to uniquely identify the most likely model. In this situation, one could be confident in model projections, whereas a broad range of model parameter combinations resulting in similar model fit to data, would suggest that the sample data are inadequate to uniquely identify a likely model. In this situation, one would not ascribe a great deal of confidence in modeled projections.

4.4. Implications for remedy selection

The model emulation results demonstrate the importance of generating an accurate estimation of both surficial sediment concentrations and the rate of natural recovery of the sediment surface in order for mechanistic models to provide useful information for decision-makers on the relative comparisons among remedial alternatives. If the model-predicted rate of natural recovery is too high, the magnitude of the difference between MNA and an active remedy or between various active remedies, such as the selected remedy for the Hudson River site and a more comprehensive alternative, will be underestimated. USEPA considered two alternative dredging scenarios: the selected remedy (REM1) and a full section removal. The full section removal scenario essentially doubled the area to be dredged (additional 190 ha). According

to USEPA's review of alternatives, full section removal would have been more protective, but the projected difference in fish concentrations (and risk) between the two remedial scenarios was considered too small to warrant the increased cost (USEPA, 2002). The difference between those two dredging alternatives was understated because of the overly optimistic rate of recovery of the surface sediment considered. This is illustrated in Fig. 7, which clearly discriminates between the different alternatives and shows the large difference in time to reach risk thresholds for emulated fish concentrations for the selected dredging remedy (REM1) and for the updated scenario with a more aggressive (but less than full section removal) remedy (REM3). This hypothetical remedy, which maintained the same target cleanup levels for surface sediment throughout the UHR, would involve removing an estimated additional 71 ha, <50% of the area under the full section removal scenario.

While we estimate risk thresholds would be reached meaningfully sooner under this hypothetical and more aggressive remedy (REM3) than under the selected remedy with updated sediment surface concentrations and decay rate (REM2), the estimated time to thresholds would still be longer than the original mechanistic model projections (REM1). Our analysis suggests that achievement of LHR fish PCB threshold concentrations targeted as remedial action objectives to protect human health will be delayed for up to several decades. Our analysis also implies that the remedial action objectives will not be met in the time frame identified in the 2002 ROD for the Hudson River (USEPA, 2002) without implementing a more comprehensive remedy.

Models are often considered to be most useful for evaluating uncertainty in predictions of the relative, as opposed to absolute, benefits for alternative remedial options (Glaser and Bridges, 2007). In such situations management teams may rationalize potential inaccuracies in model forecasts by assuming that relative comparison of forecast remedial effectiveness is possible even when absolute forecasts may be inaccurate or highly uncertain. Our analyses suggest that when models are biased or imprecise the relative differences between remedial alternatives can be significantly under- or over-estimated. In addition, the model emulation approach can serve to improve precision and reduce bias in model output, therefore more reliably discriminating among remedial alternatives. Box and Draper (1987) stated "All models are wrong, some are useful". The models discussed in this paper rely on accurate surface sediment concentrations and the rate of change to make reliable projections of concentrations in sediment, water, and biota. The best way for resource managers and decision-makers to know if the models used for comparing remedial options are useful is to collect systematic, unbiased data on surface sediment concentrations that can be used to estimate the rate of natural recovery and to regularly monitor fish tissues for bioaccumulative contaminants.

5. Conclusions

Our analyses demonstrate that pre-remedial surface sediment Tri+ PCBs in the Upper Hudson River were two to three times higher and estimated post-remediation Tri+ PCBs averaged about four times higher than predicted by the original mechanistic models used by USEPA in the Hudson River 2002 ROD. The rate of recovery, as measured by the exponential decay rate of Tri+ PCBs in surface sediment, was overestimated by the original mechanistic models. We estimated a mean of 1.3% and a 95% upper CI of ~3% compared to the ~8% derived from the original EPA and GE mechanistic models.

The emulated models successfully reproduced the mechanistic model projections for sediment and water in the UHR and fish in the LHR. The emulated models were used to incorporate the updated information on higher surface sediment concentrations and reduced rate of sediment recovery. Our model projections suggest that the original mechanistic model projections greatly underestimated the time to reach risk thresholds in the LHR fish, thereby extending by decades the time period for the project to reach its fish PCB-based remedial action objectives in the LHR.

The results also demonstrated the adverse impact of over-estimation of the rate of sediment recovery on the potential ability of risk managers to discriminate among alternative remedial scenarios.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.scitotenv.2016.02.072.

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Appendix A. Mathematical formulation for emulator

Table A.1 summarizes the locations of the four dams (*River Mile* = d_i), acres of cohesive sediments (A_i), distances between dams ($\delta_i = d_i - d_{i-1}$), area remediated and average distance between deposits and downstream dams ($\overline{d}_i$). Table 2 lists the Tri+ PCB concentrations (c_{si}) in surface sediment in 2003 and 2010 for each of the five scenarios evaluated in this study. The load at the ith dam is represented by L_i and the transfer coefficients from water to sediment and sediment to water are represented by γ_i and g_i respectively. With this notation, the processes for deposition and resuspension at each model annual timestep were described mathematically in the following set of four equations which are nonlinear in the transfer coefficients

$$L_i = L_{i-1} \times (1 - g_i \times \delta_i) + \left\{ \gamma_i \times (c_{si} \times A_i) \times \left(1 - g_i \times \overline{d}_i\right) + \beta_i \times (R_i \times c_{si} \times A_i) \right\} \times Q_i \quad \text{(A.1)}$$

where $i = 1,2,3,4$ indexes each of the four modeled sections of the river, β_i represents the sediment to water net transfer coefficient for dredged residuals and R_i represents the 8% decay of post-dredge residual concentrations. If discharge at successive dams is similar ($Q_i = Q_{i-1}$), Eq. (A.1) can also be expressed in terms of water column concentrations as opposed to loads by dividing both sides of Eq. (A.1) by Q_i giving the following Eq. (A.2).

$$c_{wi} = c_{wi-1} \times (1 - g_i \times \delta_i) + \left\{ \gamma_i \times (c_{si} \times A_i) \times \left(1 - g_i \times \overline{d}_i\right) + \beta_i \times (R_i \times c_{si} \times A_i) \right\} \quad \text{(A.2)}$$

For the Hudson River, results were similar for Eqs. (A.1) and (A.2) so the simpler Eq. (A.2) was used for these analyses.

