Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards

EPA-452/R-11-011
December 2011

Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

CONTENTS

LIST OF FIGURES

LIST OF TABLES

EXECUTIVE SUMMARY

This Regulatory Impact Analysis (RIA) presents the health and welfare benefits, costs, and other impacts of the final Mercury and Air Toxics Standards (MATS) in 2016.

ES.1 Key Findings

This rule will reduce emissions of Hazardous Air Pollutants (HAP), including mercury, from the electric power industry. As a co-benefit, the emissions of certain $PM_{2.5}$ precursors such as SO_2 will also decline. EPA estimates that this final rule will yield annual monetized benefits (in 2007$) of between $37 to $90 billion using a 3% discount rate and $33 to $81 billion using a 7% discount rate. The great majority of the estimates are attributable to co-benefits from 4,200 to 11,000 fewer $PM_{2.5}$-related premature mortalities. The monetized benefits from reductions in mercury emissions, calculated only for children exposed to recreationally caught freshwater fish, are expected to be $0.004 to $0.006 billion in 2016 using a 3% discount rate and $0.0005 to $0.001 billion using a 7% discount rate. The annual social costs, approximated by the compliance costs, are $9.6 billion (2007$) and the annual monetized net benefits are $27 to $80 billion using 3% discount rate or $24 to $71 billion using a 7% discount rate.[1] The benefits outweigh costs by between 3 to 1 or 9 to 1 depending on the benefit estimate and discount rate used. There are some costs and important benefits that EPA could not monetize, such as other mercury reduction benefits and those for the HAP other than mercury being reduced by this final rule. Upon considering these limitations and uncertainties, it remains clear that the benefits of the MATS are substantial and far outweigh the costs. Employment impacts associated with the final rule are estimated to be small.

The benefits and costs in 2016 of the final rule are in Table ES-1. The emission reductions from the electricity sector that are expected to result from the rule are reported in Table ES-2.

[1] As discussed in Chapter 3, costs were annualized using a 6.15% discount rate.

Table ES-1. Summary of EPA's Estimates of Annualized[a] Benefits, Costs, and Net Benefits of the Final MATS in 2016[b] (billions of 2007$)

Description	Estimate (3% Discount Rate)	Estimate (7% Discount Rate)
Costs[c]	$9.6	$9.6
Benefits[d,e,f]	$37 to $90 + B	$33 to $81 + B
Net benefits (benefits-costs)[g]	$27 to $80 + B	$24 to $71 + B

[a] All estimates presented in this report represent annualized estimates of the benefits and costs of the final MATS in 2016 rather than the net present value of a stream of benefits and costs in these particular years of analysis.

[b] Estimates rounded to two significant figures and represent annualized benefits and costs anticipated for the year 2016.

[c] Total social costs are approximated by the compliance costs. Compliance costs consist of IPM projections, monitoring/reporting/recordkeeping costs, and oil-fired fleet analysis costs. For a complete discussion of these costs refer to Chapter 3. Costs were annualized using a 6.15% discount rate.

[d] Total benefits are composed primarily of monetized PM-related health benefits. The reduction in premature fatalities each year accounts for over 90% of total monetized benefits. Benefits in this table are nationwide and are associated with directly emitted $PM_{2.5}$ and SO_2 reductions. The estimate of social benefits also includes CO_2-related benefits calculated using the social cost of carbon, discussed further in Chapter 5.

[e] Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Data limitations prevented us from quantifying these endpoints, and as such, these benefits are inherently more uncertain than those benefits that we were able to quantify. Estimates here are subject to uncertainties discussed further in the body of the document. Potential benefit categories that have not been quantified and monetized are listed in Table ES-5.

[f] Mortality risk valuation assumes discounting over the SAB-recommended 20-year segmented lag structure. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

[g] Net benefits are rounded to two significant figures. Columnar totals may not sum due to rounding.

Table ES-2: Projected Electricity Generating Unit (EGU) Emissions of SO_2, NO_X, Mercury, Hydrogen Chloride, PM, and CO_2 with the Base Case and with MATS, 2015 [a,b]

		Million Tons			Thousand Tons		CO_2
		SO_2	NO_X	Mercury (Tons)	HCl	$PM_{2.5}$	(Million Metric Tonnes)
Base	All EGUs	3.4	1.9	28.7	48.7	277	2,230
	Covered EGUs	3.3	1.7	26.6	45.3	270	1,906
MATS	All EGUs	2.1	1.9	8.8	9.0	227	2,215
	Covered EGUs	1.9	1.7	6.6	5.5	218	1,883

[a] Source: Integrated Planning Model run by EPA, 2011

[b] The year 2016 is the compliance year for MATS, though as we explain in later chapters, we use 2015 as a proxy for compliance in 2016 for IPM emissions and costs due to availability of modeling impacts in that year.

ES.1.1 Health Co-Benefits

The final MATS Rule is expected to yield significant health co-benefits by reducing emissions not only of HAP such as mercury, but also significant co-benefits by reducing to direct fine particles ($PM_{2.5}$) and sulfur dioxide, which contributes to the formation of $PM_{2.5}$.

Our analyses suggest this rule would yield co-benefits in 2016 of $37 to $90 billion (based on a 3% discount rate) and $33 to $81 billion (based on a 7% discount rate). This estimate reflects the economic value of a range of avoided health outcomes including 510 fewer mercury-related IQ points lost as well as avoided $PM_{2.5}$-related impacts, including 4,200 to 11,000 premature deaths, 4,700 nonfatal heart attacks, 2,600 hospitalizations for respiratory and cardiovascular diseases, 540,000 lost work days, and 3.2 million days when adults restrict normal activities because of respiratory symptoms exacerbated by $PM_{2.5}$. We also estimate substantial additional health improvements for children from reductions in upper and lower respiratory illnesses, acute bronchitis, and asthma attacks. See Table ES-3 for a list of the annual reduction in health effects expected in 2016 and Table ES -4 for the estimated value of those reductions. In addition, we include in our monetized co-benefits estimates the effect from the reduction in CO_2 emissions resulting from this rule. We calculate the co-benefits associated with these emission reductions using the interagency estimates of the social cost of carbon (SCC)[1].

It is important to note that the health co-benefits from reduced $PM_{2.5}$ exposure reported here contain uncertainty, including from the following key assumptions:

1. The $PM_{2.5}$-related co-benefits of the regulatory alternatives were derived through a benefit per-ton approach, which does not fully reflect local variability in population density, meteorology, exposure, baseline health incidence rates, or other local factors that might lead to an over-estimate or under-estimate of the actual co-benefits of controlling PM precursors. In addition, differences in the distribution of emissions reductions across states between the modeled scenario and the final rule scenario add uncertainty to the final benefits estimates.

[1] Docket ID EPA-HQ-OAR-2009-0472-114577, *Technical Support Document: Social Cost of Carbon for Regulatory Impact Analysis Under Executive Order 12866,* Interagency Working Group on Social Cost of Carbon, with participation by Council of Economic Advisers, Council on Environmental Quality, Department of Agriculture, Department of Commerce, Department of Energy, Department of Transportation, Environmental Protection Agency, National Economic Council, Office of Energy and Climate Change, Office of Management and Budget, Office of Science and Technology Policy, and Department of Treasury (February 2010). Also available at http://www.epa.gov/otaq/climate/regulations.htm

2. We assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because $PM_{2.5}$ produced via transported precursors emitted from EGUs may differ significantly from direct $PM_{2.5}$ released from diesel engines and other industrial sources, but the scientific evidence is not yet sufficient to allow differential effects estimates by particle type.

3. We assume that the health impact function for fine particles is linear within the range of ambient concentrations under consideration. Thus, the estimates include health co-benefits from reducing fine particles in areas with varied concentrations of $PM_{2.5}$, including both regions that are in attainment with fine particle standard and those that do not meet the standard down to the lowest modeled concentrations.

A large fraction of the $PM_{2.5}$-related benefits associated with this rule occur below the level of the National Ambient Air Quality Standard (NAAQS) for annual $PM_{2.5}$ at 15 $\mu g/m^3$, which was set in 2006. It is important to emphasize that NAAQS are not set at a level of zero risk. Instead, the NAAQS reflect the level determined by the Administrator to be protective of public health within an adequate margin of safety, taking into consideration effects on susceptible populations. While benefits occurring below the standard may be less certain than those occurring above the standard, EPA considers them to be legitimate components of the total benefits estimate.

Based on the modeled interim baseline which is approximately equivalent to the final baseline (see Appendix 5A), 11% and 73% of the estimated avoided premature deaths occur at or above an annual mean $PM_{2.5}$ level of 10 $\mu g/m^3$ (the LML of the Laden et al. 2006 study) and 7.5 $\mu g/m^3$ (the LML of the Pope et al. 2002 study), respectively. These are the source studies for the concentration-response functions used to estimate mortality benefits. As we model avoided premature deaths among populations exposed to levels of $PM_{2.5,}$ we have lower confidence in levels below the LML for each study. However, studies using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality. EPA briefly describes these uncertainties below and in more detail in the benefits chapter of this RIA.

ES.1.2 Welfare Co-Benefits

The term *welfare co-benefits* covers both environmental and societal benefits of reducing pollution, such as reductions in damage to ecosystems, improved visibility and improvements in recreational and commercial fishing, agricultural yields, and forest

productivity. EPA did not quantify any of the important welfare co-benefits expected from the final MATS, but these are discussed in detail in Chapter 5.

Table ES-3. Estimated Reduction in Incidence of Adverse Health Effects of the Mercury and Air Toxics Standards (95% confidence intervals)[a,b]

Impact	Eastern U.S.[c]	Western U.S.	Total
Mercury-Related Endpoints			
IQ Points Lost			510.8
PM-Related Endpoints			
Premature death			
Pope et al. (2002) (age >30)	4,100 (1,100 – 7,000)	130 (30 – 220)	4,200 (1,200 – 7,200)
Laden et al. (2006) (age >25)	10,000 (4,800 – 16,000)	320 (140 – 510)	11,000 (5,000 – 17,000)
Infant (< 1 year)	19 (-21 – 59)	1 (-1 – 2)	20 (-22 – 61)
Chronic bronchitis	2,700 (89 – 5,400)	100 (-1 – 210)	2,800 (88 – 5,600)
Non-fatal heart attacks (age > 18)	4,600 (1,200 – 8,100)	120 (25 – 210)	4,700 (1,200 – 8,300)
Hospital admissions—respiratory (all ages)	820 (320 – 1,300)	17 (6 – 27)	830 (330 – 1,300)
Hospital admissions—cardiovascular (age > 18)	1,800 (1,200 – 2,100)	42 (27 – 50)	1,800 (1,200 – 2,200)
Emergency room visits for asthma (age < 18)	3,000 (1,500 – 4,500)	110 (52 – 160)	3,100 (1,600 – 4,700)
Acute bronchitis (age 8-12)	6,000 (-1,400 – 13,000)	250 (-69 – 560)	6,300 (-1,400 – 14,000)
Lower respiratory symptoms (age 7-14)	77,000 (30,000 – 120,000)	3,100 (1,100 – 5,200)	80,000 (31,000 – 130,000)
Upper respiratory symptoms (asthmatics age 9-18)	58,000 (11,000 – 110,000)	2,400 (360 – 4,400)	60,000 (11,000 – 110,000)
Asthma exacerbation (asthmatics age 6-18)	130,000 (4,500 – 430,000)	5,200 (-6 – 18,000)	130,000 (4,500 – 450,000)
Lost work days (ages 18-65)	520,000 (440,000 – 600,000)	21,000 (18,000 – 24,000)	540,000 (460,000 – 620,000)
Minor restricted-activity days (ages 18-65)	3,100,000 (2,500,000 – 3,700,000)	120,000 (99,000 – 150,000)	3,200,000 (2,600,000 – 3,800,000)

[a] Estimates rounded to two significant figures; column values will not sum to total value.

[b] The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts.

[c] Includes Texas and those states to the north and east.

Table ES-4. Estimated Economic Value of Health and Welfare Co-Benefits of the Mercury and Air Toxics Standards (95% confidence intervals, billions of 2007$)[a]

Impact	Pollutant	Eastern U.S.[b]	Western U.S.	Total
Avoided IQ loss associated with methylmercury exposure from self-caught fish consumption among recreational anglers				
3% discount rate	Hg			$0.004 – $0.006
7% discount rate	Hg			$0.0005 – $0.001
Adult premature death (Pope et al., 2002 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$33 ($2.6 - $99)	$1.0 (<$0.01 - $3.1)	$34 ($2.6 - $100)
7% discount rate	$PM_{2.5}$	$30 ($2.3 - $90)	$0.9 (<$0.01 - $2.8)	$30 ($2.4 - $92)
Adult premature death (Laden et al., 2006 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$84 ($7.4 - $240)	$2.6 ($0.1 - $7.6)	$87 ($7.5 - $250)
7% discount rate	$PM_{2.5}$	$76 ($6.7 - $220)	$2.3 ($0.1 - $6.9)	$78 ($6.8 - $230)
Infant premature death	$PM_{2.5}$	$0.2 ($-0.2 – $0.8)	<$0.01	$0.2 ($-0.2 - $0.8)
Chronic bronchitis	$PM_{2.5}$	$1.3 ($0.1 - $6.1)	$0.1 (<$0.01 - $0.2)	$1.4 ($0.1 - $6.4)
Non-fatal heart attacks				
3% discount rate	$PM_{2.5}$	$0.5 ($0.1 - $1.3)	<$0.01	$0.5 ($0.1 - $1.3)
7% discount rate	$PM_{2.5}$	$0.4 ($0.1 - $1.0)	<$0.01	$0.4 ($0.1 - $1.0)
Hospital admissions—respiratory	$PM_{2.5}$	$0.01 (<$0.01 - $0.02)	<$0.01	$0.01 ($0.01 - $0.02)
Hospital admissions—cardiovascular	$PM_{2.5}$	$0.03 (<$0.01 - $0.05)	<$0.01	$0.03 (<$0.01 - $0.05)
Emergency room visits for asthma	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Acute bronchitis	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lower respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Upper respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Asthma exacerbation	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lost work days	$PM_{2.5}$	$0.1 ($0.1 - $0.1)	<$0.01	$0.1 ($0.1 - $0.1)

(continued)

Table ES-4. Estimated Economic Value of Health and Welfare Co-Benefits of the Mercury and Air Toxics Standards (95% confidence intervals, billions of 2007$)[a] (continued)

Impact	Pollutant	Eastern U.S.[b]	Western U.S.	Total
Minor restricted-activity days	$PM_{2.5}$	$0.2 ($0.1 - $0.3)	<$0.01	$0.2 ($0.1 - $0.3)
CO_2-related benefits				
(3% discount rate)	CO_2			$0.36
Monetized total Benefits (Pope et al., 2002 $PM_{2.5}$ mortality estimate)				
3% discount rate		$35+B ($2.8 - $110)	$1.1+B ($0.03 - $3.4)	$37+B ($3.2 - $110)
7% discount rate		$32+B ($2.5 - $98)	$1.0+B ($0.03 - $3.1)	$33+B ($2.9 - $100)
Monetized total Benefits (Laden et al., 2006 $PM_{2.5}$ mortality estimate)				
3% discount rate		$87+B ($7.5 - $250)	$2.7+B ($0.1 - $7.9)	$90+B ($8.0 - $260)
7% discount rate		$78+B ($6.8 - $230)	$2.4+B ($0.1 - $7.2)	$81+B ($7.3 - $240)

[a] Economic value adjusted to 2007$ using GDP deflator. Estimates rounded to two significant figures. The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts. Confidence intervals reflect random sampling error and not the additional uncertainty associated with accounting for differences in air quality baseline forecasts described in Chapter 5. The net present value of reduced CO_2 emissions are calculated differently than other benefits. The same discount rate used to discount the value of damages from future emissions (SCC at 5, 3, 2.5 percent) is used to calculate net present value of SCC for internal consistency. This table shows monetized CO_2 co-benefits at discount rates at 3 and 7 percent that were calculated using the global average SCC estimate at a 3% discount rate because the interagency workgroup on this topic deemed this marginal value to be the central value. In section 5.6 we also report CO_2 co-benefits using discount rates of 5 percent (average), 2.5 percent (average), and 3 percent (95^{th} percentile).

[b] Includes Texas and those states to the north and east.

Figure ES-1 summarizes an array of $PM_{2.5}$-related monetized benefits estimates based on alternative epidemiology and expert-derived PM-mortality estimate.

Figure ES-2 summarizes the estimated net benefits for the final rule by displaying all possible combinations of health and climate co-benefits and costs. Each of the 14 bars in each graph represents a separate point estimate of net benefits under a certain combination of cost and benefit estimation methods. Because it is not a distribution, it is not possible to infer the likelihood of any single net benefit estimate.

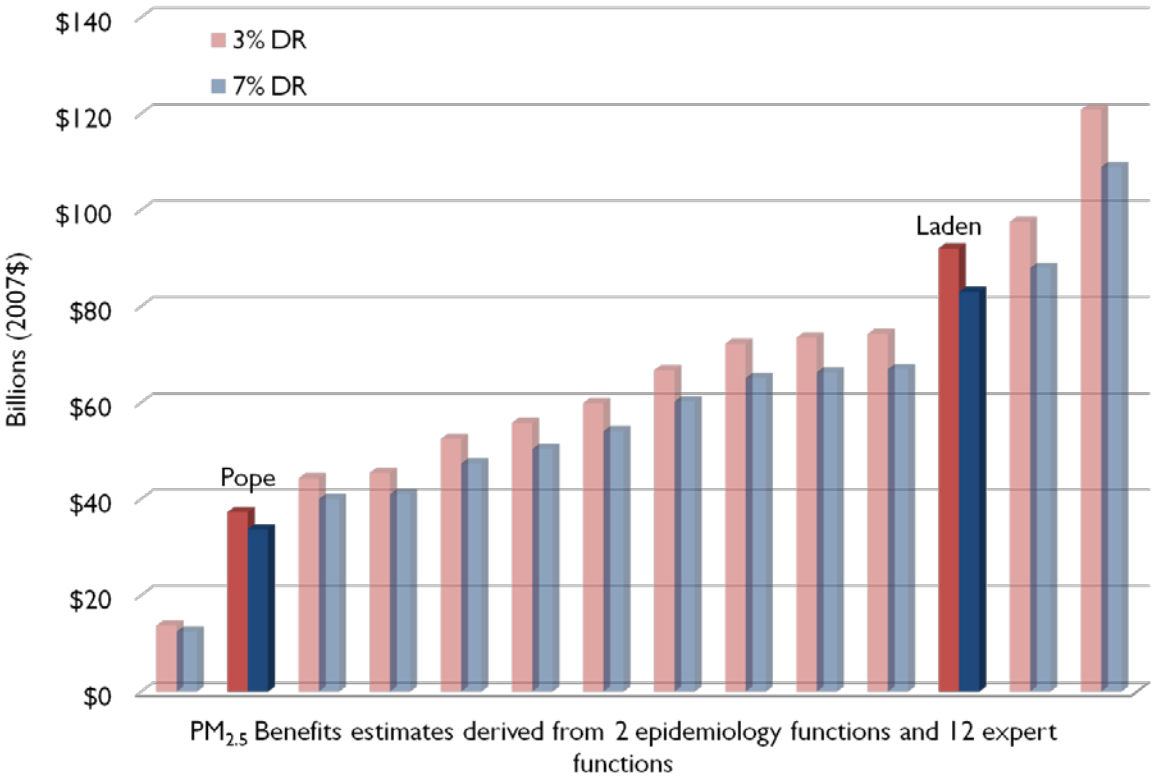

Figure ES-1. Economic Value of Estimated $PM_{2.5}$-Related Health Co-Benefits According to Epidemiology or Expert-Derived PM Mortality Risk Estimate[a,b]

[a] Based on the modeled interim baseline, which is approximately equivalent to the final baseline (see Appendix 5A)

[b] Column total equals sum of $PM_{2.5}$-related mortality and morbidity benefits.

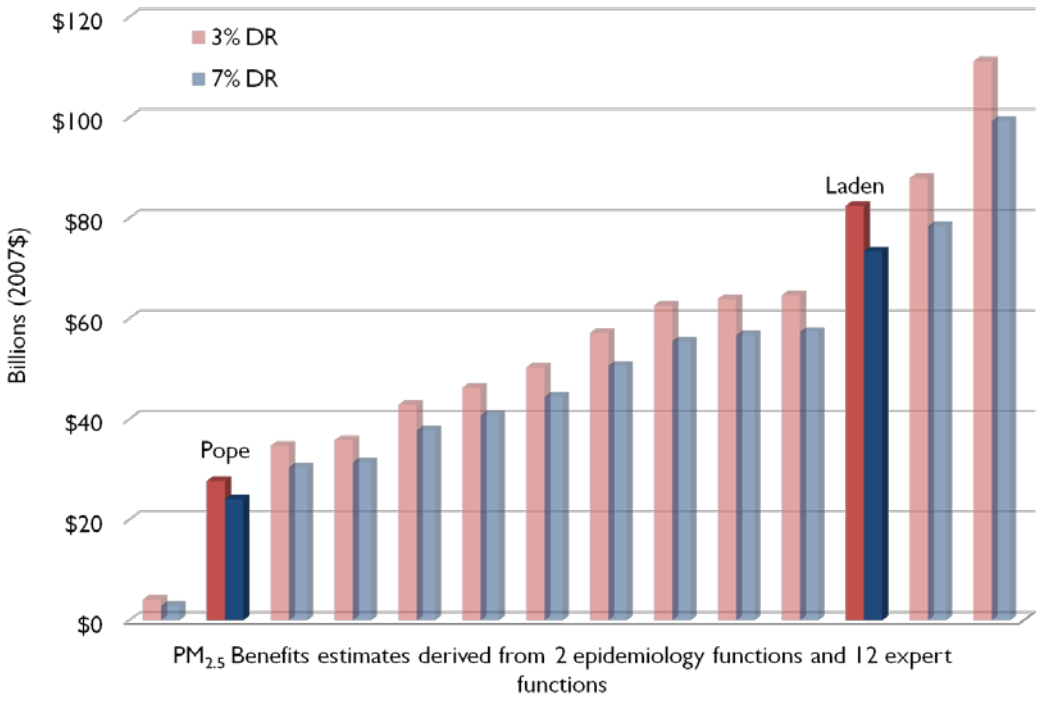

Figure ES-2. Net Benefits of the MATS Rule According to $PM_{2.5}$ Epidemiology or Expert-Derived Mortality Risk Estimate[a,b]

[a] Based on the modeled interim baseline, which is approximately equivalent to the final baseline (see Appendix 5A)

[b] Column total equals sum of $PM_{2.5}$-related mortality and morbidity benefits.