Each of the 25 years from 2010 through 2034 provides a different set of modeled sediment bed and water column Tri+ PCB concentrations from which the best estimates of emulator net transfer coefficients (g_i, γ_i and β_i, $i = 1,2,3,4$) can be estimated using constrained nonlinear least squares. These paired inputs and outputs from the EPA mechanistic model were available for two remedial scenarios; 1) natural recovery (MNA1), and 2) the selected remedy (REM1A). Each of these scenarios was also simulated with the assumptions of 0 and 2 ng/l PCBs entering from upstream of RS1. Modeled time series spanning 30 (2005–2034) and 25 (2010–2034) year time frames for MNA and active remediation respectively, under two sets of upstream input assumptions and four river sections provided 440 ($2 \times 25 \times 4 + 2 \times 30 \times 4$) nonlinear equations in 12 unknown net transfer coefficients (i.e., g_i, γ_i and β_i, $i = 1,2,3,4$). The transfer coefficients were estimated

Table A.1
Summary of input parameters and initial conditions for calibrating model emulator.

Reach	River section	Downstream river kilometer	River section length (km)	Area (ha)			
				Cohesive sediment area (ha)[a]	Alternative REM1 remediated area[b]	Alternative REM2 remediated area[c]	Alternative REM3 remediated area[d]
Thompson Island Pool	RS1	303.4	10.1	42	114	124	124
Schuylerville	RS2	295.2	8.2	54	31	35	56
Stillwater	RS3A	270.7	24.5	93	38	29	64
Waterford	RS3B	263.1	7.6	52	17	13	28
Total					200	201	272

[a] Cohesive sediment area from Tables 5.2a–5.2b in USEPA (2000b).
[b] Area for alternative REM1 from Tables 8–9 in USEPA (2000a).
[c] Alternative REM2 area calculated based on delineated dredge area.
[d] Alternative REM3 area based on delineated dredge area for the selected remedy and additional area estimated from number of cores exceeding RS1 target cleanup levels.

Table A.2
Estimated model emulation nonlinear regression coefficients.

Model coefficients	River section			
	RS1	RS2	RS3A	RS3B
Water to sed	0.0000	0.0350	0.0157	0.0641
Sed to water	0.0160	0.0095	0.0078	0.0451
Post dredge resuspension	0.0251	0.0143	0.0283	0.0357

by constrained nonlinear least squares with MATLAB© Release 2011a (The MathWorks 2011).

Appendix B. Probability model for synthetic sediment time series

The residual process $C_i(t) = C_{0i}e^{-kt+\varepsilon_i(t)}$ was simulated by randomly drawing an exponential decay rate (k) from a uniform probability distribution on the interval 0.02–0.05, followed by simulation of $\varepsilon_i(t)$ as a mean zero normally distributed random variable with covariance matrix $\boldsymbol{C}$ with the entries c_{ij} defined as $cov(\varepsilon_i(t),\varepsilon_i(t+h)) = e^{-a_i h2}$, and covariance between subsections i and j given by $cov(\varepsilon_i(t),\varepsilon_j(t)) = c_{ij}$ for $i \neq j$. The constants a_i and c_{ij} were estimated from the four mechanistic modeled sediment Tri+ PCB concentration time series. The expected mean of the simulated sediment series for the ith subsection is $C_{0i}e^{-kt}$. The simulated series are distributed log-normally because $\varepsilon_i(t)$ is a normally distributed random variable.

The estimated coefficient a_i defining the rate of decline in temporal auto correlation was 0.1. The resulting correlation matrix C was a real symmetric banded matrix with diagonal entries $C_{ii} = 1.0$ and with 5 non-zero off diagonal with values $C_{i,i\pm j} = 1$, 0.90, 0.67, 0.41, 0.20, and 0.08; for $j = 1, 2, \ldots, 5$ respectively and $i = 1, 2, 3, \ldots, 200$ years. The remaining values $C_{i,i\pm j} = 0$; for $j > 5$.

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ATTACHMENT B

Attachment B

Responses to Main Themes in NYSDEC's August 30, 2017 Comments

On August 30, 2017, the New York State Department of Environmental Conservation (NYSDEC) submitted a cover letter and comments on EPA's *Proposed Second Five-Year Review Report for Hudson River PCBs Superfund Site* (Second FYR). Those submittals contain several assertions that are unsupported by the available information and warrant correction.

As a brief introductory observation, the post-dredging data collection process currently underway and relied on in the Second FYR is precisely what EPA contemplated in the ROD, which required extensive monitoring for decades in order to determine recovery of the river and the success of the remedy. The ROD recognized that several years of post-dredging data will be needed to assess decline rates, and that only then can a rational assessment be credibly made of the need for any potential additional remedial work. NYSDEC's comments and arguments appear to reject this long-established process. Instead, they reflect a prejudgment that the remedy has failed, in disregard of the data collected to date, which show that recovery has begun, and in advance of collecting the necessary additional data, as contemplated by the ROD.

The Remedy Is Meeting the Expectations of the ROD

NYSDEC's principal claim is that the dredging remedy that was implemented will not achieve its ultimate objective for more than 55 years, which is "unacceptable" and "not what the people of the State of New York were promised when EPA announced its remedial decision for the Hudson River in 2002." At that time, NYSDEC asserts, "EPA predicted that the dredging remedy would result in rapid reductions in PCB levels in fish so that fish consumption restrictions could be relaxed in five to ten years." According to NYSDEC, EPA has now abandoned the ROD's interim PCB targets of 0.4 and 0.2 ppm in fish, which would allow for such relaxation, and thus has undermined an important goal of the ROD. NYSDEC contends that this makes the remedy unprotective, since the "current human health and ecological risks . . . are well in excess of EPA's acceptable risk range."

This claim is wrong and inaccurate on several levels. The ROD clearly recognized that it would take some time to achieve the interim targets of 0.4 and 0.2 ppm. EPA's model projections at that time indicated achievement of those interim targets would take several years after completion of the remedy, ranging from 16 to over 59 years in River Section (RS) 1 and RS 2, with lesser times in RS 3 (ROD, pp. 72-73). Moreover, the specific times presented in the ROD to reach the various target levels in fish were presented for the purpose of comparing the relative effectiveness of the remedial alternatives, not as absolute predictions of those time periods. The ROD further recognized that, until the ROD's ultimate objective of 0.05 ppm PCBs in fish was met, exposure to PCBs would be

controlled, to the extent practicable, through fish consumption advisories and fishing restrictions, and that where those controls would not eliminate exposure, the human health and ecological risks would remain above EPA's acceptable risk range. The ROD nevertheless found the remedy to be protective of human health and the environment, and NYSDEC concurred.