ES.2 Not All Benefits Quantified

EPA was unable to quantify or monetize all of the health and environmental benefits associated with the final MATS Rule. EPA believes these unquantified benefits could be substantial, including the overall value associated with HAP reductions, value of increased agricultural crop and commercial forest yields, visibility improvements, and reductions in nitrogen and acid deposition and the resulting changes in ecosystem functions. Tables ES-5 and ES-6 provide a list of these benefits.

Table ES-5. Human Health Effects of Pollutants Affected by the Mercury and Air Toxics Standards

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information[a]
Improved Human Health				
Reduced incidence of premature mortality from exposure to $PM_{2.5}$	Adult premature mortality based on cohort study estimates and expert elicitation estimates (age >25 or age >30)	✓	✓	Section 5.4
	Infant mortality (age <1)	✓	✓	Section 5.4
Reduced incidence of morbidity from exposure to $PM_{2.5}$	Non-fatal heart attacks (age > 18)	✓	✓	Section 5.4
	Hospital admissions—respiratory (all ages)	✓	✓	Section 5.4
	Hospital admissions—cardiovascular (age >18)	✓	✓	Section 5.4
	Emergency room visits for asthma (age <18)	✓	✓	Section 5.4
	Acute bronchitis (age 8–12)	✓	✓	Section 5.4
	Lower respiratory symptoms (age 7–14)	✓	✓	Section 5.4
	Upper respiratory symptoms (asthmatics age 9-11)	✓	✓	Section 5.4
	Asthma exacerbation (asthmatics age 6–18)	✓	✓	Section 5.4
	Lost work days (age 18-65)	✓	✓	Section 5.4
	Minor restricted-activity days (age 18–65)	✓	✓	Section 5.4
	Chronic bronchitis (age >26)	✓	✓	Section 5.4
	Other cardiovascular effects (e.g., other ages)	—	—	PM ISA[c]
	Other respiratory effects (e.g., pulmonary function, non-asthma ER visits, non-bronchitis chronic diseases, other ages and populations)	—	—	PM ISA[c]
	Reproductive and developmental effects (e.g., low birth weight, pre-term births, etc)	—	—	PM ISA[c, d]
	Cancer, mutagenicity, and genotoxicity effects	—	—	PM ISA[c, d]
Reduced incidence of mortality from exposure to ozone	Premature mortality based on short-term study estimates (all ages)	—	—	Ozone CD, Draft Ozone ISA[b]
	Premature mortality based on long-term study estimates (age 30–99)	—	—	Ozone CD, Draft Ozone ISA[b]
Reduced incidence of morbidity from exposure to ozone	Hospital admissions—respiratory causes (age > 65)	—	—	Ozone CD, Draft Ozone ISA[b]
	Hospital admissions—respiratory causes (age <2)	—	—	Ozone CD, Draft Ozone ISA[b]
	Emergency room visits for asthma (all ages)	—	—	Ozone CD, Draft Ozone ISA[b]
	Minor restricted-activity days (age 18–65)	—	—	Ozone CD, Draft Ozone ISA[b]

(continued)

Table ES-5. Human Health Effects of Pollutants Affected by the Mercury and Air Toxics Standards (continued)

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information
	School absence days (age 5–17)	—	—	Ozone CD, Draft Ozone ISA[b]
	Decreased outdoor worker productivity (age 18-65)	—	—	Ozone CD, Draft Ozone ISA[b]
	Other respiratory effects (e.g., premature aging of lungs)	—	—	Ozone CD, Draft Ozone ISA[c]
	Cardiovascular and nervous system effects	—	—	Ozone CD, Draft Ozone ISA[d]
	Reproductive and developmental effects	—	—	Ozone CD, Draft Ozone ISA[d]
Reduced incidence of morbidity from exposure to NO_2	Asthma hospital admissions (all ages)	—	—	NO_2 ISA[b]
	Chronic lung disease hospital admissions (age > 65)	—	—	NO_2 ISA[b]
	Respiratory emergency department visits (all ages)	—	—	NO_2 ISA[b]
	Asthma exacerbation (asthmatics age 4–18)	—	—	NO_2 ISA[b]
	Acute respiratory symptoms (age 7–14)	—	—	NO_2 ISA[b]
	Premature mortality	—	—	NO_2 ISA[c,d]
	Other respiratory effects (e.g., airway hyperresponsiveness and inflammation, lung function, other ages and populations)	—	—	NO_2 ISA[c,d]
Reduced incidence of morbidity from exposure to SO_2	Respiratory hospital admissions (age > 65)	—	—	SO_2 ISA[b]
	Asthma emergency room visits (all ages)	—	—	SO_2 ISA[b]
	Asthma exacerbation (asthmatics age 4–12)	—	—	SO_2 ISA[b]
	Acute respiratory symptoms (age 7–14)	—	—	SO_2 ISA[b]
	Premature mortality	—	—	SO_2 ISA[c,d]
	Other respiratory effects (e.g., airway hyperresponsiveness and inflammation, lung function, other ages and populations)	—	—	SO_2 ISA[c,d]
Reduced incidence of morbidity from exposure to methyl mercury (through reduced mercury deposition as well as the role of sulfate in methylation)	Neurologic effects—IQ loss	✓	✓	IRIS; NRC, 2000[b]
	Other neurologic effects (e.g., developmental delays, memory, behavior)	—	—	IRIS; NRC, 2000c
	Cardiovascular effects	—	—	IRIS; NRC, 2000[c,d]
	Genotoxic, immunologic, and other toxic effects	—	—	IRIS; NRC, 2000[c,d]

[a] For a complete list of references see Chapter 5.
[b] We assess these benefits qualitatively due to time and resource limitations for this analysis.
[c] We assess these benefits qualitatively because we do not have sufficient confidence in available data or methods.
[d] We assess these benefits qualitatively because current evidence is only suggestive of causality or there are other significant concerns over the strength of the association.

Table ES-6. Environmental Effects of Pollutants Affected by the Mercury and Air Toxics Standards

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information[a]
Improved Environment				
Reduced visibility impairment	Visibility in Class I areas in SE, SW, and CA regions	—	—	PM ISA[b]
	Visibility in Class I areas in other regions	—	—	PM ISA[b]
	Visibility in residential areas	—	—	PM ISA[b]
Reduced climate effects	Global climate impacts from CO_2	—	✓	Section 5.6
	Climate impacts from ozone and PM	—	—	Section 5.6
	Other climate impacts (e.g., other GHGs, other impacts)	—	—	IPCC[c]
Reduced effects on materials	Household soiling	—	—	PM ISA[c]
	Materials damage (e.g., corrosion, increased wear)	—	—	PM ISA[c]
Reduced effects from PM deposition (metals and organics)	Effects on Individual organisms and ecosystems	—	—	PM ISA[c]
Reduced vegetation and ecosystem effects from exposure to ozone	Visible foliar injury on vegetation	—	—	Ozone CD, Draft Ozone ISA[c]
	Reduced vegetation growth and reproduction	—	—	Ozone CD, Draft Ozone ISA[b]
	Yield and quality of commercial forest products and crops	—	—	Ozone CD, Draft Ozone ISA[b,d]
	Damage to urban ornamental plants	—	—	Ozone CD, Draft Ozone ISA[c]
	Carbon sequestration in terrestrial ecosystems	—	—	Ozone CD, Draft Ozone ISA[c]
	Recreational demand associated with forest aesthetics	—	—	Ozone CD, Draft Ozone ISA[c]
	Other non-use effects			Ozone CD, Draft Ozone ISA[c]
	Ecosystem functions (e.g., water cycling, biogeochemical cycles, net primary productivity, leaf-gas exchange, community composition)	—	—	Ozone CD, Draft Ozone ISA[c]

(continued)

Table ES-6. Environmental Effects of Pollutants Affected by the Mercury and Air Toxics Standards (continued)

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information
Reduced effects from acid deposition	Recreational fishing	—	—	NO_x SO_x ISA[b]
	Tree mortality and decline	—	—	NO_x SO_x ISA[c]
	Commercial fishing and forestry effects	—	—	NO_x SO_x ISA[c]
	Recreational demand in terrestrial and aquatic ecosystems	—	—	NO_x SO_x ISA[c]
	Other nonuse effects			NO_x SO_x ISA[c]
	Ecosystem functions (e.g., biogeochemical cycles)	—	—	NO_x SO_x ISA[c]
Reduced effects from nutrient enrichment	Species composition and biodiversity in terrestrial and estuarine ecosystems	—	—	NO_x SO_x ISA[c]
	Coastal eutrophication	—	—	NO_x SO_x ISA[c]
	Recreational demand in terrestrial and estuarine ecosystems	—	—	NO_x SO_x ISA[c]
	Other non-use effects			NO_x SO_x ISA[c]
	Ecosystem functions (e.g., biogeochemical cycles, fire regulation)	—	—	NO_x SO_x ISA[c]
Reduced vegetation effects from ambient exposure to SO_2 and NO_x	Injury to vegetation from SO_2 exposure	—	—	NO_x SO_x ISA[c]
	Injury to vegetation from NO_x exposure	—	—	NO_x SO_x ISA[c]
Reduced incidence of morbidity from exposure to methyl mercury (through reduced mercury deposition as well as the role of sulfate in methylation)	Effects on fish, birds, and mammals (e.g., reproductive effects)	—	—	Mercury Study RTC[c,d]
	Commercial, subsistence and recreational fishing	—	—	Mercury Study RTC[c]

[a] For a complete list of references see Chapter 5.
[b] We assess these benefits qualitatively due to time and resource limitations for this analysis.
[c] We assess these benefits qualitatively because we do not have sufficient confidence in available data or methods.
[d] We assess these benefits qualitatively because current evidence is only suggestive of causality or there are other significant concerns over the strength of the association.

ES.3 Costs and Employment Impacts

The projected annual incremental private costs of the final MATS Rule to the electric power industry are $9.6 billion in 2015.[1] These costs represent the total cost to the electricity-generating industry of reducing HAP emissions to meet the emissions limits set out in the rule. Estimates are in 2007 dollars. These total costs of the rule are estimated using the Integrated Planning Model (IPM), as well as additional analyses for oil-fired units and monitoring/record-keeping costs.

There are several national changes in energy prices that result from the final MATS Rule. Retail electricity prices are projected to increase in the contiguous US by an average of 3.1% in 2015 with the final MATS Rule. On a weighted average basis between 2015 and 2030, consumer natural gas price anticipated to increase from 0.3% to 0.6% depending on consumer class in response to the final MATS Rule.

There are several other types of energy impacts associated with the final MATS Rule. A small amount of coal-fired capacity, about 4.7 GW (less than 2 percent of all coal-fired capacity in 2015), is projected to become uneconomic to maintain by 2015. These units are predominantly smaller and less frequently-used generating units dispersed throughout the contiguous US. If current forecasts of either natural gas prices or electricity demand were revised in the future to be higher, that would create a greater incentive to keep these units operational. Coal production for use in the power sector is projected to decrease by 1 percent by 2015, and we expect slightly reduced coal demand in Appalachia and the West with the final MATS Rule.

In addition to addressing the costs and benefits of the final MATS Rule, EPA has estimated a portion of the employment impacts of this rulemaking. We have estimated two types of impacts. One provides an estimate of the employment impacts on the regulated industry over time. The second covers the short-term employment impacts associated with the construction of needed pollution control equipment until the compliance date of the regulation. We expect that the rule's impact on employment will be small, but will (on net) result in an expected increase in employment.

[1] The year 2016 is the compliance year for MATS, though as we explain in later chapters, we use 2015 as a proxy for compliance in 2016 for IPM emissions, costs and economic impact analysis due to availability of modeling impacts in that year.

The approaches to estimate employment impacts use different analytical techniques, are applied to different industries during different time periods, and use different units of analysis. No overlapping estimates are summed. Estimates of employment changes per dollar of expenditure on pollution control from Morgenstern et al. (2002) are used to estimate the ongoing annual employment impacts for the regulated entities (the electric power sector) as a result of this rule. The short term estimates for employment needed to design, construct, and install the control equipment in the three year period before the compliance date are also provided using an approach that estimates employment impacts for the environmental protection sector based on forecast changes from IPM on the number and scale of pollution controls and labor intensities in relevant sectors. Finally, some of the other types of employment impacts that will be ongoing are estimated using IPM outputs and labor intensities, as reported in Chapter 6, but not included in this table because they omit some potentially important categories.

In Table ES-7, we show the employment impacts of the MATS Rule as estimated by the environmental protection sector approach and by the Morgenstern approach.

Table ES-7. Estimated Employment Impact Table

	Annual (Reoccurring)	One Time (Construction During Compliance Period)
Environmental protection sector approach[a]	Not applicable	46,000
Net effect on electric utility sector employment from Morgenstern et al., approach[c]	8,000[b] −15,000 to 30,000[d]	Not Applicable

[a] These one-time impacts on employment are estimated in terms of job-years.

[b] This estimate is not statistically different from zero.

[c] These annual or reoccurring employment impacts are estimated in terms of production workers as defined by the US Census Bureau's Annual Survey of Manufacturers (ASM).

[d] 95% confidence interval

ES.4 Small Entity and Unfunded Mandates Impacts

After preparing an analysis of small entity impacts, EPA cannot certify that there will be no SISNOSE (significant economic impacts on a substantial number of small entities) for this rule. Of the 82 small entities affected, 40 are projected to have costs greater than 1 percent of their revenues. The exclusion of units smaller than 25 Megawatt capacity (MW) as per the requirements of the Clean Air Act has already significantly reduced the burden on small entities,

and EPA participated in a Small Business Regulatory Enforcement Fairness Act (SBREFA) Panel to examine ways to mitigate the impact of the proposed Toxics Rule on affected small entities

EPA examined the potential economic impacts on state and municipality-owned entities associated with this rulemaking based on assumptions of how the affected states will implement control measures to meet their emissions. These impacts have been calculated to provide additional understanding of the nature of potential impacts and additional information.

According to EPA's analysis, of the 96 government entities considered in this, EPA projects that 42 government entities will have compliance costs greater than 1 percent of base generation revenue in 2015, based on our assumptions of how the affected states implement control measures to meet their emissions budgets as set forth in this rulemaking.

Government entities projected to experience compliance costs in excess of 1 percent of revenues may have some potential for significant impact resulting from implementation of MATS.

ES.5 Limitations and Uncertainties

Every analysis examining the potential benefits and costs of a change in environmental protection requirements is limited to some extent by data gaps, limitations in model capabilities (such as geographic coverage), and variability or uncertainties in the underlying scientific and economic studies used to configure the benefit and cost models. Despite the uncertainties, we believe this benefit-cost analysis provides a reasonable indication of the expected economic benefits and costs of the final MATS Rule.

For this analysis, such uncertainties include possible errors in measurement and projection for variables such as population growth and baseline incidence rates; uncertainties associated with estimates of future-year emissions inventories and air quality; variability in the estimated relationships between changes in pollutant concentrations and the resulting changes in health and welfare effects; and uncertainties in exposure estimation.

Below is a summary of the key uncertainties of the analysis:

Costs

- Compliance costs are used to approximate the social costs of this rule. Social costs may be higher or lower than compliance costs and differ because of preexisting distortions in the economy, and because certain compliance costs may represent shifts in rents.

- Analysis does not capture employment shifts as workers are retrained at the same company or re-employed elsewhere in the economy.

- We do not include the costs of certain relatively small permitting costs associated with updating Title V permits.

- Technological innovation is not incorporated into these cost estimates. Thus, these cost estimates may be potentially higher than what may occur in the future, all other things being the same.

Benefits

- The mercury concentration estimates for the analysis come from several different sources.

- The mercury concentration estimates used in the model were based on simple temporal and spatial averages of reported fish tissue samples. This approach assumes that the mercury samples are representative of "local" conditions (i.e., within the same HUC 12) in similar waterbodies (i.e., rivers or lakes).

- State-level averages for fishing behavior of recreational anglers are applied to each modeled census tract in the state; which does not reflect within-state variation in these factors.

- Application of state-level fertility rates to specific census tracts (and specifically to women in angler households.

- Applying the state-level individual level fishing participation rates to approximate the household fishing rates conditions at a block level.

- Populations are only included in the model if they are within a reasonable distance of a waterbody with fish tissue MeHg samples. This approach undercounts the exposed population (by roughly 40 to 45%) and leads to underestimates of national aggregate baseline exposures and risks and underestimates of the risk reductions and benefits resulting from mercury emission reductions.

- Assumption of 8 g/day fish consumption rate for the general population in freshwater angler households.

- The dose-response model used to estimate neurological effects on children because of maternal mercury body burden has several important uncertainties, including selection of IQ as a primary endpoint when there may be other more sensitive endpoints, selection of the blood-to-hair ratio for mercury, and the dose-response estimates from the epidemiological literature. Control for confounding from the

potentially positive cognitive effects of fish consumption and, more specifically, omega-3 fatty acids.

- Valuation of IQ losses using a lost earning approach has several uncertainties, including (1) there is a linear relationship between IQ changes and net earnings losses, (2) the unit value applies to even very small changes in IQ, and (3) the unit value will remain constant (in real present value terms) for several years into the future. Each unit value for IQ losses has two main sources of uncertainty (1). The statistical error in the average percentage change in earnings as a result of IQ changes and (2) estimates of average lifetime earnings and costs of schooling.

- Based on the modeled interim baseline which is approximately equivalent to the final baseline (see Appendix 5A), 11% and 73% of the estimated avoided premature deaths occur at or above an annual mean $PM_{2.5}$ level of 10 $\mu g/m^3$ (the LML of the Laden et al. 2006 study) and 7.5 $\mu g/m^3$ (the LML of the Pope et al. 2002 study), respectively. These are the source studies for the concentration-response functions used to estimate mortality benefits. As we model avoided premature deaths among populations exposed to levels of $PM_{2.5}$ that are successively lower than the LML of each study our confidence in the results diminishes. However, studies using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality.

- There are uncertainties related to the health impact functions used in the analysis. These include: within study variability; across study variation; the application of concentration-response (C-R) functions nationwide; extrapolation of impact functions across population; and various uncertainties in the C-R function, including causality and thresholds. Therefore, benefits may be under- or over-estimates.

- Analysis is for 2016, and projecting key variables introduces uncertainty. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions and source level emissions, as well as population, health baselines, incomes, technology, and other factors.

- This analysis omits certain unquantified effects due to lack of data, time and resources. These unquantified endpoints include other health and ecosystem effects. EPA will continue to evaluate new methods and models and select those most appropriate for estimating the benefits of reductions in air pollution. Enhanced collaboration between air quality modelers, epidemiologists, toxicologists, ecologists, and economists should result in a more tightly integrated analytical framework for measuring benefits of air pollution policies.

- $PM_{2.5}$ mortality co-benefits represent a substantial proportion of total monetized benefits (over 90%), and these estimates have following key assumptions and uncertainties.

- The $PM_{2.5}$-related co-benefits of the alternative scenarios were derived through a benefit per-ton approach, which does not fully reflect local variability in population density, meteorology, exposure, baseline health incidence rates, or other local factors that might lead to an over-estimate or under-estimate of the actual benefits of this rule.

- We assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because $PM_{2.5}$ produced via transported precursors emitted from EGUs may differ significantly from direct $PM_{2.5}$ released from diesel engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.

- We assume that the health impact function for fine particles is linear within the range of ambient concentrations under consideration. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of $PM_{2.5,}$ including both regions that are in attainment with fine particle standard and those that do not meet the standard down to the lowest modeled concentrations.

- To characterize the uncertainty in the relationship between $PM_{2.5}$ and premature mortality, we include a set of twelve estimates based on results of the expert elicitation study in addition to our core estimates. Even these multiple characterizations omit the uncertainty in air quality estimates, baseline incidence rates, populations exposed and transferability of the effect estimate to diverse locations. As a result, the reported confidence intervals and range of estimates give an incomplete picture about the overall uncertainty in the $PM_{2.5}$ estimates. This information should be interpreted within the context of the larger uncertainty surrounding the entire analysis.

ES.6 References

Laden, F., J. Schwartz, F.E. Speizer, and D.W. Dockery. 2006. "Reduction in Fine Particulate Air Pollution and Mortality." American Journal of Respiratory and Critical Care Medicine 173:667-672. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. Washington, DC: The National Academies Press.

Levy JI, Baxter LK, Schwartz J. 2009. Uncertainty and variability in health-related damages from coal-fired power plants in the United States. Risk Anal. doi: 10.1111/j.1539-6924.2009.01227.x [Online 9 Apr 2009].

Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." Journal of the American Medical Association 287:1132-1141.

U.S. Environmental Protection Agency (EPA). December 2010. Guidelines for Preparing Economic Analyses. EPA 240-R-10-001.

U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance to Federal Agencies on Preparation of Regulatory Analysis.

Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Infant Mortality and Particulate Air Pollution in the United States." Environmental Health Perspectives 105(6):608-612.

CHAPTER 1
INTRODUCTION AND BACKGROUND

1.1 Introduction

In this action, EPA is addressing the emissions of mercury and other hazardous air pollutants by coal- and oil-fired electricity generating units. This document presents the health and welfare benefits of the final Mercury and Air Toxics Standards and compares the benefits of this rule to the estimated costs of implementing the rule in 2016. This chapter contains background information on the rule and an outline of the chapters of this Regulatory Impact Analysis (RIA).

1.2 Background for Final Mercury and Air Toxics Standards

1.2.1 NESHAP

This action finalizes National Emission Standards for Hazardous Air Pollutants (NESHAP) for new and existing coal- and oil-fired electricity generating units (EGUs) meeting the definition found in Clean Air Act (CAA) section 112(a)(8). EPA is promulgating these standards to meet its statutory obligation to address HAP emissions from these sources under CAA section 112(d). The final NESHAP for new and existing coal- and oil-fired EGUs will be promulgated under 40 CFR part 63, subpart UUUUU.

On December 20, 2000 (65 FR 79825), EPA determined that regulation of coal- and oil-fired EGUs under CAA section 112 was appropriate and necessary, in accordance with CAA section 112(n)(1)(A). EPA at the same time added coal- and oil-fired EGUs to the list of source categories requiring regulation under CAA section 112(d). The December 2000 listing triggered the deadline established by Congress in CAA section 112(c)(5) under which EPA has two years from the date of listing in which to promulgate "emissions standards under section (d) of this section."

In 2002, EPA initiated a CAA section 112(d) standard setting process for coal- and oil-fired EGUs, and on January 30, 2004, proposed CAA section 112(d) standards for mercury (Hg) emissions from coal-fired EGUs and nickel (Ni) emissions from oil-fired EGUs, and, in the alternative, proposed to remove EGUs from the CAA section 112(c) list based on a finding that it was neither appropriate nor necessary to regulate EGUs pursuant to CAA section 112. EPA never finalized the proposed CAA section 112(d) standard. The Agency finalized the CAA section 111 alternative, after taking and responding to extensive public comments on both sets of regulatory options, by issuing a de-listing rule (Section 112(n) Revision Rule; 70 FR 15994;

March 29, 2005) and a final rule, the Clean Air Mercury Rule (CAMR), establishing Hg emissions standards for coal-fired EGUs under CAA section 111 on May 18, 2005 (70 FR 28606). The removal of EGUs from the CAA section 112 list was challenged in the United States (U.S.) Court of Appeals for the District of Columbia Circuit (D.C. Circuit Court).

Petitions for reconsideration were filed by a number of parties in summer 2005. EPA responded to the petitions with a final notice of reconsideration on June 9, 2006 (71 FR 33388). Petitions for judicial review were filed on November 29, 2006, by a number of parties[1] (*State of New Jersey, et al., v. EPA, 517 F.3d 574)*.

On February 8, 2008, the D.C. Circuit Court vacated the Section 112(n) Revision Rule (*State of New Jersey, et al., v. EPA, 517 F.3d 574)*, and subsequently denied rehearing and rehearing *en banc* of that decision. As a part of the decision, the D.C. Circuit Court also vacated CAMR, reverting to the December 2000 regulatory determination and requiring the development of emission standards under CAA section 112(d) (MACT standards) for coal- and oil-fired EGUs. The litigation process continued until, on January 29, 2009, EPA requested of the Department of Justice (DOJ) that the Government's appeals be withdrawn.

On December 18, 2008, several environmental and public health organizations ("Plaintiffs")[2] filed a complaint in the D.C. District Court (Civ. No. 1:08-cv-02198 (RMC)) alleging that the Agency had failed to perform a nondiscretionary duty under CAA section 304(a)(2) by failing to promulgate final section 112(d) standards for HAP from coal- and oil-fired EGUs by the statutorily-mandated deadline, December 20, 2002, 2 years after such sources were listed under section 112(c). EPA settled that litigation. A Consent Decree was issued on April 15, 2010, that calls for EPA to, no later than March 16, 2011, sign for publication in the Federal Register a notice of proposed rulemaking setting forth EPA's proposed emission standards for coal- and oil-fired EGUs and, no later than November 16, 2011, sign for publication in the Federal Register a notice of final rulemaking. EPA and the litigants agreed to a 30-day extension in order to fully respond to the 960,000 comments received on the proposed rule. This agreement extended the signing deadline to December 16, 2011.

[1] Environmental Petitioners; the National Congress of American Indians and Treaty Tribes; ARIPPA; American Coal for Balanced Mercury Regulations, et al.; United Mine Workers of America; Alaska Industrial Development and Export Authority; the States of New Jersey, California, Connecticut, Delaware, Illinois, Maine, Maryland, Massachusetts, Michigan, Minnesota, New Hampshire, New Mexico, New York, Pennsylvania, Rhode Island, Vermont, and Wisconsin; and the City of Baltimore, MD.

[2] American Nurses Association, Chesapeake Bay Foundation, Inc., Conservation Law Foundation, Environment America, Environmental Defense Fund, Izaak Walton League of America, Natural Resources Council of Maine, Natural Resources Defense Council, Physicians for Social Responsibility, Sierra Club, The Ohio Environmental Council, and Waterkeeper Alliance, Inc.

On March 16, 2011, in response to the D.C. Circuit Court's vacatur, EPA proposed CAA section 112(d) NESHAP for all coal- and oil-fired EGUs that reflect the application of the maximum achievable control technology (MACT) consistent with the requirements of CAA sections 112(d)(2) and (3). This action finalizes that proposed rule. This final rule is intended to protect air quality and promote public health by reducing emissions of the hazardous air pollutants (HAP) listed in CAA section 112(b).

1.2.2 NSPS

Section 111(b)(1)(b) of the CAA requires EPA to periodically review and revise the New Source Performance Standards (NSPS) as necessary to reflect improvements in methods for reducing emissions. The NSPS for EGUs (40 CFR part 60, subpart Da) were originally promulgated on June 11, 1979 (44 FR 33580). On February 27, 2006, EPA promulgated amendments to the NSPS for particulate matter (PM), sulfur dioxide (SO_2), and nitrogen oxides (NO_X) contained in the standards of performance for EGUs (71 FR 9866). EPA was subsequently sued by the offices of multiple states attorneys general and environmental organizations on the amendments. The Petitioners alleged that EPA failed to correctly identify the best system of emission reductions for the amended SO_2 and NO_X standards. The Petitioners also claimed that it is appropriate to establish emission limits for fine particulate matter and condensable particulate matter. Based upon further examination of the record, EPA has determined that certain issues in the rule warrant further consideration. On September 4, 2009, EPA was granted a voluntary remand without vacatur of the 2006 amendments. EPA considers it appropriate to respond to the NSPS voluntary remand in conjunction with the EGU NESHAP since it allows EPA to more comprehensively consider the impact on the utility sector. Therefore, even though there was no judicial timetable to complete the NSPS remand, EPA proposed it in conjunction with the NESHAP. We also proposed several minor amendments, technical clarifications, and corrections to existing provisions of the fossil fuel-fired EGU and large and small industrial-commercial-institutional steam generating units NSPS, 40 CFR part 60, subparts D, Db, and Dc. The NSPS and amendments are being finalized along with the NESHAP in this action.

The title Mercury and Air Toxics Standards (MATS) used in the remainder of this RIA refers to the combination of the EGU NESHAP and NSPS.

1.3 Appropriate & Necessary Analyses

In the preamble to the proposed rule, EPA confirmed the December 2000 finding that it is appropriate to regulate emissions of Hg and other HAP from EGUs because emissions of

those pollutants pose hazards to public health and the environment and EGUs are the largest or among the largest contributors of many of those HAP. We also confirmed that it is necessary to regulate EGUs under section 112 for a variety of reasons, including that hazards to public health and the environment posed by HAP emissions from EGUs remain after imposition of the requirements of the Clean Air Act. This confirmation was supported in part by several new analyses of the hazards to public health posed by both mercury and non-mercury HAP. For more information on the finding and the analyses to support them, please refer to the preamble of the final rule.

1.4 Provisions of the Final Mercury and Air Toxics Standards

1.4.1 What Is the Source Category Regulated by the Final Rule?

The final MATS addresses emissions from new and existing coal- and oil-fired EGUs. In general, if an EGU burns coal (either as a primary fuel or as a supplementary fuel) or any combination of coal with another fuel where the coal accounts for more than 10 percent of the average annual heat input during any 3 calendar years or for more than 15 percent of the annual heat input during any one calendar year, the unit is considered to be coal-fired under this final rule. If a unit is not a coal-fired unit and burns only oil or burns oil in combination with a fuel other than coal where the oil accounts for more than 10 percent of the average annual heat input during any 3 calendar years or for more than 15 percent of the annual heat input during any one calendar year, the unit is considered to be oil-fired under this final rule.

CAA section 112(a)(8) defines an EGU as:

> a fossil fuel-fired combustion unit of more than 25 megawatts electric (MWe) that serves a generator that produces electricity for sale. A unit that cogenerates steam and electricity and supplies more than one-third of its potential electric output capacity and more than 25 MWe output to any utility power distribution system for sale is also an electric utility steam generating unit.

This action established 40 CFR part 63, subpart UUUUU, to address HAP emissions from new and existing coal- and oil-fired EGUs. EPA must determine what is the appropriate maximum achievable control technology (MACT) for those units under sections 112(d)(2) and (d)(3) of the CAA.

EPA has divided coal- and oil-fired EGUs into the following subcategories:

- Units designed for not low rank virgin coal;

- units designed for low rank virgin coal;
- IGCC units;
- Solid oil-derived fuel-fired units;
- Continental liquid oil-fired units; and
- Non-continental liquid oil-fired units.

1.4.2 What Are the Pollutants Regulated by the Rule?

The final NESHAP regulates emissions of HAP. Available emissions data show that several HAP that are formed during the combustion process or which are contained within the fuel burned are emitted from coal- and oil-fired EGUs. The individual HAP include mercury, arsenic, cadmium, lead, and nickel, among others. EPA describes the health effects of these and other HAP emitted from the operation of coal- and oil-fired EGUs in Chapter 4 of this RIA. These HAP emissions are known to cause or contribute significantly to air pollution, which may reasonably be anticipated to endanger public health or welfare.

In addition to reducing HAP, the emission control technologies that will be installed on coal- and oil-fired EGUs to reduce HAP will also reduce sulfur dioxide (SO_2) and particulate matter (PM). A wide range of human health and welfare effects are linked to the emissions of PM and SO_2. These human health and welfare effects are discussed extensively in Chapter 5 of this RIA.

1.4.3 What Are the Emissions Limits?

Under section 112(d), EPA must establish emission standards for major sources that "require the maximum degree of reduction in emissions of the HAP subject to this section" that EPA determines is achievable taking into account certain statutory factors. These are referred to as maximum achievable control technology or MACT standards. The MACT standards for existing sources must be at least as stringent as the average emissions limitation achieved by the best performing 12 percent of existing sources in the category (for which the Administrator has emissions information) or the best performing 5 sources for source categories with less than 30 sources. This level of minimum stringency is referred to as the MACT floor, and EPA cannot consider cost in setting the floor. For new sources, MACT standards must be at least as stringent as the control level achieved in practice by the best controlled similar source.

The numerical emission standards that are being finalized for new and existing coal- and oil-fired EGUs units are shown in Tables 1-1 and 1-2. In some cases, affected sources have the

choice of complying with an emissions standard per unit of input or an output based standard, which are provided in parentheses below the input-based standard. These standards must be complied with on a 30-day rolling average basis if using continuous monitoring. If demonstrating compliance on the basis of a stack test, units must demonstrate compliance by conducting periodic stack tests on a quarterly basis.

Table 1-1. Emission Limitations for Coal-Fired and Solid Oil-Derived Fuel-Fired EGUs

Subcategory	Filterable Particulate Matter	Hydrogen Chloride	Mercury
Existing Unit designed for not low rank virgin coal	0.030 lb/MMBtu (0.30 lb/MWh)	0.0020 lb/MMBtu (0.020 lb/MWh)	1.2 lb/TBtu (0.020 lb/GWh)
Existing Unit designed for low rank virgin coal	0.030 lb/MMBtu (0.30 lb/MWh)	0.0020 lb/MMBtu (0.020 lb/MWh)	4.0 lb/TBtu[a] (0.040 lb/GWh[a])
Existing - IGCC	0.040 lb/MMBtu (0.40 lb/MWh)	0.00050 lb/MMBtu (0.0050 lb/MWh)	2.5 lb/TBtu (0.030 lb/GWh)
Existing – Solid oil-derived	0.0080 lb/MMBtu (0.090 lb/MWh)	0.0050 lb/MMBtu (0.080 lb/MWh)	0.20 lb/TBtu (0.0020 lb/GWh)
New unit designed for not low rank virgin coal	0.0070 lb/MWh	0.00040 lb/MWh	0.00020 lb/GWh
New unit designed for coal low rank virgin coal	0.0070 lb/MWh	0.00040 lb/MWh	0.040 lb/GWh
New – IGCC	0.070 lb/MWh[b] 0.090 lb/MWh[c]	0.0020 lb/MWh[d]	0.0030 lb/GWh[e]
New – Solid oil-derived	0.020 lb/MWh	0.00040 lb/MWh	0.0020 lb/GWh

Note: In some cases, affected units may comply with either an input-based standard or an output-based standard, shown in parentheses below the input-based standard.

lb/MMBtu = pounds pollutant per million British thermal units fuel input

lb/TBtu = pounds pollutant per trillion British thermal units fuel input

lb/MWh = pounds pollutant per megawatt-hour electric output (gross)

lb/GWh = pounds pollutant per gigawatt-hour electric output (gross)

[a] Beyond-the-floor limit. The MACT floor for this subcategory is 11.0 lb/TBtu (0.20 lb/GWh)

[b] Duct burners on syngas; based on permit levels in comments received

[c] Duct burners on natural gas; based on permit levels in comments received

[d] Based on best-performing similar source

[e] Based on permit levels in comments received

Table 1-2. Emission Limitations for Liquid Oil-Fired EGUs

Subcategory	Filterable PM	Hydrogen Chloride	Hydrogen Fluoride
Existing – Liquid oil-continental	0.030 lb/MMBtu (0.30 lb/MWh)	0.0020 lb/MMBtu (0.010 lb/MWh)	0.00040 lb/MMBtu (0.0040 lb/MWh)
Existing – Liquid oil-non-continental	0.030 lb/MMBtu (0.30 lb/MWh)	0.00020 lb/MMBtu (0.0020 lb/MWh)	0.000060 lb/MMBtu (0.00050 lb/MWh)
New – Liquid oil - continental	0.070 lb/MWh	0.00040 lb/MWh	0.00040 lb/MWh
New – Liquid oil - non-continental	0.20 lb/MWh	0.0020 lb/MWh	0.00050 lb/MWh

Note: In some cases, affected units may comply with either an input-based standard or an output-based standard, shown in parentheses below the input-based standard.

We are also finalizing alternate equivalent emission standards for certain subcategories in three areas: SO_2 (for HCl), individual non-Hg metals, and total non-Hg metals (for filterable PM) from coal- and solid oil-derived fuel-fired EGUs, and individual and total metals (for filterable PM) from oil-fired EGUs. These alternate emission limitations are provided in Tables 1-3 and 1-4. We are finalizing an alternate limitation of 1 percent moisture in the liquid oil as an alternate to the HCl and HF emission limits for both liquid oil subcategories (i.e., continental and non-continental).

Table 1-3. Alternate Emission Limitations for Existing Coal- and Oil-Fired EGUs

			Liquid Oil		
	Coal-fired EGUs	**IGCC**	**Continental**	**Non-continental**	**Solid Oil-derived**
SO_2	0.20 lb/MMBtu (1.5 lb/MWh)	NA	NA	NA	0.30 lb/MMBtu (2.0 lb/MWh)
Total non-Hg metals	0.000050 lb/MMBtu (0.50 lb/GWh)	0.000060 lb/MMBtu (0.50 lb/GWh)	0.00080 lb/MMBtu (0.0080 lb/MWh)[a]	0.00060 lb/MMBtu (0.0070 lb/MWh)[a]	0.000040 lb/MMBtu (0.6 lb/GWh)
Antimony, Sb	0.80 lb/TBtu (0.0080 lb/GWh)	1.4 lb/TBtu (0.020 lb/GWh)	13 lb/TBtu (0.20 lb/GWh)	2.2 lb/TBtu (0.020 lb/GWh)	0.80 lb/TBtu (0.0080 lb/GWh)
Arsenic, As	1.1 lb/TBtu (0.020 lb/GWh)	1.5 lb/TBtu (0.020 lb/GWh)	2.8 lb/TBtu (0.030 lb/GWh)	4.3 lb/TBtu (0.080 lb/GWh)	0.30 lb/TBtu (0.0050 lb/GWh)
Beryllium, Be	0.20 lb/TBtu (0.0020 lb/GWh)	0.10 lb/TBtu (0.0010 lb/GWh)	0.20 lb/TBtu (0.0020 lb/GWh)	0.60 lb/TBtu (0.0030 lb/GWh)	0.060 lb/TBtu (0.00060 lb/GWh)
Cadmium, Cd	0.30 lb/TBtu (0.0030 lb/GWh)	0.15 lb/TBtu (0.0020 lb/GWh)	0.30 lb/TBtu (0.0020 lb/GWh)	0.30 lb/TBtu (0.0030 lb/GWh)	0.30 lb/TBtu (0.0040 lb/GWh)
Chromium, Cr	2.8 lb/TBtu (0.030 lb/GWh)	2.9 lb/TBtu (0.030 lb/GWh)	5.5 lb/TBtu (0.060 lb/GWh)	31 lb/TBtu (0.30 lb/GWh)	0.8 lb/TBtu (0.020 lb/GWh)
Cobalt, Co	0.80 lb/TBtu (0.0080 lb/GWh)	1.2 lb/TBtu (0.020 lb/GWh)	21 lb/TBtu (0.30 lb/GWh)	110 lb/TBtu (1.40 lb/GWh)	1.1 lb/TBtu (0.020 lb/GWh)
Lead, Pb	1.2 lb/TBtu (0.020 lb/GWh)	190 lb/MMBtu (1.8 lb/MWh)	8.1 lb/TBtu (0.080 lb/GWh)	4.9 lb/TBtu (0.080 lb/GWh)	0.80 lb/TBtu (0.020 lb/GWh)
Manganese, Mn	4.0 lb/TBtu (0.050 lb/GWh)	2.5 lb/TBtu (0.030 lb/GWh)	22 lb/TBtu (0.30 lb/GWh)	20 lb/TBtu (0.30 lb/GWh)	2.3 lb/TBtu (0.040 lb/GWh)
Mercury, Hg	NA	NA	0.20 lb/TBtu (0.0020 lb/GWh)	0.040 lb/TBtu (0.00040 lb/GWh)	NA
Nickel, Ni	3.5 lb/TBtu (0.040 lb/GWh)	6.5 lb/TBtu (0.070 lb/GWh)	110 lb/TBtu (1.1 lb/GWh)	470 lb/TBtu (4.1 lb/GWh)	9.0 lb/TBtu (0.2 lb/GWh)
Selenium, Se	5.0 lb/TBtu (0.060 lb/GWh)	22 lb/TBtu (0.30 lb/GWh)	3.3 lb/TBtu (0.040 lb/GWh)	9.8 lb/TBtu (0.20 lb/GWh)	1.2 lb/TBtu (0.020 lb/GWh)

NA = Not applicable

[a] Includes Hg

Table 1-4. Alternate Emission Limitations for New Coal- and Oil-Fired EGUs

			Liquid Oil, lb/GWh		
	Coal-fired EGUs	IGCC[a]	Continental	Non-continental	Solid Oil-Derived
SO_2	0.40 lb/MWh	0.40 lb/MWh	NA	NA	0.40 lb/MWh
Total metals	0.060 lb/GWh	0.40 lb/GWh	0.00020 lb/MWh[b]	0.0070 lb/MWh[b]	0.60 lb/GWh
Antimony, Sb	0.0080 lb/GWh	0.020 lb/GWh	0.010	0.0080	0.0080 lb/GWh
Arsenic, As	0.0030 lb/GWh	0.020 lb/GWh	0.0030	0.060	0.0030 lb/GWh
Beryllium, Be	0.00060 lb/GWh	0.0010 lb/GWh	0.00050	0.0020	0.00060 lb/GWh
Cadmium, Cd	0.00040 lb/GWh	0.0020 lb/GWh	0.00020	0.0020	0.00070 lb/GWh
Chromium, Cr	0.0070 lb/GWh	0.040 lb/GWh	0.020	0.020	0.0060 lb/GWh
Cobalt, Co	0.0020 lb/GWh	0.0040 lb/GWh	0.030	0.30	0.0020 lb/GWh
Lead, Pb	0.0020 lb/GWh	0.0090 lb/GWh	0.0080	0.030	0.020 lb/GWh
Mercury, Hg	NA	NA	0.00010	0.00040	NA
Manganese, Mn	0.0040 lb/GWh	0.020 lb/GWh	0.020	0.10	0.0070 lb/GWh
Nickel, Ni	0.040 lb/GWh	0.070 lb/GWh	0.090	4.1	0.040 lb/GWh
Selenium, Se	0.0060 lb/GWh	0.30 lb/GWh	0.020	0.020	0.0060 lb/GWh

NA = Not applicable
[a] Based on best-performing similar source
[b] Includes Hg

EPA is finalizing a beyond-the-floor standard for Hg only of 4.0 lbs/trillion BTU for all existing and new units designed to burn low BTU virgin coal based on the availability of activated carbon injection (ACI) for cost-effective Hg control. When considering beyond-the-floor options, EPA must consider not only the maximum degree of reduction in emissions of HAP, but must take into account costs, energy, and non-air quality health and environmental impacts when doing so. We are finalizing a beyond-the-floor standard for these units because the Agency considers the cost of incremental reductions beyond the MACT floor standard of 11 lbs/trillion BTUs to be reasonable. While the primary IPM analysis discussed in Chapter 3 requires compliance with the beyond-the-floor limit, EPA performed a supplemental analysis at proposal that estimates the difference in impacts between regulating coal-fired units designed for lignite at the floor limit and at the beyond-the-floor limit modeled. This analysis (the IPM Beyond the Floor Cost TSD) shows that if the units were only required to meet a standard of 11 lbs/trillion BTUs, the units would emit approximately an additional 3,854 lbs at a

reduced annualized cost of $86.7 million. EPA also performed an analysis of beyond-the-floor alternatives which can be found in the Beyond the MACT Floor Analysis TSD. Based on these analyses, EPA concluded that the beyond-the-floor standard achieved significant additional benefits when compared to the costs of the standard.

Pursuant to CAA section 112(h), we are finalizing a work practice standard for organic HAP, including emissions of dioxins and furans, from all subcategories of EGU. The work practice standard being finalized for these EGUs would require the implementation of an annual performance test program as described the preamble. We are finalizing work practice standards because the data confirm that the significant majority of the measured organic HAP emissions from EGUs are below the detection levels of the EPA test methods, and, as such, EPA considers it impracticable to reliably measure emissions from these units.

The EGU NESHAP PM and SO_2 standards for new and modified facilities are as stringent or more stringent than the NSPS amendments. Thus, the only impacts unique to the NSPS amendments are those for the NOx emissions limits for new and modified facilities. In the baseline for this analysis and in compliance with MATS, no source is expected to trigger the NSPS limitations for new or modified sources. Therefore, we have concluded that there are no costs or benefits associated with the NSPS amendments that are unique to these amendments.[3] The NSPS requirements are described in detail in the preamble.

1.4.4 What are the Startup, Shutdown, and Malfunction Requirements?

Consistent with *Sierra Club* v. *EPA* (551 F.3d 1019 (DC Cir. 2008), cert. denied, 130 S. Ct. 1735 (U.S. 2010)), EPA proposed numerical emission standards that would apply at all times, including during periods of startup, shutdown, and malfunction. In this final rule, EPA has evaluated comments and other data concerning startup and shutdown periods and, for the reasons explained below, is establishing work practice standards for startup and shutdown periods as the terms are defined in the final rule.

EPA has revised this final rule to require sources to meet a work practice standard, which requires following the manufacturer's recommended procedures for minimizing periods of startup and shutdown, for all subcategories of new and existing coal- and oil-fired EGUs (that would otherwise be subject to numeric emission limits) during periods of startup and shutdown. As discussed elsewhere in the preamble, we considered whether performance testing, and therefore, enforcement of numeric emission limits, would be practicable during

[3] If the NESHAP requirements were not simultaneously analyzed with the NSPS amendments, then we would expect that the cost and benefits of the NSPS would be small.

periods of startup and shutdown. After reviewing comments and other data regarding the nature of these periods of operation, the EPA is finalizing a work practice standard for periods of start up and shut down. EPA will revisit this decision during the mandatory 8-year review cycle.