As shown in GE's main comments, the current information demonstrates that the remedy is functioning as anticipated in the ROD. Contrary to NYSDEC's contention, EPA has not abandoned the ROD's interim targets, but continues to evaluate the data in relation to those targets (see Second FYR, p. 45 & Appendix 3, p. 6-3). And NYSDEC's prediction that the remedy will not achieve those targets is speculative and based on only one year of data (from 2016), when the river had not yet fully recovered from the effects of dredging. As EPA recognizes, several years of monitoring are necessary to evaluate the rate of recovery in the river. NYSDEC's prediction based on one year of data reflects an unjustified prejudgment of the monitoring results. It is essentially attempting to require a re-initiation of the remedy selection process, which is not the purpose of a five-year review.

Current River Conditions

NYSDEC asserts that "EPA appears desperate to come to a conclusion which simply is not supported by the current conditions of the Hudson River," and "[i]t is obvious that the remedy is not protective of public health and the environment."'

Again, this claim ignores the fact that the information to date indicates that the remedy is performing as expected in the ROD. As shown in GE's comments, current conditions are as expected and indicate that the river is beginning to recover. Thus, the ROD's conclusion on protectiveness remains valid at this time. Long-term monitoring of fish, water, and sediment will be necessary to evaluate the river's rate of recovery and thus to determine the long-term protectiveness of the remedy.

Amount of PCBs Remaining in River

NYSDEC argues that, "because greater levels of PCBs were found in the river during project design, and again during project implementation, significantly more PCBs were left behind than was intended when EPA selected [the] remedy." It states further that "EPA has never considered adjusting the remedial work to take the increases in known PCB mass into account . . . . "

As shown in GE's main comments (Section 2.1), the remedy used numerical removal criteria so that it could be adapted to the new data collected after the ROD and before design and scaled to those results if more or fewer PCBs were found. Moreover, the fact that the ROD underestimated the PCB mass in the dredge areas does not mean that it underestimated the mass in non-dredge areas. In fact, as shown in GE's comments (Section 6.1 and Attachment C), GE has estimated that the amount

of PCBs remaining in the non-dredge areas is very comparable to the amount estimated in the ROD to be left in those areas.

Scope of Additional Sampling

NYSDEC contends further that EPA has refused to collect sufficient monitoring data to evaluate the remedy's effectiveness, and that "EPA's persistent refusal to collect and analyze a full array of data has run counter to EPA's original commitment to clean up the site."

Contrary to this assertion, EPA and GE have implemented a comprehensive monitoring program that will effectively allow assessment of the remedy effectiveness. This long-term monitoring program was developed by EPA with input from NYSDEC and was embodied in the Consent Decree covering this remedy. The additional sampling that NYSDEC has requested goes beyond that established program and appears to be more focused on gathering data to aid in design of another dredging project. In addition, EPA has conducted a statistical analysis of the data needed to evaluate the remedy's effectiveness and the recovery of the river, and determined that the current program is adequate for that purpose. NYSDEC's claims regarding additional sampling are based largely on its view that such sampling is necessary to evaluate the remedy at the scale of each river reach or pool. However, as EPA recognizes, that is not necessary to assess the recovery of the river. Expectations for recovery on which EPA based the remedy were at the scale of River Sections, not reaches, as shown in Figures 6-21 through 6-23 of the Feasibility Study. That is thus the appropriate scale to assess recovery of the river.

Lower Hudson River

NYSDEC asserts that the "Lower Hudson River is contaminated with PCBs from the Upper Hudson River" and "the remedial work in the Upper Hudson River to date will not result in any significant reductions in public health and environmental risks," and that thus "[t]here is no longer any reason to delay the Lower Hudson River investigation."

As shown in GE's comments (Section 6.4), the existing data demonstrate that the dredging project did and will continue to benefit the Lower Hudson. As also shown in GE's comments (Section 5.1 and Attachment A), a model developed by the National Oceanic and Atmospheric Administration (NOAA), which the additional dredging advocates rely upon to argue that the fish in the Lower Hudson will recover at a much slower rate than predicted in the ROD, is demonstrably invalid for a number of reasons, including the fact that it fails to mimic actual data. However, as with the Upper River, it is critical to continue to obtain monitoring data to evaluate trends in PCB levels in fish and water in the Lower River before the need for other response actions can be assessed. That has always been part of the remedy, and GE has been collecting these data and will continue to do so. That is the appropriate approach at present to addressing the Lower River.

ATTACHMENT C

Attachment C

Estimating PCB Mass in the Upper Hudson River

Overview

In issuing the Record of Decision (ROD) for remediation of the Hudson River PCBs Superfund Site (EPA 2002), the U.S. Environmental Protection Agency (EPA) considered the mass of polychlorinated biphenyls (PCBs) to be removed by the dredging. The EPA Responsiveness Summary that was part of the ROD provided an estimation of the PCB mass to be remediated and the PCB mass that would remain after the project's completion. During remediation, the estimate of PCB mass removed was used to assess project efficiencies for both phases of the remediation and to track compliance with the Engineering Performance Standards (EPS) during Phase 2 (EPA 2010). After the completion of remedial activities, understanding the amount of PCB mass remaining relative to that which was removed and capped/backfilled provides insight on the overall project success.

The ROD indicated that the selected remedy would remove 69,800 kilograms (kg) of PCBs, or 65% of the total PCB mass estimated to be in the river. The ROD also estimated that 37,500 kg would remain in the non-dredge areas. The General Electric Company's (GE's) analysis of PCB mass data, conducted after the completion of remedial activities, has indicated that 145,890 kg were removed by dredging, 3,910 kg remain in dredged areas underneath backfill or an engineered cap, and between 34,530 kg and 37,900 kg remain in non-dredge areas. These estimates translate into the removal of 78% to 79% of the PCB mass that was present in the river before dredging, which is similar to, or better than, the PCB mass removal efficiency predicted in the ROD. The basis for GE's estimate is presented below.

PCB Mass Removed

During Phase 1, total PCB mass removed was calculated after each dredging pass. This information was used to evaluate mass removal efficiency of each pass, and to support resuspension analyses. Cores collected prior to dredging (i.e., the Sediment Sampling and Analysis Program [SSAP] dataset) were used to calculate the PCB mass removed during the design dredge cut. Cores collected after each subsequent dredge cut were used to calculate the PCB mass removed during each re-dredge cut. Methods used to calculate mass were consistent with the approach used in the Dredge Area Delineation (DAD) Reports (QEA 2005, 2007), which used Thiessen polygons combined with mass per unit area (MPA)[1] and accounted for factors such as spatial variability in PCBs, bulk density variations,

[1] PCB MPA consists of the grams of PCBs per square meter of sediment surface area of a core. This is calculated by summing the products of the PCB concentration, length, and bulk density of each core segment. Further information on the calculation of MPA can be found in the Hudson River DAD Reports (QEA 2005, 2007).

and cores with PCB profiles that do not reach clean sediment. A summary of the PCB mass removed during Phase 1 activities was provided in Attachment H to GE's Phase 1 Evaluation Report (Anchor QEA and Arcadis 2010). By GE's estimate, 18,230 kg of PCBs were removed during Phase 1 dredging[2] (see also the GE's Remedial Action Completion Report; Parsons 2016b).[3]

After the completion of Phase 1, an independent peer review panel provided recommendations, which guided the development of the EPS for Phase 2. The 2010 EPS specified performance standards for dredging residuals, resuspension, and productivity in Phase 2 (EPA 2010). The approach to mass removed outlined in the 2010 EPS was different from that for Phase 1 in that it divided the roughly 5-acre Certification Units (CUs), which were used for assessing compliance with the EPS and evaluating dredging completion and cover type, into subareas and used all the cores within a subarea to establish a representative mass per unit volume. Specifically, the 2010 EPS directed GE to "estimate mass of dry PCBs per unit volume of wet-sediment for each CU and multiply by the volume of wet-sediment to obtain an estimate of total mass of PCBs removed" (EPA 2010, Section 7.3, page 7-2). Similar to Phase 1, pre-dredge cores (from the SSAP and Supplemental Engineering Data Collection [SEDC] programs) were used in the calculation of the mass per unit volume during the design dredge cut, while post-dredging data (i.e., residual core data) were used during subsequent cuts. The volume of sediment removed was based on the difference between pre- and post-dredging bathymetric surveys. Details of the calculations, including equations, are outlined in Section 7 of the 2010 EPS and further qualified in Section 2.1.2 of each year's Phase 2 Performance Standard Compliance Plans (PSCPs) (GE 2011, 2012, 2013, 2014, 2015). The mass removed each year was reported in GE's annual Phase 2 reports (Parsons 2012, 2013, 2014, 2015, 2016a). By GE's estimates, 127,660 kg of PCBs were removed during Phase 2 dredging (Parsons 2016b).[4]

PCB Mass Remaining in the Target Areas

The PCB mass remaining in the CUs after dredging (i.e., mass capped or backfilled in place) was estimated consistent with the approaches described above for Phases 1 and 2 (i.e., using Thiessen

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[2] As noted above, the mass removed in the design pass (i.e., the first dredging cut in each CU) in Phase 1 used only the SSAP data. However, Appendix H of GE's Phase 1 Evaluation Report presented a sensitivity analysis to understand the impact of the incorporation of the SSAP data into the mass removed estimates from the re-dredge pass(es). Scenario 1 used a combination of all SSAP and residual data, Scenario 2 included residual data and SSAP cores with high confidence in the depth of contamination, and Scenario 3 used only the residual data. These three scenarios resulted in estimates of 16,320, 17,110, and 18,235 kg of PCBs removed during Phase 1, respectively. After a review of these results, GE and EPA agreed to follow the Scenario 3 approach (residual data only) for the mass removal and mass remaining analyses. Therefore, GE uses 18,235 kg to characterize the mass removed during Phase 1 dredging.

[3] While the ROD, annual reports, and the Completion Report show mass estimates on Tri+ and Total PCB basis, all PCB masses presented in this attachment are on a Total PCB basis.

[4] GE's Remedial Action Completion Report lists the estimate of PCB mass removed in Phase 2 as 127,785 kg. The slight difference between that estimate and the estimate used herein is the result of slight modifications during quality assurance/quality control of the calculations.

polygons for Phase 1 and the mass per unit volume approach for Phase 2). For all dredging years, the PCB mass remaining was estimated using the data from the last post-dredging core collected at each residual node.

In Phase 1, Thiessen polygons were used to represent the area of influence of each residual core location. Each polygon was assigned the MPA of its core to estimate PCB mass remaining after dredging. A total of 1,570 kg of PCBs was estimated as remaining in the Phase 1 CUs, and subsequently covered with a cap or backfill, after Phase 1 dredging (Anchor QEA and Arcadis 2010, Appendix H, Table H-2).

For Phase 2 CUs, the PCB mass remaining was estimated using the mass per unit volume approach described in the PCB Mass Removed section above with one exception: The volume of un-dredged inventory was estimated using the data from the last core sampled at each residual node, as opposed to using bathymetric survey data.[5] Details of the calculations, including equations, are outlined in the 2010 EPS and subsequent PSCPs (GE, 2011, 2012, 2013, 2014, 2015). Using this approach, 2,340 kg of PCBs was estimated as remaining in the Phase 2 CUs after dredging and was covered with backfill or an engineered cap.

PCB Mass in Non-Dredge Areas

To assess the removal efficiency of the project, the PCB mass that exists in non-dredge areas was also estimated.

Data Usage

To estimate the areal extent of non-dredge areas in the Upper Hudson River, the spatial polygon of the full river, as developed during design, was used. This polygon was processed to exclude the dredge areas and all areas not within the main stem of the river, such as tributaries, the Coveville backwater area, and navigational land cuts. The non-dredge area map was then spatially merged with the 2003 side-scan sonar map of sediment type to partition the non-dredge areas by primary sediment type. Sediment types include fine-grained material, sand, gravel, transitional material, and rock; and areas not covered by the side-scan sonar data were deemed unclassified. Rock areas were treated as having little to no PCBs. A summary of the non-dredge area acreage by sediment type and river section is provided in Table 1.