Periods of startup, normal operations, and shutdown are all predictable and routine aspects of a source's operations. However, by contrast, malfunction is defined as a "sudden, infrequent, and not reasonably preventable failure of air pollution control and monitoring equipment, process equipment or a process to operate in a normal or usual manner..." (40 CFR 63.2) EPA has determined that malfunctions should not be viewed as a distinct operating mode and, therefore, any emissions that occur at such times do not need to be factored into development of CAA section 112(d) standards, which, once promulgated, apply at all times.

In the event that a source fails to comply with the applicable CAA section 112(d) standards as a result of a malfunction event, EPA would determine an appropriate response based on, among other things, the good faith efforts of the source to minimize emissions during malfunction periods, including preventative and corrective actions, as well as root cause analyses to ascertain and rectify excess emissions. EPA would also consider whether the source's failure to comply with the CAA section 112(d) standard was, in fact, "sudden, infrequent, not reasonably preventable" and was not instead "caused in part by poor maintenance or careless operation" (40 CFR 63.2).

1.5 Baseline and Years of Analysis

The emissions scenarios for the RIA reflect the Cross-State Air Pollution Rule (CSAPR) as finalized in July 2011 and the emissions reductions of SOx, NOx, directly emitted PM, and CO_2 are consistent with application of federal rules, state rules and statutes, and other binding, enforceable commitments in place by December 2010 for the analysis timeframe. Consistent with the mercury risk deposition modeling for MATS, EPA did not model non-federally enforceable mercury-specific emissions reduction rules in the base case or MATS policy case (see preamble Section III.A for further detail). This approach does not significantly affect the projections underlying the cost and benefit results presented in this RIA. The baseline specifications used for these analyses are described in more detail in Chapter 3, Chapter 4, and Chapter 5 of this RIA. The EGU and non-EGU regulatory and air quality baseline used for the co-benefits analysis is described in Appendix 5A.

The costs and co-benefits from reductions in SO_2 and direct PM emissions are calculated using a baseline that includes the Cross State Air Pollution Rule (CSAPR; 76 FR 48208) finalized

July 6, 2011. EPA has subsequently proposed minor modifications to the state-level SO_2 budgets in CSAPR. These modifications are expected to result in small changes in the levels of SO_2 emission reductions in a number of states. These changes in the baseline levels of SO_2 emissions may result in slightly larger reductions in emissions and, consequently, slightly higher benefits being attributed to MATS. The impact on control costs is uncertain, but likely to be minimal given that only 1% of units are potentially affected. These modifications have not yet been finalized, but EPA expects the overall impact on MATS to be low.

Mercury reductions were not remodeled between the proposal and final rule for either the appropriate and necessary analysis or the RIA. As a result, the analysis presented in Chapter 4 uses the MATS proposal baseline that includes proposed, but not final, CSAPR, as well as the mercury standards as proposed rather than as finalized. Furthermore, there were some differences in the treatment of the baseline at proposal relative to the baseline included here in that it included non-federally enforceable state rules. These differences do not have a significant impact on total mercury emissions. Mercury benefits are magnitudes smaller than the co-benefits presented here and do not impact the final rounded benefits estimates.

The year 2016 is the compliance year for MATS, though as we explain in later chapters, we use 2015 as a proxy for compliance in 2016 for our cost analysis due to availability of modeling impacts in that year. All estimates presented in this report represent annualized estimates of the benefits and costs of the final MATS in 2016 rather than the net present value of a stream of benefits and costs in these particular years of analysis.

1.6 Benefits of Emission Controls

The benefits of the final MATS are discussed in Chapters 4 and 5 of this report. Annual monetized benefits of $37 to 90 billion (3 percent discount rate, 2007$) or $33 to 81 billion (7 percent discount rate, 2007$) are expected for the final rule in 2016.

Since the final rule requirements were finalized after the completion of the air quality modeling for this rule, EPA used benefit-per-ton (BPT) factors to quantify the changes in $PM_{2.5}$-related health impacts and monetized benefits based on changes in SO_2 and direct $PM_{2.5}$ emissions. These BPT factors were based on an interim baseline and policy scenario for which full-scale ambient air quality modeling and air quality-based human health benefits assessments were performed. These BPT estimates were then multiplied by the amount of emission reductions expected from MATS as finalized to estimate the benefits of the rule. The BPT approach is methodologically consistent with the technique reported in Fann, Fulcher, & Hubbell (2009), and has been used in previous RIAs, including the recent Ozone NAAQS RIA

(U.S. EPA, 2008), the NO_2 NAAQS RIA (U.S. EPA, 2010), the proposed Mercury and Air Toxics Standards RIA (U.S. EPA 2011a), and the Cross-State Air Pollution Rule (U.S. EPA, 2011b).

1.7 Cost of Emission Controls

EPA analyzed the costs of the final MATS using the Integrated Planning Model (IPM). EPA has used this model in the past to analyze the impacts of regulations on the power sector, including the proposed and final CSAPR and proposed MATS. EPA estimates the annual incremental compliance costs of the rule to the power sector to be $9.6 billion in 2016 (2007$).[4] A description of the methodology used to model the costs and economic impacts to the power sector is discussed in Chapter 3 of this report. A description of how the employment impacts associated with this final rule are estimated is provided in Chapter 6 of this report.

1.8 Organization of the Regulatory Impact Analysis

This report presents EPA's analysis of the benefits, costs, and other economic effects of the final MATS to fulfill the requirements of a Regulatory Impact Analysis (RIA). This RIA includes the following chapters:

- Chapter 2, Electric Power Sector Profile, describes the industry affected by the rule.
- Chapter 3, Cost, Economic, and Energy Impacts, describes the modeling conducted to estimate the cost, economic, and energy impacts to the power sector.
- Chapter 4, Mercury and Other HAP Benefits Analysis, describes the methodology and results of the benefits analysis for mercury and other HAP.
- Chapter 5, Co-Benefits Analysis, describes the methodology and results of the benefits analysis for $PM_{2.5}$, Ozone, and other benefit categories.
- Chapter 6, Employment and Economic Impacts, describes the analysis to estimate the employment impacts and economic impacts associated with the final rule.
- Chapter 7, Statutory and Executive Order Impact Analyses, describes the small business, unfunded mandates, paperwork reduction act, environmental justice, and other analyses conducted for the rule to meet statutory and Executive Order requirements.

[4] This total includes compliance costs of $9.4 billion modeled in IPM for coal fired EGUs, monitoring, recordkeeping, and reporting costs of $158 million, and compliance costs modeled in a separate analysis for oil-fired EGUs of $56 million.

- Chapter 8, Comparison of Benefits and Costs, shows a comparison of the total benefits to total costs of the rule.

1.9 References

Fann, N., C.M. Fulcher, B.J. Hubbell. 2009. The influence of location, source, and emission type in estimates of the human health benefits of reducing a ton of air pollution. Air Qual Atmos Health 2:169–176.

U.S. Environmental Protection Agency (U.S. EPA). 2008a. Regulatory Impact Analysis, 2008 National Ambient Air Quality Standards for Ground-level Ozone, Chapter 6. Office of Air Quality Planning and Standards, Research Triangle Park, NC. March. Available at <http://www.epa.gov/ttn/ecas/regdata/RIAs/6-ozoneriachapter6.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2010. Final Regulatory Impact Analysis (RIA) for the NO_2 National Ambient Air Quality Standards (NAAQS). Office of Air Quality Planning and Standards, Research Triangle Park, NC. January. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/RIAs/FinalNO2RIAfulldocument.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2011a. Proposed Regulatory Impact Analysis (RIA) for the Toxics Rule. Office of Air Quality Planning and Standards, Research Triangle Park, NC. March. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/RIAs/ToxicsRuleRIA.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2011b. Regulatory Impact Analysis for the Federal Implementation Plans to Reduce Interstate Transport of Fine Particulate Matter and Ozone in 27 States; Correction of SIP Approvals for 22 States. Office of Air Quality Planning and Standards, Research Triangle Park, NC. June. Available on the Internet at <http://www.epa.gov/airtransport/pdfs/FinalRIA.pdf>.

CHAPTER 2
ELECTRIC POWER SECTOR PROFILE

2.1 Introduction

This chapter discusses important aspects of the power sector that relate to the final MATS Rule, including the types of power-sector sources affected by the rule, and provides background on the power sector and electric generating units (EGUs). In addition, this chapter provides some historical background on EPA regulation of and future projections for the power sector. The specific impacts of MATS are discussed in Chapter 3.

2.2 Power Sector Overview

The production and delivery of electricity to customers consists of three distinct segments: generation, transmission, and distribution.

2.2.1 Generation

Electricity generation is the first process in the delivery of electricity to consumers. Most of the existing capacity for generating electricity involves creating heat to rotate turbines which, in turn, create electricity. The power sector consists of over 17,000 generating units, comprising fossil-fuel-fired units, nuclear units, and hydroelectric and other renewable sources dispersed throughout the country (see Table 2-1).

Table 2-1. Existing Electricity Generating Capacity by Energy Source, 2009

Energy Source	Number of Generators	Generator Nameplate Capacity (MW)	Generator Net Summer Capacity (MW)
Coal	1,436	338,723	314,294
Petroleum	3,757	63,254	56,781
Natural Gas	5,470	459,803	401,272
Other Gases	98	2,218	1,932
Nuclear	104	106,618	101,004
Hydroelectric Conventional	4,005	77,910	78,518
Wind	620	34,683	34,296
Solar Thermal and Photovoltaic	110	640	619
Wood and Wood Derived Fuels	353	7,829	6,939
Geothermal	222	3,421	2,382
Other Biomass	1,502	5,007	4,317
Pumped Storage	151	20,538	22,160
Other	48	1,042	888
Total	17,876	1,121,686	1,025,402

Source: EIA (2009).

These electric generating sources provide electricity for commercial, industrial, and residential uses, each of which consumes roughly a quarter to a third of the total electricity produced (see Table 2-2). Some of these uses are highly variable, such as heating and air conditioning in residential and commercial buildings, while others are relatively constant, such as industrial processes that operate 24 hours a day.

Table 2-2. Total U.S. Electric Power Industry Retail Sales in 2009 (Billion kWh)

		Sales/Direct Use (Billion kWh)	Share of Total End Use
Retail Sales	Residential	1,364	37%
	Commercial	1,307	35%
	Industrial	917	25%
	Transportation	8	0.2%
Direct Use		127	3%
Total End Use		**3,723**	100%

Source: EIA (2009).

In 2009, electric generating sources produced 3,950 billion kWh to meet electricity demand. Roughly 70 percent of this electricity was produced through the combustion of fossil fuels, primarily coal and natural gas, with coal accounting for the largest single share (see Table 2-3).

Table 2-3. Electricity Net Generation in 2009 (Billion kWh)

	Net Generation (Billion kWh)	Fuel Source Share
Coal	1,756	44.5%
Petroleum	39	1.0%
Natural Gas	921	23.3%
Other Gases	11	0.3%
Nuclear	799	20.2%
Hydroelectric	273	6.9%
Other	151	3.8%
Total	**3,950**	**100%**

Source: EIA (2009).

Note: Retail sales are not equal to net generation because net generation includes net exported electricity and loss of electricity that occurs through transmission and distribution.

Coal-fired generating units typically supply "base-load" electricity, the portion of electricity loads which are continually present, and typically operate throughout the day. Along with nuclear generation, these coal units meet the part of demand that is relatively constant. Although much of the coal fleet operates as base load, there can be notable differences across various facilities (see Table 2-4). For example, coal-fired units less than 100 MW in size compose 37 percent of the total number of coal-fired units, but only 6 percent of total coal-fired capacity. Gas-fired generation is better able to vary output and is the primary option used to meet the variable portion of the electricity load and typically supplies "peak" power, when there is increased demand for electricity (for example, when businesses operate throughout the day or when people return home from work and run appliances and heating/air-conditioning), versus late at night or very early in the morning, when demand for electricity is reduced. However, the evolving economics of the power sector, in particular the increased natural gas supply and relatively low natural gas prices, have resulted in more gas being utilized as base load energy. Figure 2-1 shows the distribution and relative size of the fossil-fuel fired generating capacity across the United States.

Table 2-4. Coal Steam Electricity Generating Units, by Size, Age, Capacity, and Efficiency (Heat Rate)

Unit Size Grouping (MW)			No. Units	% of All Units	Avg. Age	Avg. Net Summer Capacity (MW)	Total Net Summer Capacity (MW)	% Total Capacity	Avg. Heat Rate (Btu/kWh)
0	*to*	25	193	15%	45	15	2,849	1%	11,154
>25	*to*	49	108	9%	42	38	4,081	1%	11,722
50	*to*	99	162	13%	47	75	12,132	4%	11,328
100	*to*	149	269	21%	49	141	38,051	12%	10,641
150	*to*	249	81	6%	43	224	18,184	6%	10,303
250	*and up*		453	36%	34	532	241,184	76%	10,193
Total			**1,266**				**316,480**		

Source: National Electric Energy Data System (NEEDS) v.4.10

Note: The average heat rate reported is the mean of the heat rate of the units in each size category (as opposed to a generation-weighted or capacity-weighted average heat rate.) A lower heat rate indicates a higher level of fuel efficiency. Table is limited to coal-steam units online in 2010 or earlier, and excludes those units with planned retirements.

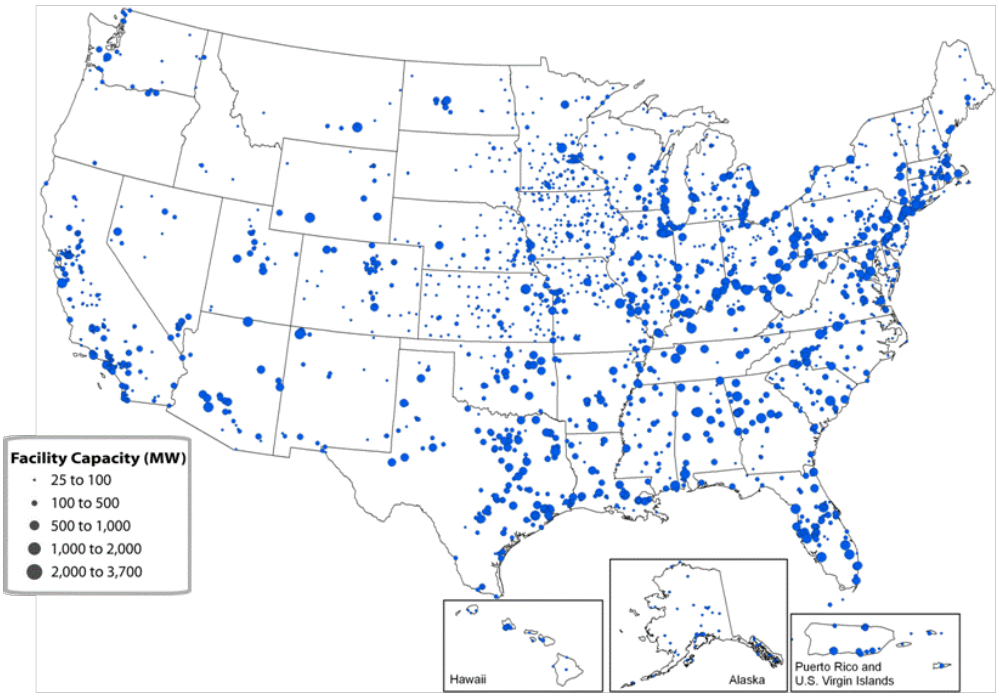

Figure 2-1. Fossil Fuel-Fired Electricity Generating Facilities, by Size

Source: National Electric Energy Data System (NEEDS) 4.10

Note: This map displays facilities in the NEEDS 4.10 IPM frame. NEEDS reflects available capacity on-line by the end of 2011. This includes planned new builds and planned retirements. In areas with a dense concentration of facilities, some facilities may be obscured.

2.2.2 Transmission

Transmission is the term used to describe the movement of electricity over a network of high voltage lines, from electric generators to substations where power is stepped down for local distribution. In the US and Canada, there are three separate interconnected networks of high voltage transmission lines,[1] each operating at a common frequency. Within each of these transmission networks, there are multiple areas where the operation of power plants is monitored and controlled to ensure that electricity generation and load are kept in balance. In some areas, the operation of the transmission system is under the control of a single regional operator; in others, individual utilities coordinate the operations of their generation, transmission, and distribution systems to balance their common generation and load needs.

[1] These three network interconnections are the western US and Canada, corresponding approximately to the area west of the Rocky Mountains; eastern US and Canada, not including most of Texas; and a third network operating in most of Texas. These are commonly referred to as the Western Interconnect Region, Eastern Interconnect Region, and ERCOT, respectively.

2.2.3 Distribution

Distribution of electricity involves networks of lower voltage lines and substations that take the higher voltage power from the transmission system and step it down to lower voltage levels to match the needs of customers. The transmission and distribution system is the classic example of a natural monopoly, in part because it is not practical to have more than one set of lines running from the electricity generating sources to substations or from substations to residences and business.

Transmission has generally been developed by the larger vertically integrated utilities that typically operate generation and distribution networks. Distribution is handled by a large number of utilities that often purchase and sell electricity, but do not generate it. Over the last couple of decades, several jurisdictions in the United States began restructuring the power industry to separate transmission and distribution from generation, ownership, and operation. As discussed below, electricity restructuring has focused primarily on efforts to reorganize the industry to encourage competition in the generation segment of the industry, including ensuring open access of generation to the transmission and distribution services needed to deliver power to consumers. In many states, such efforts have also included separating generation assets from transmission and distribution assets to form distinct economic entities. Transmission and distribution remain price-regulated throughout the country based on the cost of service.

2.3 Deregulation and Restructuring

The process of restructuring and deregulation of wholesale and retail electric markets has changed the structure of the electric power industry. In addition to reorganizing asset management between companies, restructuring sought a functional unbundling of the generation, transmission, distribution, and ancillary services the power sector has historically provided, with the aim of enhancing competition in the generation segment of the industry.

Beginning in the 1970s, government policy shifted against traditional regulatory approaches and in favor of deregulation for many important industries, including transportation (notably commercial airlines), communications, and energy, which were all thought to be natural monopolies (prior to 1970) that warranted governmental control of pricing. However, deregulation efforts in the power sector were most active during the 1990s. Some of the primary drivers for deregulation of electric power included the desire for more efficient investment choices, the economic incentive to provide least-cost electric rates through market competition, reduced costs of combustion turbine technology that opened the door for

more companies to sell power with smaller investments, and complexity of monitoring utilities' cost of service and establishing cost-based rates for various customer classes.

The pace of restructuring in the electric power industry slowed significantly in response to market volatility in California and financial turmoil associated with bankruptcy filings of key energy companies. By the end of 2001, restructuring had either been delayed or suspended in eight states that previously enacted legislation or issued regulatory orders for its implementation (shown as "Suspended" in Figure 2-2 below). Another 18 other states that had seriously explored the possibility of deregulation in 2000 reported no legislative or regulatory activity in 2001 (EIA, 2003) ("Not Active" in Figure 2-2 below). Currently, there are 15 states where price deregulation of generation (restructuring) has occurred ("Active" in Figure 2-2 below). Power sector restructuring is more or less at a standstill; there have been no recent proposals to the Federal Energy Regulatory Commission (FERC) for actions aimed at wider restructuring, and no additional states have recently begun retail deregulation activity.

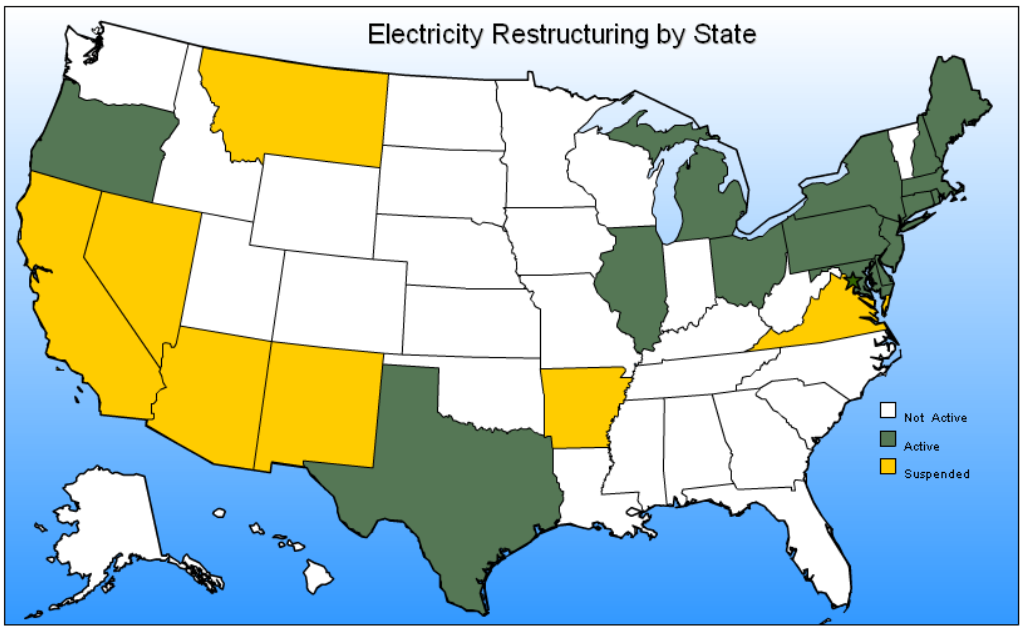

Figure 2-2. Status of State Electricity Industry Restructuring Activities

Source: EIA (2010b).

2.4 Emissions of Mercury and Other Hazardous Air Pollutants from Electric Utilities

The burning of fossil fuels, which generates about 70 percent of our electricity nationwide, results in air emissions of Hazardous Air Pollutants (HAPs): mercury, acid gasses, and non-mercury metallic particulates. Additionally, SO_2 and NO_x emissions from the power

sector are important precursors in the formation of fine particles and ozone (NO_x only). The power sector is a major contributor of all of these pollutants.

The Emissions Overview Memorandum Technical Support Document (TSD) to the proposed air toxics standards (Docket number EPA-HQ-OAR-2009-0234) details the emissions of mercury and other HAPs emitted by EGUs. In 2005, EGU emissions of mercury accounted for approximately half of all anthropogenic mercury emissions in the U.S. Table 2-5 shows the trend in EGU and total anthropogenic mercury emissions from 1990–2005 and EGU mercury emissions reported in the Utility MACT Information Collection Request (ICR) in 2010.