The total PCB MPA was calculated for each of the design sediment locations (i.e., SSAP and SEDC cores) sampled in the non-dredge areas. Locations at which no sediment was recovered and probing indicated less than 6 inches of sediment present were assigned a total MPA of zero, consistent with

[5] The volume of un-dredged inventory was estimated using the depth of contamination (the depth from the surface of the sediment to the point at which the Total PCB concentration is less than 1 milligram per kilogram [mg/kg]) of the last residual core collected at each residual node, multiplied by the area of influence of each node, as defined using Thiessen polygons.

data treatments outlined in the DAD Reports. A summary of the number of data points used to estimate non-dredge PCB mass is provided by river section in Table 1.

Table 1
Extents and Data Counts used in Non-Dredge PCB Mass Evaluation

River Section	Sediment Type	Non-Dredge Extent[1,2] (acres)	Non-Dredge Location Count[3]
1	Silt	15	142
	Transitional	92	563
	Sand	35	255
	Gravel	30	87
	Unclassified	16	23
	Total for RS 1	***189***	***1,070***
2	Silt	92	503
	Transitional	32	93
	Sand	101	293
	Gravel	49	52
	Unclassified	33	5
	Total for RS 2	***307***	***946***
3	Silt	318	1,909
	Transitional	264	319
	Sand	700	345
	Gravel	726	102
	Unclassified	439	248
	Total for RS 3	***2,447***	***2,923***
Total for All River Sections		**2,943**	**4,939**

Notes:

1. Areas identified as rock by the 2003 side-scan sonar survey were excluded.
2. Only areas in the main stem of the river are included in this acreage.
3. Abandoned locations with 6 inches of sediment or more were excluded.

RS: River Section

Estimating Average MPA

PCB mass remaining in the non-dredge areas was estimated using an area-weighted average MPA approach. The total PCB MPA for each location was calculated in two ways – one using the measured PCB concentrations in the cores, and the other using the measured concentrations plus, for cores that did not penetrate to clean sediments, an extrapolation developed to estimate any missed PCBs

below the cores in accordance with procedures presented in the DAD Reports.[6] For each sediment type within a River Section, an average MPA was calculated under each of these methodologies by area-weighting the MPAs for each location within that sediment type.

The area of influence for each sampling location was determined using Thiessen polygons. The polygons were clipped to sediment type boundaries as defined by the side-scan sonar. For example, the polygons of sampling locations in fine sediment were not permitted to extend beyond the fine sediment boundary. The area of each Thiessen polygon was used to weight the associated MPA for the purpose of calculating an average MPA for a sediment type within a river section. Areas remote from the data points were flagged as orphan areas and were not included in the area-weighting calculation. To calculate total PCB mass, orphan areas were assigned the area-weighted average PCB MPA.[7] The area-weighted average total PCB MPA for each river section and sediment type is summarized in Table 2.

Table 2
Area-Weighted Average Total PCB MPA by River Section and Sediment Type

River Section	Sediment Type	Area-Weighted Average Total PCB MPA (g/m^2)	
		Using Measured Data Only	Using Measured and Extrapolated Data
1	Fine	2.1	2.1
	Transitional	1.2	1.3
	Sand	1.3	1.3
	Gravel	2.0	2.1
	Unclassified	0.8	0.9
2	Fine	5.5	5.6
	Transitional	2.7	2.7
	Sand	4.3	4.5
	Gravel	2.4	2.6
	Unclassified	0.02	0.02
3	Fine	7.0	7.5
	Transitional	2.3	2.5
	Sand	2.1	2.2
	Gravel	1.0	1.2
	Unclassified	4.8	5.6

[6] While MPA calculations for extrapolated data followed data treatments outlined in the DAD Reports, data from paired data gap core locations were not excluded in this analysis.

[7] The area-weighted MPA in a given river section and sediment type was assigned to orphan areas of the same sediment type within the same river section. In this way, all non-dredge areas within the main stem of the river were accounted for in the non-dredge PCB mass calculation, except for bedrock areas which were assumed to have no PCBs.

Percent of Total PCB Mass Removed and Mass Remaining in Non-Dredge Areas

The total PCB mass in the non-dredge areas was calculated by multiplying the area-weighted average total PCB MPA in each river section and sediment type by the non-dredge area of that sediment type. These calculations indicate that a total of 34,530 to 37,900 kg of PCBs (depending on the approach used for cores that did not reach clean sediments) is estimated to remain outside the CUs (Table 3).

Table 3
Non-Dredge Mass by River Section and Sediment Type

River Section	Sediment Type	Non-Dredge Mass (kg)	
		Using Measured Data Only	Using Measured and Extrapolated Data
1	Silt	130	130
	Transitional	440	470
	Sand	180	180
	Gravel	270	280
	Unclassified	60	70
	Total for RS 1	***1,080***	***1,130***
2	Silt	2,040	2,100
	Transitional	350	360
	Sand	1,770	1,830
	Gravel	440	480
	Unclassified	<5	<5
	Total for RS 2	***4,600***	***4,770***
3	Silt	9,000	9,600
	Transitional	2,500	2,660
	Sand	5,880	6,260
	Gravel	2,820	3,490
	Unclassified	8,650	9,990
	Total for RS 3	***28,850***	***32,000***
Total for all River Sections		**34,530**	**37,900**

Note:
RS: River Section

Combining these estimates with the above-discussed estimates of the mass removed and the mass remaining within the CUs indicates that 78% to 79% of the PCB mass in the Upper Hudson River was removed during dredging (Table 4). These estimates are similar to or better than the PCB mass removal efficiency reported in the ROD. The ROD indicated that the selected remedy would result in

the removal of 65% of the total PCB mass in the river, and it estimated that a total of 37,500 kg would remain in the non-dredge areas (Table 4).

Table 4
Summary of River-Wide Total PCB Mass

River Section	ROD Estimate[1]			Post-Remedy Evaluation			
	PCB Mass Remediated (kg)	Non-dredge Mass (kg)	% PCB Mass Removed	PCB Mass Removed (kg)	Mass Capped/ Backfilled (kg)	Non-dredge Mass (kg)	% PCB Mass Removed
1	36,000	9,200	80%	84,360	2,860	1,080-1,130	95-96%
2	24,300	3,800	86%	32,380	510	4,600-4,770	86%
3	9,500	24,500	28%	29,150	540	28,850-32,000	47-50%
Total	**69,800**	**37,500**	**65%**	**145,890**	**3,910**	**34,530-37,900**	**78-79%**

Note:
1. Per Table 363334-1 in the ROD Responsiveness Summary.