Table 2-5. U.S. Anthropogenic Mercury Emissions, 1990–2010

	1990 (tons)	1999 (tons)	2005 (tons)	2010[a] (tons)
EGU Hg Emissions	59	49	53	29
Non-EGU Hg Emission	205	66	52	Not Available[b]
Total U.S. Hg Emissions	**264**	**115**	**105**	**Not Available[b]**

[a] The estimate of the current level of Hg emissions based on the 2010 ICR database may underestimate total EGU Hg emissions due to targeting of the 2010 ICR on the best performing EGUs.

[b] Information on recent U.S. EGU emissions was obtained using an ICR for EGUs only. This same information is not available for other sources, which were not covered by the ICR.

In 2005, EGUs contributed 82 percent of U.S. hydrogen chloride emissions. Table 2-6 shows the total HCl emissions from EGU and non-EGU sources in 2005 and the EGU HCl emissions reported in the Utility MACT ICR in 2010.

Table 2-6. U.S. Hydrogen Chloride Emissions, 2005 and 2010

	2005[a] (tons)	2010[b] (tons)
EGU HCl Emissions	350,000	106,000
Non-EGU HCl Emissions	78,000	Not Available[c]
Total U.S. HCl Emissions	**428,000**	**Not Available[c]**

[a] 2005 emissions from the National Air Toxics Assessment Inventory. Available online at http://www.epa.gov/ttn/atw/nata2005/. EGU emissions were extracted from the total using the MACT code field (1808).

[b] The estimate of the current level of Hg emissions based on 2010 may underestimate the total EGU emissions due to targeting of the 2010 ICR on the best performing EGUs.

[c] Information on recent U.S. EGU emissions was obtained using an ICR for EGUs only. This same information is not available for other sources, which were not covered by the ICR.

Individual fossil fuel-fired units vary widely in their air emissions levels for HAPs, particularly when uncontrolled. In 2010, as reported in the Utility MACT ICR, mercury emissions range from less than 0.3 lb/trillion Btu (TBtu) to more than 20 lbs/TBtu. HCl emissions from coal-fired units range from less than 0.00002 lb/million Btu (mmBtu) (for a unit with a scrubber) to over 0.1 lb/mmBtu. Additionally, emissions of fine particulates less than or equal to 2.5 microns ($PM_{2.5}$) range from 0.002 lb/mmBtu to over 0.06 lb/mmBtu. For an uncontrolled plant, mercury, acid gas, and particulate emissions are directly related to the elemental profile and ash content of the coal burned.

Oil-fired units also have a wide range of HAP emissions. Based on the Utility MACT ICR, Mercury emissions range from less than 0.01 lb/TBtu to more than 60 lbs/TBtu. HCl emissions from oil-fired units range from less than 0.00001 lb/mmBtu (for a unit with a scrubber) to over 0.003 lb/mmBtu. Emissions of $PM_{2.5}$ range from less than 0.004 lb/mmBtu to over 0.07 lb/mmBtu.

2.5 Pollution Control Technologies

Acid gas HAPs (e.g., hydrogen chloride (HCl), hydrogen fluoride (HF), sulfur dioxide (SO_2)) from coal-fired power plants can be controlled by fuel selection, fuel blending, or post combustion controls. Fossil fuels, particularly coal, vary widely in the content of pollutants like chlorine (Cl), fluorine (F), sulfur (S) and other HAPs, making fuel blending and/or switching an effective method for reducing emissions of HAPs. In general, it is easier to switch fuels within a coal rank (rather than across a coal rank) due to similar heat contents and other characteristics. Switching fuels across ranks tends to trigger more costly modifications. As a compromise, blending is employed when a complete fuel switch adversely affects the unit. EGUs may also choose to retrofit post combustion controls to achieve superior pollutant removal. Post-combustion controls typically remove larger proportions of HCl and HF than SO_2 due to differences in molecular weight.

Acid gas emissions (including SO_2) can be reduced with flue gas desulfurization (FGD, also known as “scrubbers”) or with dry sorbent injection (DSI). EGUs may choose either “wet” or “dry” configurations of scrubbers. Wet scrubbers can use a variety of reagents including crushed limestone, quick lime, and magnesium-enhanced lime. The choice of reagent affects performance, size, capital and operating costs. Current wet scrubber technology is capable of removing at least 99 percent of HF and HCl emissions while simultaneously achieving 96 percent SO_2 removal. Modern dry FGD technology combines lime-based slurry with a downstream fabric filter to remove at least 93 percent SO_2 while also capturing over 99 percent

HCL and HF. An alternative to scrubber technology is dry sorbent injection (DSI), which injects an alkaline powdered material (post combustion) to react with the acid gases. The product of this reaction is removed by particulate matter (PM) control device. DSI technology is most efficient with a baghouse present downstream but can function with an electrostatic precipitator (ESP) downstream as well. Under these circumstances, the ESP requires more reagent per molecule of acid gas removed as compared to a similar operation with a baghouse. Finally, DSI may employ a multitude of sorbents (trona,[2] sodium carbonate, calcium carbonate—and their bicarbonate counterparts) for a more tailored approach to reduce emissions based on the source, cost, and unit and fuel characteristics.

Mercury capture and removal requires multiple controls. Upon combustion, mercury exits the furnace in three forms: elemental, oxidized, and as a particulate. Elemental mercury is emitted out of the stack. The particulate form is bound to the ash and removed by PM control equipment such as ESP or fabric filter. A portion of mercury that has converted to oxidized compounds may be removed by either a wet scrubber or by activated carbon injection (ACI). Each of these control devices uses a different method to remove the mercury compounds. The wet FGD system captures oxidized mercury because it is water soluble, while activated carbon injection provides a unique physical surface to which oxidized mercury can adhere. Mercury oxidation can occur at multiple locations within a unit as long as an oxidizing agent, generally a halogen, is present for reaction. This allows the unit operator some latitude in selecting a control method and injection point based on existing equipment at the particular source. A halogen can be introduced to the fuel prior to combustion, injected directly into the furnace, introduced upstream of a selective catalytic reduction (SCR) system,[3] or infused with the activated carbon injections. The unit operator may also increase halogens by blending in higher chlorine fuels (e.g., Powder River Basin fuel blended with bituminous coal). Operating a wet FGD for SO_2 control alongside selective catalytic reduction (SCR) for NO_x control with sufficient halogen present will remove more than 90 percent of the mercury within the flue gas stream. Alternatively, in the absence of a wet FGD, activated carbon injection (ACI) can be employed for mercury capture with at least 90 percent removal using a downstream fabric filter. An ESP results in less efficient mercury removal with ACI.

Non-mercury heavy metals and organics are removed by PM control equipment such as fabric filters and ESP. Unlike mercury, the heavy metals (e.g., selenium and arsenic) are non-volatile and affix to the ash. Likewise, any organics surviving the high temperature combustion

[2] Trona refers to the chemical compound sodium sesquicarbonate.
[3] SCR is primarily used for NO_x control, but can also be used to promote mercury oxidation.

process are non-volatile and bind to the ash. Both control technologies are capable of removing more than 99 percent of $PM_{2.5}$ mass from the emissions stream. ESPs sap relatively little energy from the flue gas but are less flexible for fuel switching, since they are designed for use with a specific intended fuel. Fuel switching or blending that increases gas flow rate, ash resistivity, or particle loading may render an existing ESP insufficient for removing particulate matter. ESPs also suffer from ash re-entrainment, which is the release of particulate matter from the last compartment due to the self cleaning action. On the other hand, an ESP with sufficient design margin may succeed with these fuel alterations. Conversely, a fabric filter does not suffer from these limitations. Moreover, the fabric filter readily lends itself to mercury and acid gas removal since DSI and ACI operate more efficiently with a baghouse. When considering retrofit PM control options, a unit with an existing ESP will examine upgrading the precipitator as an alternative to installing a new fabric filter to achieve emission reductions.

For more detail on the cost and performance assumptions of pollution controls, see the documentation for the Integrated Planning Model (IPM),[4] a dynamic linear programming model that EPA uses to examine air pollution control policies for various air emissions throughout the United States for the entire power system.

2.6 HAP Regulation in the Power Sector

2.6.1 Programs Targeting HAP

In 2000, EPA made a finding that it was appropriate and necessary to regulate coal- and oil-fired EGUs under CAA section 112 and listed EGUs pursuant to CAA section 112(c). This finding triggered a requirement for EPA to propose regulations to control air toxics emissions, including mercury, from these facilities.

On January 30, 2004, EPA proposed a rule with two basic approaches for controlling mercury from power plants. One approach would require power plants to meet emissions standards reflecting the application of the "maximum achievable control technology" (MACT) determined according to the procedure set forth in section 112(d) of the Clean Air Act. A second approach proposed by EPA would create a market-based "cap and trade" program that, if implemented, would reduce nationwide utility emissions of mercury in two phases under Section 111 or Section 112 of the Clean Air Act. EPA also proposed to revise its December 2000 finding that it is "appropriate and necessary" to regulate utility hazardous air emissions using the MACT standards provisions (section 112) of the Clean Air Act.

[4] Documentation for IPM can be found at www.epa.gov/airmarkets/epa-ipm.

On March 15, 2005, EPA issued the final Clean Air Mercury Rule (CAMR). CAMR established "standards of performance" limiting mercury emissions from new and existing utilities and created a market-based cap-and-trade program to reduce nationwide utility emissions of mercury in two phases. In conjunction with CAMR, EPA published a final rule (Section 112(n) Revision Rule) that removed EGUs from the list of sources for which regulation under CAA section 112 was required.

The Section 112(n) Revision Rule was vacated on February 8, 2008, by the U.S. Court of Appeals for the District of Columbia Circuit. As a result of that vacatur, CAMR was also vacated and EGUs remained on the list of sources that must be regulated under CAA section 112. This action finalizes the rule EPA proposed on March 16, 2011 to replace CAMR in response to the court's decisions.

2.6.2 Programs Targeting SO_2 and NOx

Programs to reduce SO_2 and NOx also impact emissions of mercury and other HAP. At the federal level, efforts to reduce emissions of SO_2 have been occurring since 1970. Policy makers have recognized the need to address these harmful emissions, and incremental steps have been taken to ensure that the country meets air quality standards. The recently finalized Cross State Air Pollution Rule (CSAPR) is the next step toward attainment of the national standards for $PM_{2.5}$ and ozone.

Even before widespread regulation of SO_2 and NO_X for the power sector, total suspended particulate matter (TSP) was a related target of state and federal action. Because larger particulates are visible as dark smoke from smokestacks, most states had regulations by 1970 limiting the opacity of emissions. Requirements for taller smokestacks also mitigated local impacts of TSP. Notably, such regulations effectively addressed large-diameter, filterable particulate matter rather than condensable particulate matter (such as $PM_{2.5}$) associated with SO_2 and NO_X emissions, which are not visible at the smokestack and have impacts far from their sources.

Federal regulation of SO_2 and NO_X emissions at power plants began with the 1970 Clean Air Act. The Act required the Agency to develop New Source Performance Standards (NSPS) for a number of source categories including coal-fired power plants. The first NSPS for power plants (subpart D) required new units to limit SO_2 emissions either by using scrubbers or by using low sulfur coal. NO_X was required to be limited through the use of low NO_X burners. A new NSPS (subpart Da), promulgated in 1978, tightened the standards for SO_2, requiring scrubbers on all new units.

The 1990 Clean Air Act Amendments (CAAA) placed a number of new requirements on power plants. The Acid Rain Program, established under Title IV of the 1990 CAAA, requires major reductions of SO_2 and NO_X emissions. The SO_2 program sets a permanent cap on the total amount of SO_2 that can be emitted by electric power plants in the contiguous United States at about one-half of the amount of SO_2 these sources emitted in 1980. Using a market-based cap and trade mechanism allows flexibility for individual combustion units to select their own methods of compliance with the SO_2 reduction requirements. The program uses a more traditional approach to NO_X emissions limitations for certain coal-fired electric utility boilers, with the objective of achieving a 2 million ton reduction from projected NO_X emission levels that would have been emitted in 2000 without implementation of Title IV.

The Acid Rain Program comprises two phases for SO_2 and NO_X. Phase I applied primarily to the largest coal-fired electric generating sources from 1995 through 1999 for SO_2 and from 1996 through 1999 for NO_X. Phase II for both pollutants began in 2000. For SO_2, it applies to thousands of combustion units generating electricity nationwide; for NO_X it generally applies to affected units that burned coal during 1990 through 1995. The Acid Rain Program has led to the installation of a number of scrubbers on existing coal-fired units as well as significant fuel switching to lower sulfur coals. Under the NO_X provisions of Title IV, most existing coal-fired units installed low NO_X burners.

The CAAA also placed much greater emphasis on control of NO_X to reduce ozone nonattainment. This led to the formation of several regional NO_X trading programs as well as intrastate NO_X trading programs in states such as Texas. The northeastern states of the Ozone Transport Commission (OTC) required existing sources to meet Reasonably Available Control Technology (RACT) limits on NO_X in 1995 and in 1999 began an ozone-season cap and trade program to achieve deeper reductions. In 1998, EPA promulgated regulations (the NO_X SIP Call) that required 21 states in the eastern United States and the District of Columbia to reduce NO_X emissions that contributed to nonattainment in downwind states using the cap and trade approach. This program began in May of 2003 and has resulted in the installation of significant amounts of selective catalytic reduction.

The Clean Air Interstate Rule (CAIR) built on EPA's efforts in the NO_X SIP call to address specifically interstate pollution transport for ozone, and was EPA's first attempt to address interstate pollution transport for $PM_{2.5}$. It required significant reductions in emissions of SO_2 and NO_X in 28 states and the District of Columbia (see Figure 6-4 below). EGUs were found to be a major source of the SO_2 and NO_X emissions which contributed to fine particle concentrations and ozone problems downwind. Although the D.C. Circuit remanded the rule to

EPA in 2008, it did so without vacatur, allowing the rule to remain in effect while EPA addressed the remand. Thus, CAIR continued to help states address ozone and $PM_{2.5}$ nonattainment and improve visibility by reducing transported precursors of SO_2 and NO_X through the implementation of three separate cap and trade compliance programs for annual NO_X, ozone season NO_X, and annual SO_2 emissions from power plants.

Perhaps in anticipation of complying with CAIR, especially the more stringent second phase that was set to begin in 2015, several sources began installing or planning to install advanced controls for SO_2 and NO_X to begin operating in the 2010 to 2015 timeframe. Many EPA New Source Review (NSR) settlements also required controls in those years, as do state rules in Georgia, Illinois, and Maryland. States like North Carolina, New York, Connecticut, Massachusetts, and Delaware have also moved to control these emissions to address nonattainment.

On July 6, 2011, the EPA finalized the Cross-State Air Pollution Rule (CSAPR) to replace CAIR. The rule requires states to eliminate the portion of their emissions defined as their "significant contribution" by setting a pollution limit (or budget) for each covered state. The rule allows air-quality-assured allowance trading among covered sources, utilizing an allowance market infrastructure based on existing, successful allowance trading programs. The final CSAPR allows sources to trade emissions allowances with other sources within the same program (e.g., ozone season NOx) in the same or different states, while firmly constraining any emissions shifting that may occur by requiring a strict emission ceiling in each state (the budget plus variability limit). It also includes assurance provisions that ensure each state will make the emission reductions necessary to fulfill the "good neighbor" provision of the Clean Air Act.

2.7 Revenues, Expenses, and Prices

Due to lower retail electricity sales, total utility operating revenues declined in 2009 to $276 billion from a peak of almost $300 billion in 2008. However, operating expenses were appreciably lower and as a result, net income actually rose modestly compared to 2008 (see Table 2-7). Recent economic events have put downward pressure on electricity demand, thus dampening electricity prices and consumption (utility revenues), but have also reduced the price and cost of fossil fuels and other expenses. Electricity sales and revenues associated with the generation, transmission, and distribution of electricity are expected to rebound and increase modestly by 2015, where they are projected to be roughly $360 billion (see Table 2-8).

Based on EIA's Annual Energy Outlook 2011, Table 2-8 shows that in the base case, the power sector is expected to derive revenues of $360 billion in 2015. Table 2-7 shows that

investor-owned utilities (IOUs) earned income of about 11.5% compared to total revenues in 2009. Assuming the same income ratio from IOUs (with no income kept by public power), and using the same proportion of power sales from public power as observed in 2009, EPA projects that the power sector will expend over $320 billion in 2015 alone to generate, transmit, and distribute electricity to end-use consumers.

Over the past 50 years, real retail electricity prices have ranged from around 7 cents per kWh in the early 1970s, to around 11 cents, reached in the early 1980s. Generally, retail electricity prices do not change rapidly and do not display the variability of other energy or commodity prices, although the frequency at which these prices change varies across different types of customers. Retail rate regulation has largely insulated consumers from the rising and falling wholesale electricity price signals whose variation in the marketplace on an hourly, daily, and seasonal basis is critical for driving lowest-cost matching of supply and demand. In fact, the real price of electricity today is lower than it was in the early 1960s and 1980s (see Figure 2-3).

Table 2-7. Revenue and Expense Statistics for Major U.S. Investor-Owned Electric Utilities for 2009 ($millions)

	2008	2009
Utility Operating Revenues	**298,962**	**276,124**
Electric Utility	266,124	249,303
Other Utility	32,838	26,822
Utility Operating Expenses	**267,263**	**244,243**
Electric Utility	236,572	219,544
Operation	175,887	154,925
Production	140,974	118,816
Cost of Fuel	47,337	40,242
Purchased Power	84,724	67,630
Other	8,937	10,970
Transmission	6,950	6,742
Distribution	3,997	3,947
Customer Accounts	5,286	5,203
Customer Service	3,567	3,857
Sales	225	178
Administrative and General	14,718	15,991
Maintenance	14,192	14,092
Depreciation	19,049	20,095
Taxes and Other	26,202	29,081
Other Utility	30,692	24,698
Net Utility Operating Income	**31,699**	**31,881**

Source: EIA (2009).

Note: These data do not include information for public utilities.

Table 2-8. Projected Revenues by Service Category in 2015 for Public Power and Investor-Owned Utilities (billions)

Generation	$195
Transmission	36
Distribution	129
Total	**$360**

Source: EIA (2011).

Note: Data are derived by taking either total electricity use (for generation) or sales (transmission and distribution) and multiplying by forecasted prices by service category from Table 8 (Electricity Supply, Disposition, Prices, and Emissions).

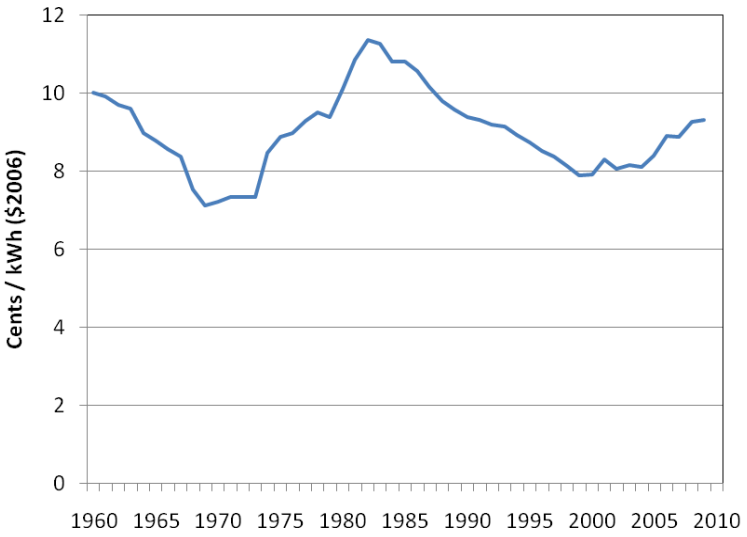

Figure 2-3. National Average Retail Electricity Price (1960–2009)

Source: EIA (2009).

On a state-by-state basis, retail electricity prices vary considerably. The Northeast and California have average retail prices that can be as much as double those of other states (see Figure 2-4).

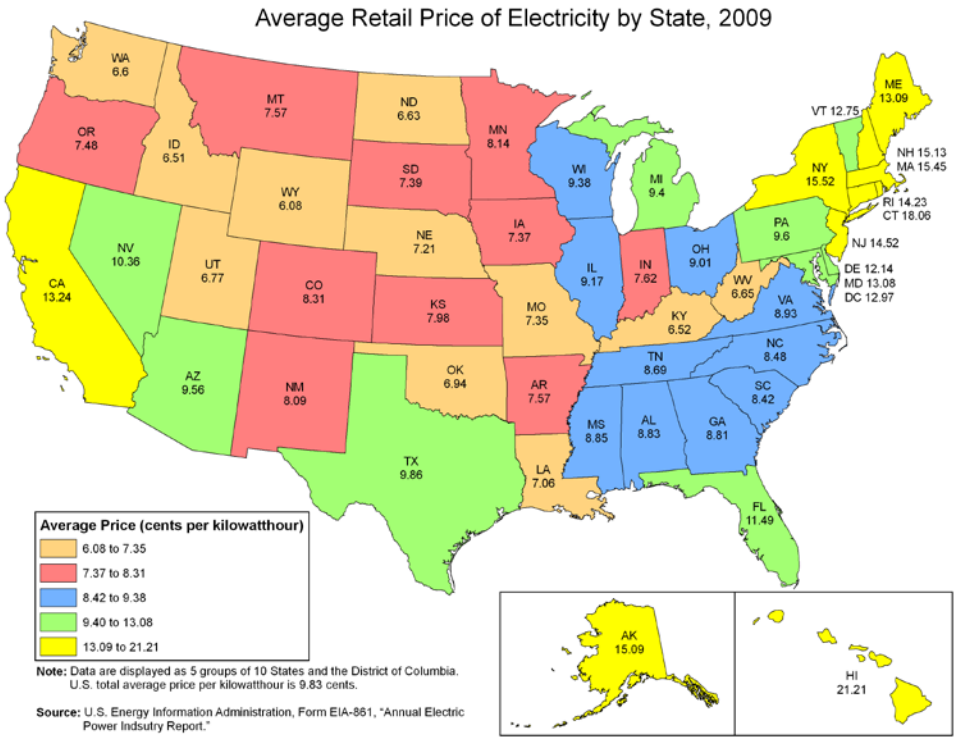

Figure 2-4. Average Retail Electricity Price by State (cents/kWh), 2009

Source: EIA (2009).