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HUDSON
DEVELOPMENT
CORPORATION

August 24, 2017

Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski:

We represent businesses along the 200-mile span of the Hudson River Superfund site. The river is the bedrock of the Hudson Valley's current and future economic vitality. It drives the region's multibillion-dollar tourism industry and is in large part responsible for the ongoing recovery of the real estate market in the Lower Hudson since the great recession. The beauty of the river and the myriad parks along it contribute significantly to residents' quality of life and serve as catalysts for attracting visitors and new jobs.

Building upon this momentum depends on a clean, healthy Hudson River. As long as unacceptable levels of PCBs pollute its water, sediment and fish, they hinder lasting economic gains—both the resumption of once-lucrative industries dependent on the river and long-stalled development opportunities along it. More important, they continue to pose a threat to the health of people living in riverfront communities.

For 70 years, the economic, recreational, cultural and scenic values of the Hudson River have been compromised by PCB contamination. This pollution has destroyed a once-vibrant commercial fishing industry, hampered the operation of marinas, led to a severe curtailment of marine transport on the Champlain Canal, tripled the costs of dredging the NY-NJ Harbor, prevented ambitious economic development opportunities on the Upper Hudson similar to those being realized along the Mohawk River, and barred generations of residents and visitors from full enjoyment of this American Heritage River.

For these reasons, we call on the EPA to:

Declare in its Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as the EPA's draft review explicitly states.

Delete the draft review's finding that the remediation "will be protective" in 53 years. The EPA makes this forecast despite admitting eight additional years of research are needed to verify it. Further, data indicate that fish toxicity in the Upper Hudson is almost 300 percent higher than the goal the EPA expected to reach in 2018. If this interim target is so off base, how can the EPA forecast with any reliability that the cleanup "will be protective" in five decades?

Conduct additional cleanup of the Upper Hudson. The draft review fails to incorporate any analysis by the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation showing that the remediation leaves behind contamination equivalent to (in NOAA's words) "a series of Superfund-caliber sites." Both NOAA and the DEC have concluded that additional dredging is needed. An "is not protective" determination will pave the way for this to happen.

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HUDSON DEVELOPMENT CORPORATION

Undertake a remedial investigation of the Lower Hudson. The draft review makes clear that PCB levels in fish and sediment in the 160-mile portion of the Lower Hudson have not benefited at all from upriver dredging. In actuality, downriver contamination is significantly higher than expected. The draft review lays out no plan for investigating and removing this contamination. This oversight must be corrected.

Data confirm that time and nature won't fix this project's shortcomings, as your draft review would lead us to believe. Only additional dredging will make the Hudson healthy as soon as possible. Therefore, we strongly urge the EPA to conclude that the remedy for the entire Hudson River Superfund site is "not protective." Then and only then can we begin to plan for the bright future our children and grandchildren deserve.

Sincerely,

Sheena Salvino

Executive Director

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Mohawk Maiden Cruises LLC

August 30, 2017

Gary Klawinski
Hudson River Field Office
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski,

My name is Marla Hodge. I'm co-owner of Mohawk Maiden Cruises, a tour boat company on the Champlain Canal, and I'm also a homeowner who lives on the banks of the original canal.

I have many concerns about the current state of the Champlain canals, both original and current, but in this letter I'd like to speak concentrate on two of those.

I run my tours from Lock C5 and travel the land cut section between Lock 5 and the Northumberland Falls on almost a daily basis in the summer. That section was not touched by the GE dredging, but my understanding is that the state is restricted in its ability to dredge that section because, who knows what's down there?

I can tell you that that particular section is only about 10 feet deep, and I know that because when I watch those huge barges come south, they're loaded to 10 feet of draft, and every time I see clouds of mud kicked up as they drag the bottom. Not only does that restrict potential commerce from using the canal, you have to wonder what kind of PCBs and other contaminants are being added to the water, sent through the lock and down into the river? I think that needs to be addressed.

My second, and biggest, concern is that the original Champlain Canal is a health hazard, not even to mention the loss of tourism potential. I'd love to put some type of historical attraction there. But many parts are completely stagnant, including a section right behind the new regional visitor's center. It's a breeding ground for

mosquitoes and other insects. It stinks. The recent rains have shown how easily its banks overflow. When we had that huge rainfall July 4 weekend, I drove by and saw water pouring from the original canal straight down to Fort Hardy Park, where all the kids sports are played. Who knows what our kids are now being exposed to?

There are plenty of local folks who would clean that original canal in a heartbeat if we could - but we can't. We can't touch it, because we don't know what's in there.

I'd like to know why EPA, GE, whoever, can't get this tested. I don't know why it wasn't part of the original agreement, because, at least until it silted up, river water flowed right through the original canal and back out into the Hudson - flowing from the same place where all the other pcbs and other industrial contaminants came from.

Right now our hands are tied - until the EPA steps up and makes sure we at least know what's in there. Our kids' futures - and really, their lives, depends on it.

The other day I made the comment to someone that, as much as I hated the dredgers when they were here - and I utterly despised them - if they need to come back to make sure the job is finished, I'll welcome them. (I'm sure I'll still hate them, but I'll welcome them!)

I don't pretend to know the answer the situation, but this is what I do know: the job is not complete, and the EPA needs to make sure that it is!

In response to the Five-Year Review Questions:

1. Is the remedy functioning as intended by the decision documents?
   a. It is clear from the initial information on habitat and fish samples that it is taking longer for recovery than anticipated. PCB's were far deeper and more dispersed than the ROD anticipated.
   b. Habitat reconstruction has not resulted in repopulation of species within the parameters that the ROD anticipated.
   c. Resuspension and down river redisposition of sediments into the flood plains has not been addressed.

2. Are the exposure assumptions, toxicity, data, cleanup levels, and remedial actions objections used at the time of the remedy selection still valid?
   a. The variability of testing methods has tainted the results to date.

b. The ROD left behind significant deposits throughout the upper Hudson that are not part of the cleanup. Those deposits are in excess of standards used in other PCB cleanup projects and leave our river subject to additional cleanup costs every time we attempt a project - whether residential or public.

3. Has any other information come to light that could call into question the protectiveness of the remedy?
   a. The original Champlain Canal was not included in the remedy and it is hydrologically part of the Hudson River. Significant PCB concentrations were found and partially removed from the canal north of Lock 5, yet the original canal was ignored. The original canal is now so silted in with blocked culverts and dead fall that it is often stagnant and overflows the banks during heavy storms.
   b. The ROD ignored the industrial and recreational use of the river when it required dredging only to the depth of the contamination - ignoring the fact that New York State has been unable to dredge to required depths for decades. Additionally, the EPA (with the ROD as an excuse) refilled areas that had silted in over the decades - impeding industrial and recreational use.
   c. The ROD focused on river sections closer to Fort Edward, ignoring contamination of the same toxicity in river sections below Lock 5. Those areas will continue to redeposit PCB's in the upper river, the flood plains and the lower river.

For these reasons - I urge the EPA to recognize that the remedy as designed is not protective. Additional dredging is required if those of us in the upper Hudson are to have a clean river. We cannot undertake projects and use of our river with the knowledge that the legacy of PCB's is still lurking in the sediments and floodplains.

Sincerely yours,

Marla A. Hodge
Master Captain, Owner
Mohawk Maiden Cruises

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31 AUG 2017 PM 3 L

Gary Klawinski
Hudson River Field Office
18A Wolfe Rd, Suite 303
Albany NY

12205-110089 12205

FW: Hudson River PCB Cleanup

Klawinski, Gary J <Klawinski.Gary@epa.gov>

Wed 9/6/2017 9:47 AM

To: 'epahrfo@outlook.com' <epahrfo@outlook.com>;

From: Seaweed Yacht Club [mailto:seaweedyachtclub@gmail.com]
Sent: Monday, August 28, 2017 6:51 PM
To: Klawinski, Gary J <Klawinski.Gary@epa.gov>
Subject: Hudson River PCB Cleanup

Hello, Mr. Klawinski,

I have read many articles and letters citing facts about the Hudson River PCB cleanup not having been completed by the standards set at the onset of the project. I won't re-state what has already been stated by others far better versed than I on the topic.

I ask on behalf of many that for future generations you ensure the cleanup will continue. This matters.

Thank you for taking a moment to read this short, but sincere, message. Thank you, in advance, for your help in demanding that those who have irresponsibly and catastrophically harmed our environment are responsible for restoring it back to optimal health - no matter how long it takes.

Janice Anderson

--

Janice Anderson
Commodore, Seaweed Yacht Club
Director, Hudson River Boat & Yacht Club Association

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August 7, 2017

Gary Klawinski
Director, Hudson River Field Office
U.S. Environmental Protection Agency
187 Wolf Road, Suite 303
Albany, NY 12205

Dear Mr. Klawinski:

My name is Wiley Harrison. I am a small business owner for 25 years, employing 15 full time Bookkeepers and Accountants. I am also a landlord, one of my properties is located on the Hudson River in Yonkers, NY. I am also very involved in the economic development at various levels within my community including the Governor's Mid-Hudson Regional Economic Development Committee and the Industrial Development Agency for Westchester County, NY

I believe I represent businesses along the 200-mile span of the Hudson River Superfund site. The river is the bedrock of the Hudson Valley's current and future economic vitality. It drives the region's multibillion-dollar tourism industry and is in large part responsible for the ongoing recovery of the real estate market in the Lower Hudson since the great recession. The beauty of the river and the myriad parks along it contribute significantly to residents' quality of life and serve as catalysts for attracting visitors and new jobs.

Building upon this momentum depends on a clean, healthy Hudson River. As long as unacceptable levels of PCBs pollute its water, sediment and fish, they hinder lasting economic gains—both the resumption of once-lucrative industries dependent on the river and long-stalled development opportunities along it. More important, they continue to pose a threat to the health of people living in riverfront communities.

For 70 years, the economic, recreational, cultural and scenic values of the Hudson River have been compromised by PCB contamination. This pollution has destroyed a once-vibrant commercial fishing industry, hampered the operation of marinas, led to a severe curtailment of marine transport on the Champlain Canal, tripled the costs of dredging the NY-NJ Harbor, prevented ambitious economic development opportunities on the Upper Hudson similar to those being realized along the Mohawk River, and barred generations of residents and visitors from full enjoyment of this American Heritage River.

For these reasons, I call on the EPA to:

Declare in your Final Five-Year Review that the PCB cleanup "is not protective" of human health and the environment—as your draft review explicitly states.

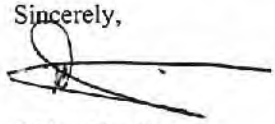
Delete the draft review's forecast that the remediation "will he protective" in 53 years. You make this assumption despite your own admission that it will take eight additional years of research to verify.

Conduct additional cleanup of the Upper Hudson. Both the National Oceanic and Atmospheric Administration and New York State Department of Environmental Conservation have concluded that without more dredging, it will take a century or longer for the Superfund project to achieve its goals. An "is not protective" determination will pave the way for the cleanup to continue.

Undertake a remedial investigation of the Lower Hudson. The draft review admits that upriver dredging has had no effect on PCB contamination in the Lower Hudson—in fact, it is significantly higher than expected. The final review must lay out a plan for investigating and removing this contamination.

Data confirm that time and nature won't fix this project's shortcomings, as your draft review would lead us to believe. Only additional dredging will make the Hudson healthy as soon as possible. Therefore, we strongly urge the EPA to conclude that the remedy for the entire Hudson River Superfund site is "not protective." Then and only then can we begin to plan for the bright future our children and grandchildren deserve.

Sincerely,



Wiley Harrison

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Wayne T. Senecal
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United Campus Holdings Company, LLC

Saratoga Office
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MEMORANDUM

TO: Gary Klawinski, Project Director EPA Region 1

CC: Catherine McCabe, EPA Acting Regional Administrator; Basil Seggos, NYS-DEC Commissioner; Senator Kirsten Gillibrand: New York Attorney General Eric Schneiderman; Peter Sheehan, Sierra Club Hudson-Mohawk Group; Stephen Williams, Reporter, The Daily Gazette

RE: The Hudson River PCB Dredging Project - The Case Against General Electric and the Individual Responsible Executives for Gross Negligence & Willful Misconduct in Their Selection of the Cheaper, but Totally Inappropriate Dredging Method to Remediate the Super Fund Designated PCB Pollution of the Hudson River

I. Introduction

The issue of General Electric's responsibility for cleaning up the PCB's they dumped into the Hudson River has long been clear, and their attempt to clean up that pollution has long been underway, and supposedly successfully completed. However, the New York State Department of Environmental Conservation, the Sierra Club and others have contested the EPA's conclusions with regard to the quality and successful conclusion of that cleanup.