2.7.1 Natural Gas Market

The natural gas market in the United States has historically experienced significant price volatility from year to year, between seasons within a year, and can undergo major price swings during short-lived weather events (such as cold snaps leading to short-run spikes in heating demand). Over the last decade, gas prices (both Henry Hub[5] prices and delivered prices to the power sector) have ranged from $3 per mmBtu to as high as $9 on an annual average basis (see Figure 2-5). During that time, the daily price of natural gas reached as high as $15/mmBtu. Recent forecasts of natural gas have also experienced considerable revision as new sources of gas have been discovered and have come to market, although there continues to be some uncertainty surrounding the precise quantity of the resource base.[6]

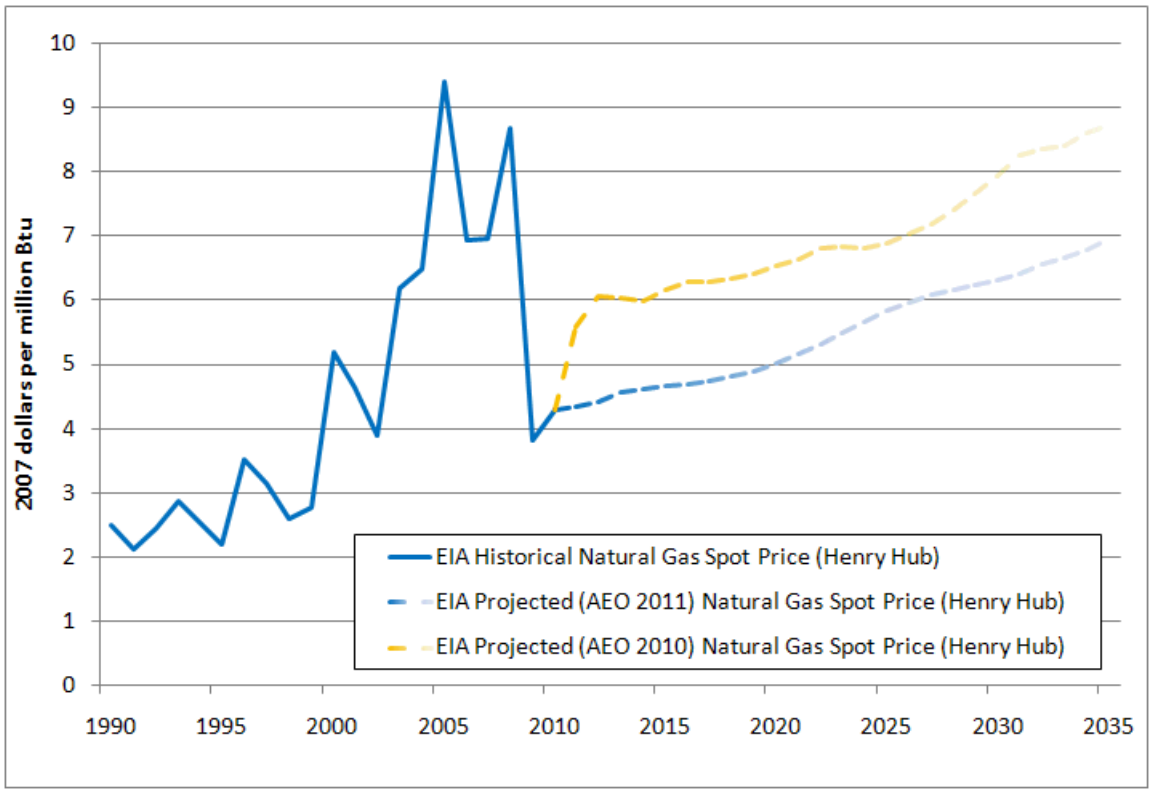

Figure 2-5. Natural Gas Spot Price, Annual Average (Henry Hub)

Source: EIA (2010a), EIA (2011).

[5] The Henry Hub is the pricing point for natural gas futures contracts traded on the New York Mercantile Exchange. It is a point on the natural gas pipeline system that interconnects nine interstate and four intrastate pipelines.

[6] In August, EIA announced it would lower its previous estimates of recoverable shale gas by nearly 80 percent. EPA's modeling of the natural gas market is discussed in more detail in Chapter 7 of this RIA.

2.8 Electricity Demand and Demand Response

Electricity performs a vital and high-value function in the economy. Historically, growth in electricity consumption has been closely aligned with economic growth. Overall, the U.S. economy has become more efficient over time, producing more output (GDP) per unit of energy input, with per capita energy use fairly constant over the past 30 years. The growth rate of electricity demanded has also been in overall decline for the past sixty years (see Figure 2-8), with several key drivers that are worth noting. First, there has been a significant structural shift in the U.S. economy towards less energy-intensive sectors, like services. Second, companies have strong financial incentives to reduce energy expenditures. Third, companies are responding to the marketplace and continually develop and bring to market new technologies that reduce energy consumption. Fourth, other policies, such as energy efficiency standards at the state and Federal level, have helped address certain market failures. These broader changes have altered the outlook for future electricity growth (see Figure 2-6).

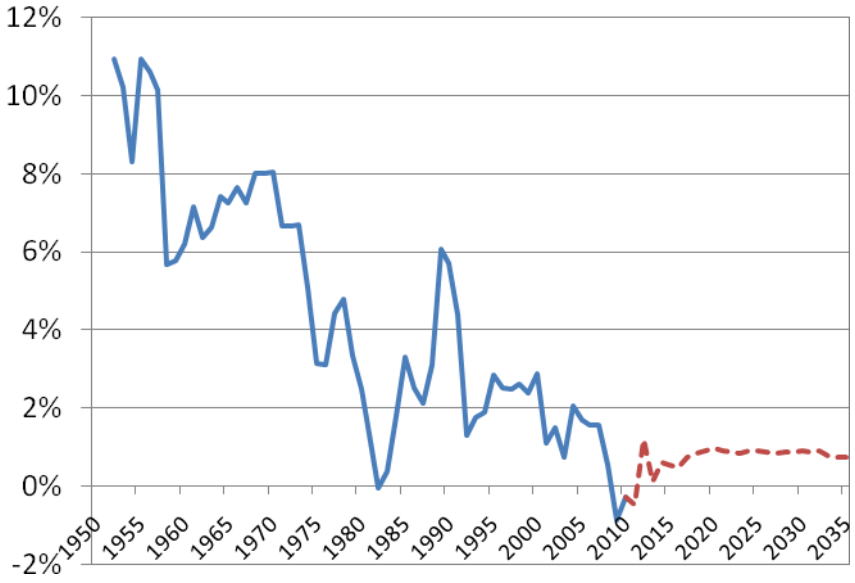

Figure 2-6. Electricity Growth Rate (3 Year Rolling Average) and Projections from the Annual Energy Outlook 2011

Source: EIA (2009), EIA (2011).

Energy efficiency initiatives have become more common, and investments in energy efficiency are projected to continue to increase for the next 5 to 10 years, driven in part by the growing number of states that have adopted energy efficiency resource standards.[7] These investments, and other energy efficiency policies at both the state and federal level, create incentives to reduce energy consumption and peak load. According to EIA, demand-side management provided actual peak load reductions of 31.7 GW in 2009. For context, the current coal fleet is roughly 320 GW of capacity.

Demand for electricity, especially in the short run, is not very sensitive to changes in prices and is considered relatively price inelastic, although some demand reduction does occur in response to price. With that in mind, EPA modeling does not typically incorporate a "demand response" in its electric generation modeling (Chapter 3) to the increases in electricity prices typically projected for EPA rulemakings. Electricity demand is considered to be constant in EPA modeling applications and the reduction in production costs that would result from lower demand is not considered in the primary analytical scenario that is modeled. This leads to some overstatement in the private compliance costs that EPA estimates. Notably, the "compliance costs" are the changes in the electric power generation costs in the base case and pollution control options that are evaluated in Chapter 3. In simple terms, it is the resource costs of what the power industry will directly expend to comply with EPA's requirements.

2.9 References

U.S. Energy Information Administration (U.S. EIA). Electric Power Annual 2003. 2003. Available online at: http://www.eia.gov/oiaf/archive/aeo03/index.html.

U.S. Energy Information Administration (U.S. EIA). *Electric Power Annual 2009*. 2009. Available online at: http://www.eia.doe.gov/cneaf/electricity/epa/epa_sum.html.

U.S. Energy Information Administration (U.S. EIA). *Annual Energy Outlook 2010*. 2010a. Available online at: http://www.eia.gov/oiaf/archive/aeo10/index.html.

U.S. Energy Information Administration (U.S. EIA). "Status of Electricity Restructuring by State." 2010b. Available online at: http://www.eia.gov/cneaf/electricity/page/restructuring/restructure_elect.html.

U.S. Energy Information Administration (U.S. EIA). *Annual Energy Outlook 2011*. 2011. Available online at: http://www.eia.gov/forecasts/aeo/.

[7] To the extent that EIA includes these measures in its baseline forecast from the Annual Energy Outlook, EPA has also incorporated them into the baseline for purposes of assessing the economic impacts of this rule. See AEO 2011 and Chapter 3 and the IPM documentation for more detail.

CHAPTER 3
COST, ECONOMIC, AND ENERGY IMPACTS

This chapter reports the compliance cost, economic, and energy impact analysis performed for the Mercury and Air Toxics Standards (MATS). EPA used the Integrated Planning Model (IPM), developed by ICF Consulting, to conduct its analysis. IPM is a dynamic linear programming model that can be used to examine air pollution control policies for SO_2, NO_x, Hg, HCl, and other air pollutants throughout the United States for the entire power system. Documentation for IPM can be found at http://www.epa.gov/airmarkets/progsregs/epa-ipm, and updates specific to the MATS modeling are in the "Documentation Supplement for EPA Base Case v.4.10_MATS – Updates for Final Mercury and Air Toxics Standards (MATS) Rule" (hereafter IPM 4.10 Supplemental Documentation for MATS).

3.1 Background

Over the last decade, EPA has on several occasions used IPM to consider pollution control options for reducing power-sector emissions.[1] Most recently EPA used IPM extensively in the development and analysis of the impacts of the Cross-State Air Pollution Rule (CSAPR).[2] As discussed in Chapter 2, MATS coincides with a period when many new pollution controls are being installed. Many are needed for compliance with NSR settlements and state rules, while others may have been planned in expectation of CAIR and its replacement, the CSAPR.

The emissions scenarios for the RIA reflects the Cross-State Air Pollution Rule (CSAPR) as finalized in July 2011 and the emissions reductions of SO_X, NO_X, directly emitted PM, and CO_2 are consistent with application of federal rules, state rules and statutes, and other binding, enforceable commitments in place by December 2010 for the analysis timeframe.[3]

[1] Many EPA analyses with IPM have focused on legislative proposals with national scope, such as EPA's IPM analyses of the Clean Air Planning Act (S.843 in 108th Congress), the Clean Power Act (S.150 in 109th Congress), the Clear Skies Act of 2005 (S.131 in 109th Congress), the Clear Skies Act of 2003 (S.485 in 108th Congress), and the Clear Skies Manager's Mark (of S.131). These analyses are available at EPA's website: (http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html). EPA also analyzed several multi-pollutant reduction scenarios in July 2009 at the request of Senator Tom Carper to illustrate the costs and benefits of multiple levels of SO_2 and NO_X control in the power sector.

[2] Additionally, IPM has been used to develop the NO_X Budget Trading Program, the Clean Air Interstate Rule programs, the Clean Air Visibility Programs, and other EPA regulatory programs for the last 15 years.

[3] Consistent with the mercury risk deposition modeling for MATS, EPA did not model non-federally enforceable mercury-specific emissions reduction rules in the base case or MATS policy case (see preamble section III.A). Note that this approach does not significantly affect SO_2 and NO_X projections underlying the cost and benefit results presented in this RIA

EPA has made these base case assumptions recognizing that the power sector will install a significant amount of pollution controls in response to several requirements. The inclusion of CSAPR and other regulatory actions (including federal, state, and local actions) in the base case is necessary in order to reflect the level of controls that are likely to be in place in response to other requirements apart from MATS. This base case will provide meaningful projections of how the power sector will respond to the cumulative regulatory requirements for air emissions in totality, while isolating the incremental impacts of MATS relative to a base case with other air emission reduction requirements separate from today's action.

The model's base case features an updated Title IV SO_2 allowance bank assumption and incorporates updates related to the Energy Independence and Security Act of 2007. Some modeling assumptions, most notably the projected demand for electricity, are based on the 2010 Annual Energy Outlook from the Energy Information Administration (EIA). In addition, the model includes existing policies affecting emissions from the power sector: the Title IV of the Clean Air Act (the Acid Rain Program); the NO_x SIP Call; various New Source Review (NSR) settlements[4]; and several state rules[5] affecting emissions of SO_2, NO_x, and CO_2 that were finalized through June of 2011. IPM includes state rules that have been finalized and/or approved by a state's legislature or environmental agency, with the exception of non-federal mercury-specific rules. The IPM 4.10 Supplemental Documentation for MATS contains details on all of these other legally binding and enforceable commitments for installation and operation of pollution controls. This chapter focuses on results of EPA's analysis with IPM for the model's 2015 run-year in connection with the compliance date for MATS.

MATS establishes National Emissions Standards for Hazardous Air Pollutants (NESHAPS) for the "electric utility steam generating unit" source category, which includes those units that combust coal or oil for the purpose of generating electricity for sale and distribution through the national electric grid to the public.

[4] The NSR settlements include agreements between EPA and Southern Indiana Gas and Electric Company (Vectren), Public Service Enterprise Group, Tampa Electric Company, We Energies (WEPCO), Virginia Electric & Power Company (Dominion), Santee Cooper, Minnkota Power Coop, American Electric Power (AEP), East Kentucky Power Cooperative (EKPC), Nevada Power Company, Illinois Power, Mirant, Ohio Edison, Kentucky Utilities, Hoosier Energy, Salt River Project, Westar, Puerto Rico Power Authority, Duke Energy, American Municipal Power, and Dayton Power and Light. These agreements lay out specific NO_x, SO_2, and other emissions controls for the fleets of these major Eastern companies by specified dates. Many of the pollution controls are required between 2010 and 2015.

[5] These include current and future state programs in Alabama, Arizona, California, Colorado, Connecticut, Delaware, Georgia, Illinois, Kansas, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Montana, New Hampshire, New Jersey, New York, North Carolina, Oregon, Pennsylvania, Tennessee, Texas, Utah, Washington, West Virginia, and Wisconsin the cover certain emissions from the power sector.

Coal-fired electric utility steam generating units include electric utility steam generating units that burn coal, coal refuse, or a synthetic gas derived from coal either exclusively, in any combination together, or in any combination with other supplemental fuels. Examples of supplemental fuels include petroleum coke and tire-derived fuels. The NESHAP establishes standards for HAP emissions from both coal- and oil-fired EGUs and will apply to any existing, new, or reconstructed units located at major or area sources of HAP. Although all HAP are pollutants of interest, those of particular concern are hydrogen fluoride (HF), hydrogen chloride (HCl), dioxins/furans, and HAP metals, including antimony, arsenic, beryllium, cadmium, chromium, cobalt, mercury, manganese, nickel, lead, and selenium.

This rule affects any fossil fuel fired combustion unit of more than 25 megawatts electric (MWe) that serves a generator that produces electricity for sale. A unit that cogenerates steam and electricity and supplies more than one-third of its potential electric output capacity and more than 25 MWe output to any utility power distribution system for sale is also considered an electric utility steam generating unit. The rule affects roughly 1,400 EGUs: approximately 1,100 existing coal-fired generating units and 300 oil-fired steam units, should those units combust oil. Of the 600 power plants potentially covered by this rule, about 430 have coal-fired units only, 30 have both coal- and oil- or gas-fired steam units, and 130 have oil- or gas-fired steam units only. Note that only steam electric units combusting coal or oil are covered by this rule.

EPA analyzed for the RIA the input-based (lbs/MMBtu) MATS control requirements shown in Table 3-1. In this analysis, EPA does not model an alternative SO_2 standard. Coal steam units with access to lignite in the modeling are subjected to the “Existing coal-fired unit low Btu virgin coal” standard. For further discussion about the scope and requirements of MATS, see the preamble or Chapter 1 of this RIA.

Table 3-1. Emissions Limitations for Coal-Fired and Solid Oil-Derived Fuel-Fired Electric Utility Steam Generating Units

Subcategory	Filterable Particulate Matter	Hydrogen Chloride	Mercury
Existing coal-fired unit not low Btu virgin coal	0.030 lb/MMBtu (0.30 lb/MWh)	0.0020 lb/MMBtu (0.020 lb/MWh)	1.2 lb/TBtu (0.020 lb/GWh)
Existing coal-fired unit low Btu virgin coal	0.030 lb/MMBtu (0.30 lb/MWh)	0.0020 lb/MMBtu (0.020 lb/MWh)	11.0 lb/TBtu (0.20 lb/GWh) 4.0 lb/TBtu[a] (0.040 lb/GWh[a])
Existing - IGCC	0.040 lb/MMBtu (0.40 lb/MWh)	0.00050 lb/MMBtu (0.0050 lb/MWh)	2.5 lb/TBtu (0.030 lb/GWh)
Existing – Solid oil-derived	0.0080 lb/MMBtu (0.090 lb/MWh)	0.0050 lb/MMBtu (0.080 lb/MWh)	0.20 lb/TBtu (0.0020 lb/GWh)
New coal-fired unit not low Btu virgin coal	0.0070 lb/MWh	0.40 lb/GWh	0.00020 lb/GWh
New coal-fired unit low Btu virgin coal	0.0070 lb/MWh	0.40 lb/GWh	0.040 lb/GWh
New – IGCC	0.070 lb/MWh[b] 0.090 lb/MWh[c]	0.0020 lb/MWh[d]	0.0030 lb/GWh[e]
New – Solid oil-derived	0.020 lb/MWh	0.00040 lb/MWh	0.0020 lb/GWh

Note: lb/MMBtu = pounds pollutant per million British thermal units fuel input
lb/TBtu = pounds pollutant per trillion British thermal units fuel input
lb/MWh = pounds pollutant per megawatt-hour electric output (gross)
lb/GWh = pounds pollutant per gigawatt-hour electric output (gross)
[a] Beyond-the-floor limit as discussed elsewhere
[b] Duct burners on syngas; based on permit levels in comments received
[c] Duct burners on natural gas; based on permit levels in comments received
[d] Based on best-performing similar source
[e] Based on permit levels in comments received

Table 3-2. Emissions Limitations for Liquid Oil-Fired Electric Utility Steam Generating Units

Subcategory	Filterable PM	Hydrogen Chloride	Hydrogen Fluoride
Existing – Liquid oil-continental	0.030 lb/MMBtu (0.30 lb/MWh)	0.0020 lb/MMBtu (0.010 lb/MWh)	0.00040 lb/MMBtu (0.0040 lb/MWh)
Existing – Liquid oil-non-continental	0.030 lb/MMBtu (0.30 lb/MWh)	0.00020 lb/MMBtu (0.0020 lb/MWh)	0.000060 lb/MMBtu (0.00050 lb/MWh)
New – Liquid oil – continental	0.070 lb/MWh	0.00040 lb/MWh	0.00040 lb/MWh
New – Liquid oil – non-continental	0.20 lb/MWh	0.0020 lb/MWh	0.00050 lb/MWh

EPA used the Integrated Planning Model (IPM) v.4.10 to assess the impacts of the MATS emission limitations for coal-fired electricity generating units (EGU) in the contiguous United States. IPM modeling did not subject oil-fired units to policy criteria.[6] Furthermore, IPM modeling did not include generation outside the contiguous U.S., where EPA is aware of only 2 facilities that would be subject to the coal-fired requirements of the final rule. Given the limited number of potentially impacted facilities, limited availability of input data to inform the modeling, and limited connection to the continental grid, EPA did not model the impacts of the rule beyond the contiguous U.S.

Mercury emissions are modeled as a function of mercury content of the fuel type(s) consumed at each plant in concert with that plant's pollutant control configuration. HCl emissions are projected in a similar fashion using the chlorine content of the fuel(s). For both mercury and HCl, EGUs in the model must emit at or below the final mercury and HCl emission rate standards in order to operate from 2015 onwards. EGUs may change fuels and/or install additional control technology to meet the standard, or they may choose to retire if it is more economic for the power sector to meet electricity demand with other sources of generation. See IPM 4.10 documentation and IPM 4.10 Supplemental Documentation for MATS for more details.

Total PM emissions are calculated exogenously to IPM, using EPA's Source Classification Code (SCC) and control-based emissions factors. SCC is a classification system that describes a generating unit's characteristics.

[6] EPA did not model the impacts of MATS on oil-fired units using IPM. Rather, EPA performed an analysis of impacts on oil-fired units for the final rule. The results are summarized in Appendix 3A.

Instead of emission limitations for the organic HAP, EPA is proposing that if requested, owners or operators of EGUs submit to the delegated authority or EPA, as appropriate, documentation showing that an annual performance test meeting the requirements of the rule was conducted. IPM modeling of the MATS policy assumes compliance with these work practice standards.

Electricity demand is anticipated to grow by roughly 1 percent per year, and total electricity demand is projected to be 4,103 billion kWh by 2015. Table 3-3 shows current electricity generation alongside EPA's base case projection for 2015 generation using IPM. EPA's IPM modeling for this rule relies on EIA's *Annual Energy Outlook for 2010*'s electric demand forecast for the US and employs a set of EPA assumptions regarding fuel supplies and the performance and cost of electric generation technologies as well as pollution controls.[7] The base case includes CSAPR as well as other existing state and federal programs for air emissions control from electric generating units, with the exception of state mercury rules.

[7] Note that projected electricity demand in AEO 2010 is about 2% higher than the AEO 2011 projection in 2015. Since this RIA assumes higher electricity demand in 2015 than is shown in the latest AEO projection, it is possible that the model may be taking compliance actions to meet incremental electricity demand that may not actually occur, and projected compliance costs may therefore be somewhat overstated in this analysis.

Table 3-3. 2009 U.S. Electricity Net Generation and EPA Base Case Projections for 2015-2030 (Billion kWh)

	Historical	Base Case		
	2009	**2015**	**2020**	**2030**
Coal	1,741	1,982	2,002	2,027
Oil	36	0.11	0.13	0.21
Natural Gas	841	710	847	1,185
Nuclear	799	828	837	817
Hydroelectric	267	286	286	286
Non-hydro Renewables	116	252	289	333
Other	10	45	45	55
Total	3,810	**4,103**	**4,307**	**4,702**

Source: 2009 data from AEO Annual Energy Review, Table 8.2c Electricity Net Generation: Electric Power Sector by Plant Type, 1989-2010; Projections from Integrated Planning Model run by EPA, 2011.

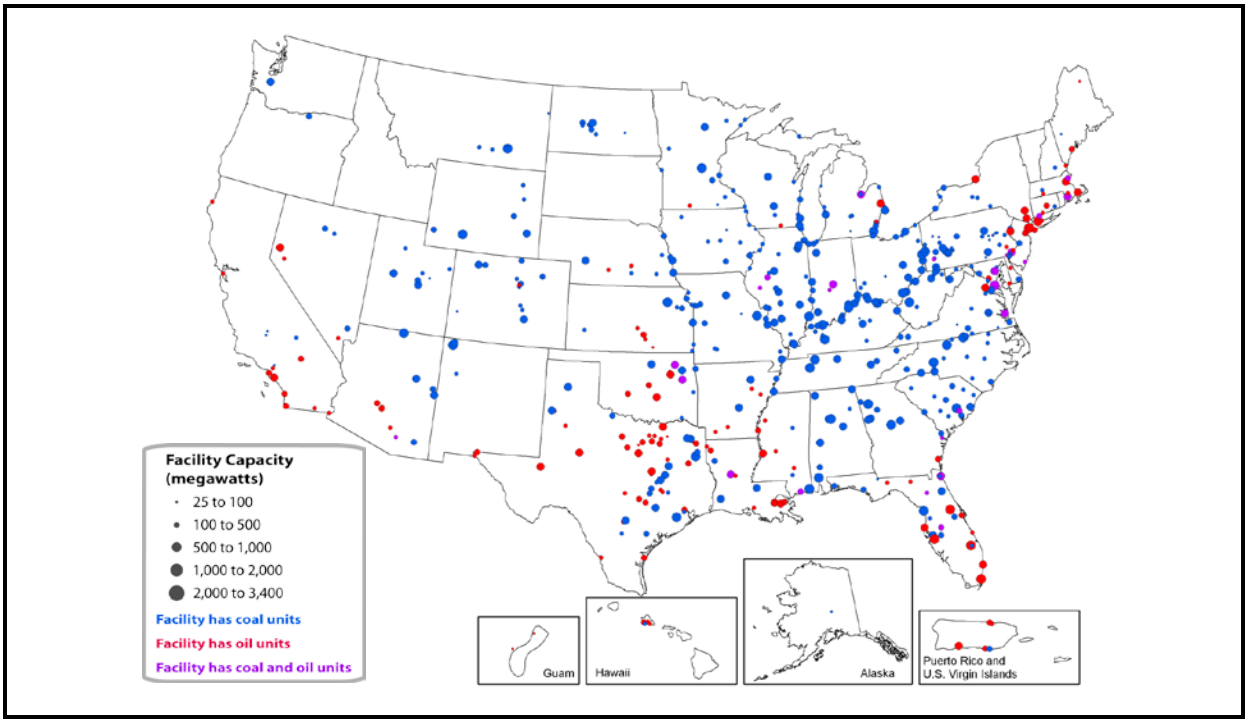

Figure 3-1. Geographic Distribution of Affected Units, by Facility, Size and Fuel Source in 2012

Source/Notes: National Electric Energy Data System (NEEDS 4.10 MATS) (EPA, December 2011) and EPA's Information Collection Request (ICR) for New and Existing Coal- And Oil-Fired Electric Utility Stream Generation Units (2010). This map displays facilities that are included in the NEEDS 4.10 MATS data base and that contain at least one oil-fired steam generating unit or one coal-fired steam generating unit that generates more than 25 megawatts of power. This includes coal-fired units that burn petroleum coke and that turn coal into gas before burning (using integrated gasification combined cycle or IGCC). NEEDS reflects available capacity on-line by the end of 2011; this includes committed new builds and committed retirements of old units. Only coal and oil-fired units are covered by this rule. Some of the oil units displayed on the map are capable of burning oil and/or gas. If a unit burns only gas, it will not be covered in the rule. In areas with a dense concentration of facilities, the facilities on the map may overlap and some may be impossible to see. IPM modeling did not include generation outside the contiguous U.S., where EPA is aware of only two facilities that would be subject to the coal-fired requirements of the final rule. Given the limited number of potentially impacted facilities, limited availability of input data to inform the modeling, and limited connection to the continental grid, EPA did not model the impacts of the rule beyond the contiguous U.S. Facilities outside the contiguous U.S. are displayed based on data from EPA's 2010 ICR for the rule.

As noted above, IPM has been used for evaluating the economic and emission impacts of environmental policies for over two decades. The economic modeling presented in this chapter has been developed for specific analyses of the power sector. Thus, the model has been designed to reflect the industry as accurately as possible. To that end, EPA uses a series of capital charge factors in IPM that embody financial terms for the various types of investments that the power sector considers for meeting future generation and environmental constraints.

The model applies a discount rate of 6.15% for optimizing the sector's decision-making over time. IPM's discount rate, designed to represent a broad range of private-sector decisions for power generation, rates differs from discount rates used in other analyses in this RIA, such as the benefits analysis which each assume alternative social discount rates of 3% and 7%. These discount rates represent social rates of time preference, whereas the discount rate in IPM represents an empirically-informed price of raising capital for the power sector. Like all other assumed price inputs in IPM, EPA uses the best available information from utilities, financial institutions, debt rating agencies, and government statistics as the basis for the capital charge rates and the discount rate used for power sector modeling in IPM.

More detail on IPM can be found in the model documentation, which provides additional information on the assumptions discussed here as well as all other assumptions and inputs to the model (http://www.epa.gov/airmarkets/progsregs/epa-ipm). Updates specific to MATS modeling are also in the IPM 4.10 Supplemental Documentation for MATS.

3.2 Projected Emissions

MATS is anticipated to achieve substantial emissions reductions from the power sector. Since the technologies available to meet the emission reduction requirements of the rule reduce multiple air pollutants, EPA expects the rule to yield a broad array of pollutant reductions from the power sector. The primary pollutants of concern under MATS from the power sector are mercury, acid gases such as hydrogen chloride (HCl), and HAP metals, including antimony, arsenic, beryllium, cadmium, chromium, cobalt, mercury, manganese, nickel, lead, and selenium. EPA has extensively analyzed mercury emissions from the power sector, and IPM modeling assesses the mercury contents in all coals and the removal efficiencies of relevant emission control technologies (e.g., ACI). EPA also models emissions and the pollution control technologies associated with HCl (as a surrogate for acid gas emissions). Like SO_2, HCl is removed by both scrubbers and DSI (dry sorbent injection). Projected emissions are based on both control technology and detailed coal supply curves used in the model that reflect the chlorine content of coals, which corresponds with the supply region, coal grade, and sulfur, mercury, and ash content of each coal type. This information is critical for accurately projecting future HCl emissions, and for understanding how the power sector will respond to a policy requiring reductions of multiple HAPs.

Generally, existing pollution control technologies reduce emissions across a range of pollutants. For example, both FGD and SCR can achieve notable reductions in mercury in addition to their primary targets of SO_2 and NO_X reductions. DSI will reduce HCl emissions while

also yielding substantial SO_2 emission reductions, but is not assumed in EPA modeling to result in mercury reductions. Since there are many avenues to reduce emissions, and because the power sector is a highly complex and dynamic industry, EPA employs IPM in order to reflect the relevant components of the power sector accurately, while also providing a sophisticated view of how the industry could respond to particular policies to reduce emissions. For more detail on how EPA models emissions from the power sector, including recent updates to include acid gases, see IPM 4.10 Supplemental Documentation for MATS.

Under MATS, EPA projects annual HCl emissions reductions of 88 percent in 2015, Hg emissions reductions of 75 percent in 2015, and $PM_{2.5}$ emissions reductions of 19 percent in 2015 from coal-fired EGUs greater than 25 MW. In addition, EPA projects SO_2 emission reductions of 41 percent, and annual CO_2 reductions of 1 percent from coal-fired EGUs greater than 25 MW by 2015, relative to the base case (see Table 3-4).[8] Mercury emission projections in EPA's base case are affected by the incidental capture in other pollution control technologies (such as FGD and SCR) as described above.

Table 3-4. Projected Emissions of SO_2, NO_X, Mercury, Hydrogen Chloride, PM, and CO_2 with the Base Case and with MATS, 2015

		Million Tons			Thousand Tons		CO_2
		SO_2	NO_X	Mercury (Tons)	HCl	$PM_{2.5}$	(Million Metric Tonnes)
Base	All EGUs	3.4	1.9	28.7	48.7	277	2,230
	Covered EGUs	3.3	1.7	26.6	45.3	270	1,906
MATS	All EGUs	2.1	1.9	8.8	9.0	227	2,215
	Covered EGUs	1.9	1.7	6.6	5.5	218	1,882

Source: Integrated Planning Model run by EPA, 2011

[8] The CO_2 emissions reported from IPM account for the direct CO_2 emissions from fuel combustion and CO_2 created from chemical reactions in pollution controls to reduced sulfur.

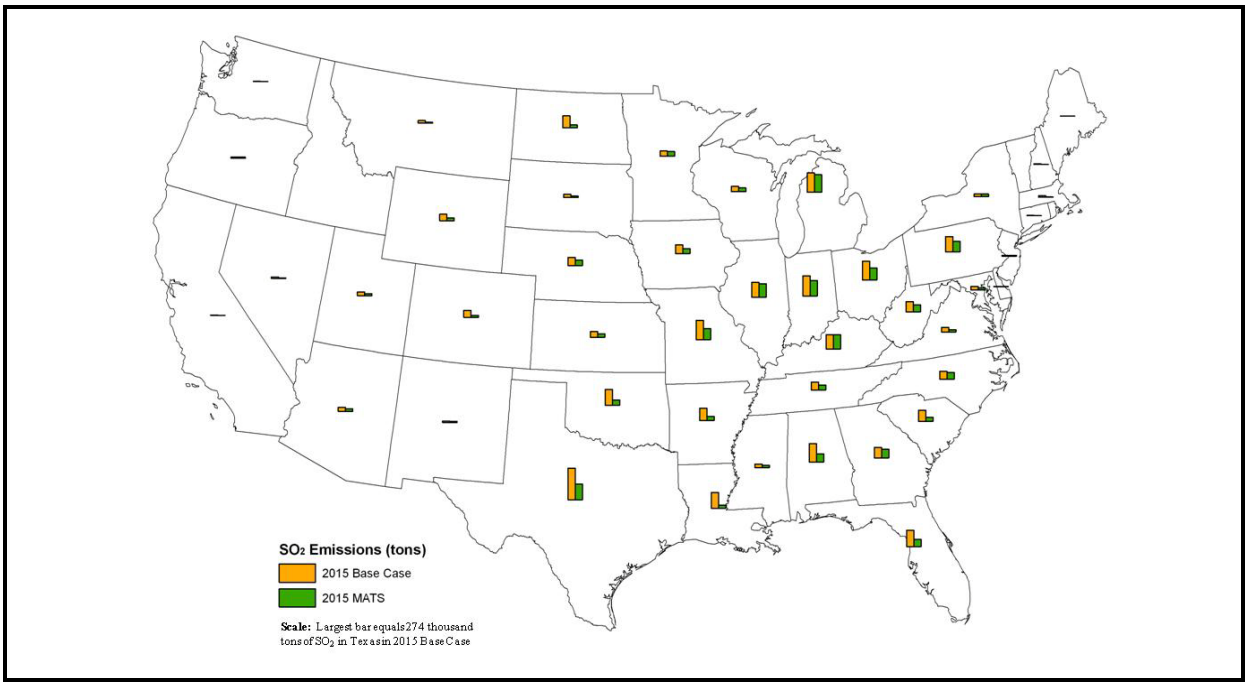

Figure 3-2. SO_2 Emissions from the Power Sector in 2015 with and without MATS

Source: 2015 emissions include coal steam (including IGCC and petroleum coke) units >25 MW from IPM v4.10 base case and control case projections (EPA, February 2011)

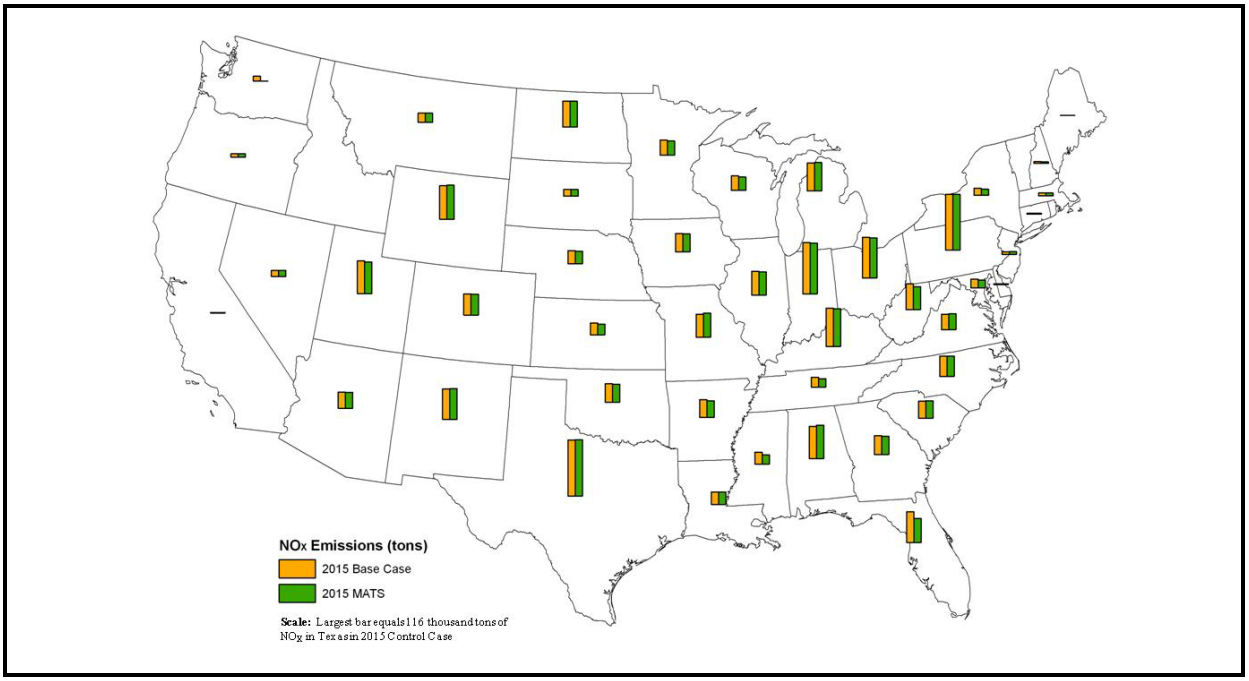

Figure 3-3. NO_X Emissions from the Power Sector in 2015 with and without MATS

Source: 2015 emissions include coal steam (including IGCC and petroleum coke) units >25 MW from IPM v4.10_MATS base case and control case projections (EPA, 2011)

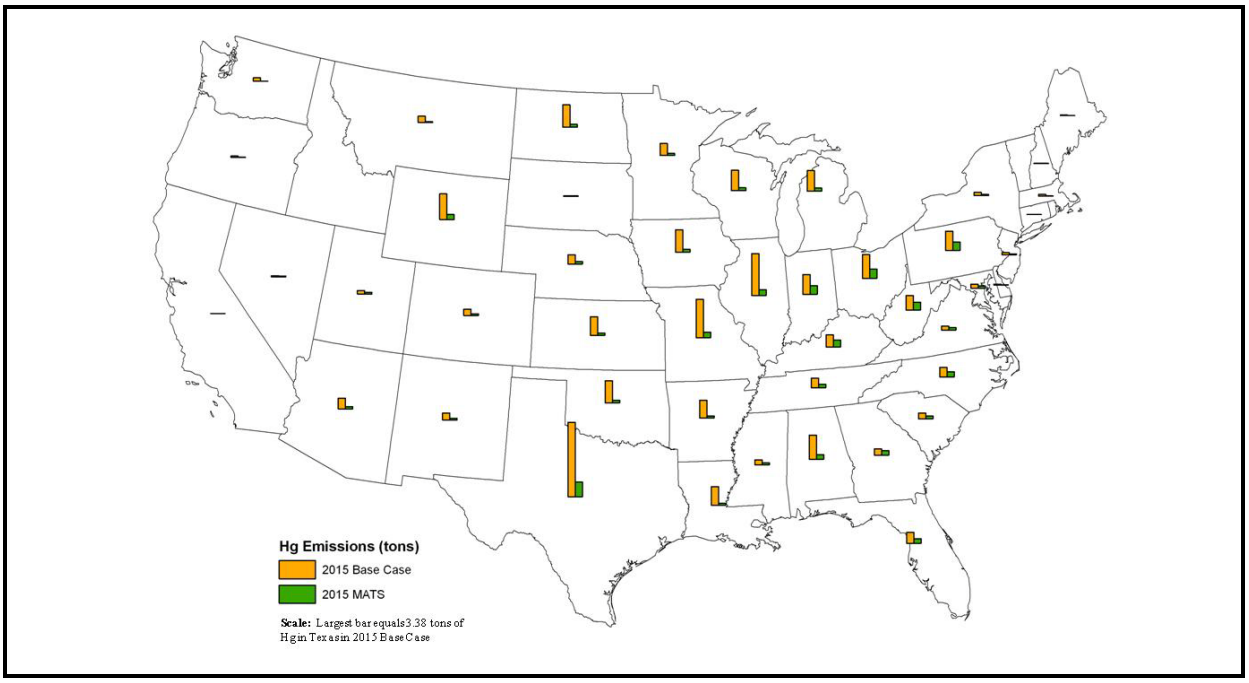

Figure 3-4. Mercury Emissions from the Power Sector in 2015 with and without MATS

Source: 2015 emissions include coal steam (including IGCC and petroleum coke) units >25 MW from IPM v4.10_MATS base case and control case projections (EPA, 2011)

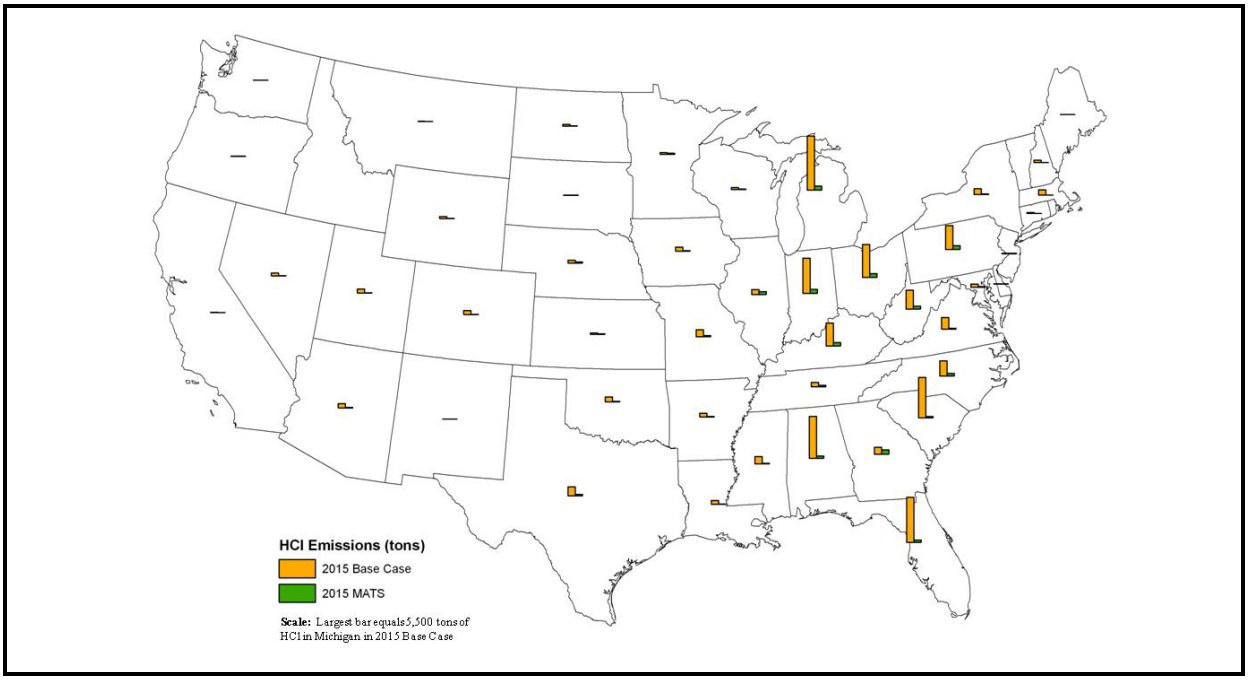

Figure 3-5. Hydrogen Chloride Emissions from the Power Sector in 2015 with and without MATS

Source: 2015 emissions include coal steam (including IGCC and petroleum coke) units >25 MW from IPM v4.10_MATS base case and control case projections (EPA, 2011)

3.3 Projected Compliance Costs

The power industry's "compliance costs" are represented in this analysis as the change in electric power generation costs between the base case and policy case in which the sector pursues pollution control approaches to meet the final HAP emission standards. In simple terms, these costs are the resource costs of what the power industry will directly expend to comply with EPA's requirements.

EPA projects that the annual incremental compliance cost of MATS is $9.4 billion in 2015 ($2007). The annual incremental cost is the projected additional cost of complying with the final rule in the year analyzed, and includes the amortized cost of capital investment (at 6.15%) and the ongoing costs of operating additional pollution controls, investments in new generating sources, shifts between or amongst various fuels, and other actions associated with compliance. This projected cost does not include the compliance calculated outside of IPM modeling, namely the compliance costs for oil-fired EGUs, and monitoring, reporting, and record-keeping costs. See section 3.14 for further details on these costs. EPA believes that the

cost assumptions used for the final rule reflect, as closely as possible, the best information available to the Agency today.

Table 3-5. Annualized Compliance Cost for MATS Requirements on Coal-fired Generation

	2015	2020	2030
Annualized Compliance Cost (billions of 2007$)	$9.4	$8.6	$7.4

Source: Integrated Planning Model run by EPA, 2011.

EPA's projection of $9.4 billion in additional costs in 2015 should be put into context for power sector operations. As shown in section 2.7, the power sector is expected in the base case to expend over $320 billion in 2015 to generate, transmit, and distribute electricity to end-use consumers. Therefore, the projected costs of compliance with MATS amount to less than a 3% increase in the cost to meet electricity demand, while securing public health benefits that are several times more valuable (as described in Chapters 4 and 5).

3.4 Projected Compliance Actions for Emissions Reductions

Fossil fuel-fired electric generating units are projected to achieve HAP emission reductions through a combination of compliance options. These actions include improved operation of existing controls, additional pollution control installations, coal switching (including blending of coals), and generation shifts towards more efficient units and lower-emitting generation technologies (e.g., some reduction of coal-fired generation with an increase of generation from natural gas). In addition, there will be some affected sources that find it uneconomic to invest in new pollution control equipment and will be removed from service. These facilities are generally amongst the oldest and least efficient power plants, and typically run infrequently. In order to ensure that any retirements resulting from MATS do not adversely impact the ability of affected sources and electric utilities from meeting the demand for electricity, EPA has conducted an analysis of the impacts of projected retirements on electric reliability. This analysis is discussed in TSD titled: "Resource Adequacy and Reliability in the IPM Projections for the MATS Rule" which is available in the docket.