The purpose of this memorandum is to call on the EPA to use its authority granted under the Super Fund Legislation to pursue General Electric and the individual responsible G.E. Executives for Gross Negligence and Willful Misconduct in executing the EPA's Cleanup Directive. The basis for that charge being their selection of a totally inappropriate dredging method, (i.e. using a "Grab" Dredger instead of a "Cutter-Suction" Dredger,) solely in order to save costs!

II. Discussion

Having had in my early career with Citibank and the Archirodon Group of Companies, nearly 15 years of experience in the Heavy Marine Construction and Dredging fields, I gained a good understanding of the various types of dredgers and the types of materials they were best suited to remove. "Grab" Dredgers, being one of the oldest and cheapest of dredging methods, are best suited for the removal of semi-hard and hard, (i.e. rocky) materials. Fundamentally being a large floating crane with a heavy, clam-shell type bucket, a "Grab" Dredger uses its weight and the teeth of the bucket to crash into, bite, grab, and haul off hard material. The PCB sludge dumped by G.E. on the river bottom of the Hudson River was a much more soft and fluid type of material, which is the type of

material for which a "Cutter-Suction" Dredger is ideally suited and for which a "Grab" Dredger is totally inappropriate!

"Cutter-Suction" Dredgers, like huge vacuum cleaners with rotating cutter heads, are full-fledged ships with a front-end, extended boom having rotating cutter heads and large vacuum tubes next to those heads, which suck up the sludge, as the rotating heads grind into it. These tubes run back to huge sucking pumps on the ship, which then pump the sludge through sealed piping to either barges or over land to dumping or remediation sites. With booster pumps, these dredgers can move that sludge material for many miles, as the Dutch dredging companies do in their constant effort to keep Holland above sea level.

The cheap and inappropriate dredger used by G.E. - Note the sludge coming out of the bucket and becoming water-borne and able to moved by the current down river!

GAZETTE FILE PHOTO

A PCB dredging operation is seen on the Hudson River in Stillwater on Aug. 14, 2014.

When the bucket of a "Grab" Dredger is dropped, it hits the bottom hard, disturbing and churning up the PCB-laden river bottom. As the bucket closes, it further disturbs the PCB-sludge,

making even more of it water-borne. As the bucket is lifted, more PCB-sludge seeps out and also becomes water-borne, which allows the river current to take those PCB's down river, spreading the pollution to other areas! Removing PCB-sludge in this manner spreads the pollution over an ever wider area, and re-dissolves it into the water so that fish and other wildlife can ingest it. It is hard to imagine a worse method of remediation!

Whom did G.E. executives consult when they chose to use this method of "Grab" Dredging? The most experienced dredging companies in the World are almost all Dutch. However, the Army Corp of Engineers has and has used "Cutter-Suction" dredging in many of the major American rivers. Was there any outreach to the Dutch or the Corp of Engineers for their advice on what method of dredging was best for this problem, or were just G.E. accountants consulted??

III. Conclusions

1. The method used by General Electric executives to remove the PCB pollution the company originally had caused cannot be explained by any reasonable analysis other than cutting corners to save costs.

2. Given the nature of "Grab" Dredging and its lower costs, the question has to be raised as to how did G.E. spend so much on this cleanup?

3. Did G.E. executives seek "Best Solutions", and conduct competitive bidding to complete the cleanup?

4. Since the Super Fund legislation allows the courts to cut through the corporate limitation of liability and allows those courts to go after the responsible individuals personally, should the EPA, ENCON, and the New York State Attorney General seriously consider bringing a Federal Criminal Investigation and Case against those executives and their employer, the General Electric Company?

5. Various other organizations will be presenting to the EPA data and other statistics as to how the PCB pollution of the Hudson River has not been adequately cleaned up, and has even been spread down river to other locations. These results give testimony as to the inadequacy of the job done by G.E. and its executives in carrying out their obligations under the EPA Super Fund Cleanup Order.

6. Whether such actions by G.E. and its Executives comes to the level of Gross Negligence and was Willful Misconduct, can only be determined by a thorough investigation and adjudication by the Federal Courts.

An example of the type of configuration of equipment that should have been used by G.E. for the PCB cleanup of the Hudson River is shown on the following page.

Below is a configuration of equipment that should have been used by G.E. to clean up that PDB-sludge: (Photos courtesy of Condreco, S.A., dredging subsidiary of Archirodon Group).

ZEUS C-104

CUTTER-SUCTION DREDGER

DISPLACEMENT:
4,334 tons
TOTAL POWER:
17,000 hp, (diesel)
SUCTION PIPE:
900 mm dia.
DISCHARGE PIPE:
800 mm dia.
DREDGING DEPTH:
25 meters
CUTTER POWER:
2,500 hp
DIMENSIONS (m):
100x18x5.25
CONSTRUCTION:
1979

TRITON

BOOSTER-STATION

DISPLACEMENT:
550 Tons
SUCTION PIPE:
850 mm dia.
DISCHARGE PIPE:
800 mm dia.
PUMP DRIVE:
3,400 hp
CRANE:
12,500 kg
DIMENSIONS (m):
24.5x10.0x3.2
CONSTRUCTION:
1978

«ZEUS C-104» is a heavy-duty, deep-water suction dredger built in Holland in 1979.
The 2,500 hp rock cutter together with a 600-ton ladder, enable this dredger to efficiently excavate solid coral, rock and limestone formations.
«ZEUS C-104» has three dredge pumps, a submerged ladder pump and two booster pumps. In order to optimize its performance, any of the three pumps can be by-passed.
The 3,400 hp «TRITON» Booster Station is matched in power and pump size with both «ZEUS C-104» and «ATLAS C-103» for high efficiency on extra long pipelines and is equipped with tow spuds for easy handling and quick positioning.