The requirements under MATS are largely met through the installation of pollution controls (see Figure 3-6). To a lesser extent, there is a small degree of shifting within and across various ranks and types of coals, and a relatively small shift from coal-fired generation to greater use of natural gas and non-emitting sources of electricity (e.g., hydro and nuclear) (see Table 3-6). The largest share of emissions reductions occur from coal-fired units installing new pollution control devices, such as FGD, ACI, and fabric filters; a smaller share of emission

reductions come from fuel shifts and unit retirements. Mercury emission reductions are largely driven by SCR/FGD combinations and ACI installations. HCl emission reductions are largely driven by FGD and DSI installations, which also incidentally provide substantial SO_2 reductions in the policy case. Mercury, $PM_{2.5}$, and HCl emission reductions are also facilitated by the installation of fabric filters, which boost mercury and HCl removal efficiencies of ACI and DSI, respectively.

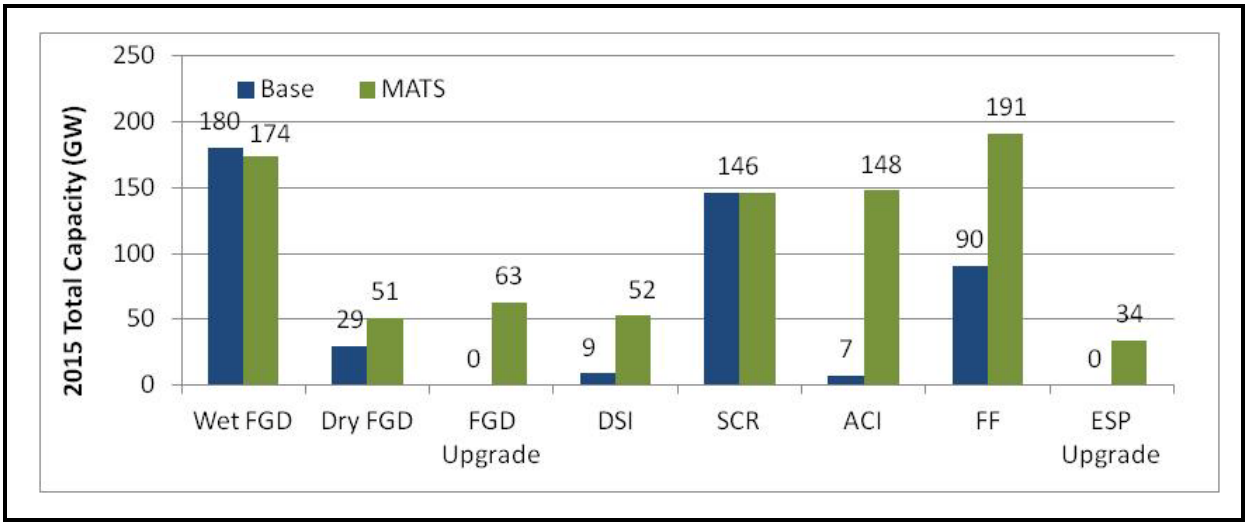

Figure 3-6. Operating Pollution Control Capacity on Coal-fired Capacity (by Technology) with the Base Case and with MATS, 2015 (GW)

Note: The difference between controlled capacity in the base case and under the MATS may not necessarily equal new retrofit construction, since controlled capacity above reflects incremental operation of dispatchable controls in 2015. Additionally, existing ACI installed on those units online before 2008 are not included in the base case to reflect removal of state mercury rules from IPM modeling. For these reasons, and due to rounding, numbers in the text below may not reflect the increments displayed in this figure. See IPM Documentation for more information on dispatchable controls.

Source: Integrated Planning Model run by EPA, 2011.

As shown in Figure 3-6, this analysis projects that by 2015, the final rule will drive the installation of an additional 20 GW of dry FGD (dry scrubbers), 44 GW of DSI, 99 GW of additional ACI, 102 GW of additional fabric filters, 63 GW of scrubber upgrades, and 34 GW of ESP upgrades. Furthermore, the final rule results in a 3 GW decrease in retrofit wet FGD capacity relative to the base, where the SO_2 allowance price under CSAPR provides an incentive for the additional SO_2 reductions achieved by a wet scrubber relative to a dry scrubber.

The difference between operating controlled capacity in the base case and under MATS in Figure 3-6 may not necessarily equal new retrofit construction, since total controlled capacity in the figure reflects incremental operation of existing controls that are projected to operate

under MATS but not under the base case. With respect to the increase in operating ACI, some of this increase represents existing ACI capacity on units built before 2008. EPA's modeling does not reflect the presence of state mercury rules, and EPA assumes that ACI controls on units built before 2008 do not operate in the absence of these rules. In the policy case, these controls are projected to operate and the projected compliance cost thus reflects the operating cost of these controls. Since these controls are in existence, EPA does not count their capacity toward new retrofit construction, nor does EPA's compliance costs projection reflect the capital cost of these controls (new retrofit capacity is reported in the previous paragraph).

3.5 Projected Generation Mix

Table 3-6 and Figure 3-7 show the generation mix in the base case and in MATS. In 2015, coal-fired generation is projected to decline slightly and natural-gas-fired generation is projected to increase slightly relative to the base case. Coal-fired generation is projected to increase above 2009 actual levels. 2015 natural gas-fired generation is projected to be lower than 2009, due in large part to the smaller relative difference in delivered natural gas and coal prices in different areas of the country projected in 2015 than occurred in 2009. The vast majority (over 98%) of base case coal capacity is projected to remain in service under MATS. In addition, the operating costs of complying coal-fired units are not so affected as to result in major changes in the electricity generation mix.

Table 3-6. Generation Mix with the Base Case and the MATS, 2015 (Thousand GWh)

	2009	2015			
				Change from	
	Historical	Base Case	Policy Case	Base	Percent Change
Coal	1,741	1,982	1,957	-25	-1.3%
Oil	36	0.11	0.11	0.00	3.6%
Natural Gas	841	710	731	22	3.1%
Nuclear	799	828	831	3	0.4%
Hydroelectric	267	286	288	2	0.8%
Non-hydro Renewables	116	252	250	-1	-0.6%
Other	10	45	45	0.0	0.0%
Total	3,810	**4,103**	**4,104**	**1**	**0.0%**

Note: Numbers may not add due to rounding.

Source: 2009 data from AEO Annual Energy Review, Table 8.2c Electricity Net Generation: Electric Power Sector by Plant Type, 1989-2010; 2015 projections are from the Integrated Planning Model run by EPA, 2011.

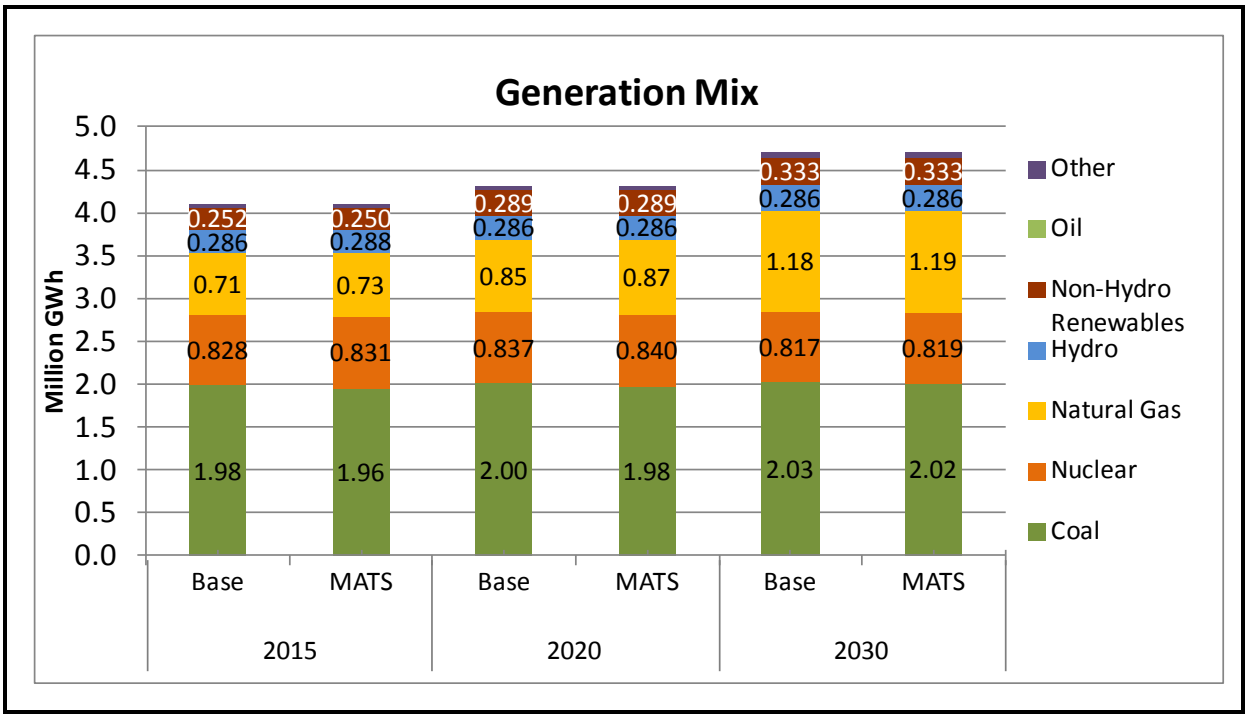

Figure 3-7. Generation Mix with the Base Case and with MATS, 2015-2030

Source: Integrated Planning Model run by EPA, 2011.

3.6 Projected Withdrawals from Service

Relative to the base case, about 4.7 GW (less than 2 percent) of coal-fired capacity is projected to be uneconomic to maintain by 2015. This projection considers various regional factors (e.g., other available capacity and fuel prices) and unit attributes (e.g., efficiency and age). These projected "uneconomic" units, for the most part, are older, smaller, and less frequently used generating units that are dispersed throughout the country (see Table 3-7).

Table 3-7. Characteristics of Covered Operational Coal Units and Additional Coal Units Projected to Withdraw as Uneconomic under MATS, 2015

	Average Age (Years)	Average Capacity	
		MW	Factor in Base
Withdrawn as Uneconomic	52	129	54%
Operational	43	322	71%

Source: Integrated Planning Model run by EPA, 2011.

These results should be considered "potential" closures. There are a variety of local factors that could make plant owners decide to keep one or more units projected to be uneconomic in service. These factors include different costs or demand estimates than what

was included in the IPM modeling, and local operating conditions or requirements that are on a smaller scale than that represented in EPA's IPM modeling. To the extent EPA's modeling does not account for plants that continue to operate due to one or more of these local factors, these results could be overestimating the capacity removed from service as a result of this rule.

For the final rule, EPA has examined whether the IPM-projected closures may adversely impact reserve margins and reliability planning. The IPM model is specifically designed to ensure that generation resource availability is maintained in the projected results subject to reserve margins in 32 modeling regions for the contiguous US, which must be preserved either by using existing resources or through the construction of new resources. IPM also addresses reliable delivery of generation resources by limiting the ability to transfer power between regions using the bulk power transmission system. Within each model region, IPM assumes that adequate transmission capacity is available to deliver any resources located in, or transferred to, the region. The IPM model projects available capacity given certain constraints such as reserve margins and transmission capability but does not constitute a detailed reliability analysis. For example, the IPM model does not examine frequency response. For more detail on IPM's electric load modeling and power system operation, please see IPM documentation (http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html) and the TSD on Resource Adequacy and Reliability in the IPM Projections for the MATS Rule.

Total operational capacity is lower in the policy scenario, primarily as a result of additional coal projected to be uneconomic to maintain. Since most regions are projected to have excess capacity above their target reserve margins, most of these withdrawals from service are absorbed by a reduction in excess reserves. Operational capacity changes from the base case in 2015 are shown in Table 3-8.

Table 3-8. Total Generation Capacity by 2015 (GW)

	2010	Base Case	MATS
Pulverized Coal	317	310	305
Natural Gas Combined Cycle	201	206	206
Other Oil/Gas	253	233	233
Non-Hydro Renewables	31	70	70
Hydro	99	99	99
Nuclear	102	104	105
Other	5	4	4
Total	1,009	1,026	1,021

Source: 2010 data from EPA's NEEDS v.4.10_PTox. Projections from Integrated Planning Model run by EPA.
Note: "Non-Hydro Renewables" include biomass, geothermal, solar, and wind electric generation capacity. 2015 capacity reflects plant closures planned to occur prior to 2015.

The policy case analyzed maintains resource adequacy in each region projected to decrease in coal capacity by using excess reserve capacity within the region, reversing base case withdrawals of non-coal capacity, building new capacity, or by importing excess reserve capacity from other regions. Although any closure of a large generation facility will need to be studied to determine potential local reliability concerns, EPA analysis suggests that projected economic withdrawals from service under the final rule could have little to no overall impact on electric reliability. Not only are projected withdrawals under MATS limited in scope, but the existing state of the power sector is also characterized by substantial excess capacity. The weighted average reserve margin at the national level is projected to be approximately 25% in the base case, while the North American Electric Reliability Corporation (NERC) recommends a margin of 15%. EPA projects that MATS would only reduce total operational capacity by less than one percent in 2015.

Moreover, coal units projected to withdraw as uneconomic are distributed throughout the power grid with limited effect at the regional level, such that any potential impacts should not adversely affect reserve margins and should be manageable through the normal industry processes. For example, in the RFC NERC reliability Region, containing coal-fired generating area in Pennsylvania, West Virginia and the Midwest, there is a decrease of less than 2% in the reserve margin in the policy case and a remaining overall reserve margin of over 20%. Furthermore, subregions may share each other's excess reserves to ensure adequate reserve margins within a larger reliability region. EPA's IPM modeling accommodates such transfers of reserves within the assumed limits of reliability of the inter-regional bulk power system. For

these reasons, the projected closures of coal plants are not expected to raise broad reliability concerns.

3.7 Projected Capacity Additions

Due in part to a low growth rate anticipated for future electricity demand levels in the latest EIA forecast, EPA analysis indicates that there is sufficient excess capacity through 2015 to compensate for capacity that is retired from service under MATS. In the short-term, most new capacity is projected as a mix of wind and natural gas in response to low fuel prices and other energy policies (such as tax credits and state renewable portfolio standards). In addition, future electricity demand expectations have trended downwards in recent forecasts, reducing the need for new capacity in the 2015 timeframe (see Chapter 2 for more discussion on future electricity demand).

Table 3-9. Total Generation Capacity by 2030 (GW)

	2010	Base Case	MATS	Change
Pulverized Coal	317	308	304	-3.9
Natural Gas Combined Cycle	201	275	278	2.9
Other Oil/Gas	253	235	235	0.6
Non-Hydro Renewables	31	79	79	0.1
Hydro	99	99	99	0.0
Nuclear	102	103	103	0.3
Other	5	4	4	0.0
Total	1,009	1,103	1,102	-0.1

Note: "Non-Hydro Renewables" include biomass, geothermal, solar, and wind electric generation capacity.
Source: 2010 data from EPA's NEEDS v.4.10_PTox. Projections from Integrated Planning Model run by EPA.

3.8 Projected Coal Production for the Electric Power Sector

Coal production for electricity generation under MATS is expected to increase from 2009 levels and decline modestly relative to the base case without the rule. The reductions in emissions from the power sector will be met through the installation and operation of pollution controls for HAP removal. Many available pollution controls achieve emissions removal rates of up to 99 percent (e.g., HCl removal by new scrubbers), which allows industry to rely more heavily on local bituminous coal in the eastern and central parts of the country that has higher contents of HCl and sulfur, and it is less expensive to transport than western subbituminous coal. Overall demand for coal is projected to be reduced as a result of MATS, with a slight

reduction in bituminous coal, and more of a reduction in subbituminous coal (see Tables 3-10 and 3-11). The trend reflects the projected reduced demand for lower-sulfur coal under MATS, where nearly all units are operating with a post-combustion emissions control. In this case, because of the additional pollution controls, many of these units no longer find it economic to pay a transportation premium to purchase lower-sulfur subbituminous coals. Instead, EGUs are generally projected to shift consumption towards nearby bituminous coal, which can achieve low emissions when combined with post-combustion emissions controls. This explains the increase from the base case in coal supplied from the Interior region, which is located in relatively close proximity to many coal-fired generators subject to MATS. This continues a trend of increased Interior supply (due to abundant Illinois Basin reserves that are relatively inexpensive to mine) and decreased Central Appalachian supply which is forecasted to occur in the base case from historic levels. The decline in Appalachia is a result of an increase in the relative cost of Central Appalachian extraction due both to rising mining cost (e.g., in 2010 major producers reported mining cost increases up to 15% with this trend continuing into 2011) and shrinking economically recoverable capacity. Growing international demand for Appalachian thermal coal is also contributing to its rising price. The increase in lignite use occurs at units blending subbituminous and lignite coals, and reflects a small shift in blended balance towards a greater use of lignite.

Table 3-10. 2015 Coal Production for the Electric Power Sector with the Base Case and MATS (Million Tons)

Supply Area	2009	2015 Base	2015 MATS	Change in 2015
Appalachia	246	184	172	-6%
Interior	129	216	236	9%
West	553	554	537	-3%
Waste Coal	14	14	13	-5%
Imports		30	30	0%
Total	942	998	989	-1%

Source: Production: U.S. Energy Information Administration (EIA), *Coal Distribution — Annual (Final)*, web site http://www.eia.doe.gov/cneaf/coal/page/coaldistrib/a_distributions.html (posted February 18, 2011); Waste Coal: U.S. EIA, *Monthly Energy Review, January 2011 Edition,* Table 6.1 Coal Overview, web site http://www.eia.doe.gov/emeu/mer/coal.html (posted January 31, 2011). All projections from Integrated Planning Model run by EPA, 2011.

Table 3-11. 2015 Power Sector Coal Use with the Base Case and the MATS, by Coal Rank (TBtu)

Coal Rank	Base	MATS	Change
Bituminous	11,314	11,248	-0.6%
Subbituminous	7,736	7,554	-2%
Lignite	849	895	5%
Total	19,900	19,698	-1%

Source: Integrated Planning Model run by EPA, 2011.

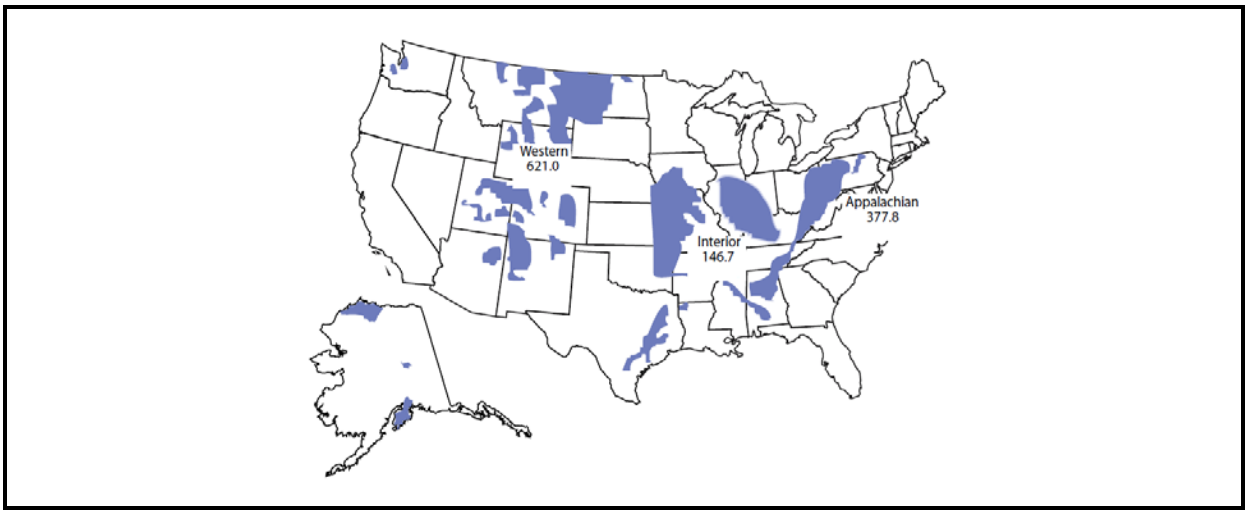

Figure 3-8. Total Coal Production by Coal-Producing Region, 2007 (Million Short Tons)

Note: Regional totals do not include refuse recovery

Source: EIA Annual Coal Report, 2007

3.9 Projected Retail Electricity Prices

EPA's analysis projects a near-term increase in the average retail electricity price of 3.1% in 2015 falling to 2% by 2020 under the final rule in the contiguous U.S. The projected price impacts vary by region and are provided in Table 3-12 (see Figure 3-9 for regional classifications).

Regional retail electricity prices are projected to range from 1 to 6 percent higher with MATS in 2015. The extent of regional retail electricity increases correlates with states that have considerable coal-fired generation in total generation capacity and that coal-fired generation is less well-controlled (such as in the ECAR and SPP regions). Retail electricity prices embody generation, transmission, and distribution costs. IPM modeling projects changes in regional

wholesale power prices, capacity payments, and actual costs of compliance in areas that are "cost of service" regions that are combined with EIA regional transmission and distribution costs to complete the retail price picture.

Table 3-12. Projected Contiguous U.S. and Regional Retail Electricity Prices with the Base Case and with the MATS (2007 cents/kWh)

	Base Case			MATS			Percent Change		
	2015	2020	2030	2015	2020	2030	2015	2020	2030
ECAR	8.2	8.2	9.8	8.5	8.5	9.9	4.5%	2.8%	1.0%
ERCOT	8.9	8.8	11.3	9.2	8.8	11.3	3.3%	0.6%	-0.2%
MAAC	9.5	10.4	12.7	9.8	10.4	12.7	2.8%	0.4%	-0.2%
MAIN	8.1	8.4	9.7	8.3	8.6	9.7	2.8%	2.2%	0.2%
MAPP	8.0	7.9	8.5	8.5	8.3	8.8	5.3%	5.6%	3.4%
NY	13.8	13.4	16.6	14.1	13.5	16.6	2.2%	0.7%	-0.1%
NE	12.3	11.8	13.8	12.6	11.9	13.8	2.0%	0.8%	0.0%
FRCC	10.2	9.7	11.0	10.4	9.8	11.0	2.2%	0.9%	0.4%
STV	7.9	7.8	8.4	8.2	8.0	8.6	3.1%	2.4%	1.6%
SPP	7.7	7.4	8.1	8.1	7.8	8.4	6.3%	6.1%	4.6%
PNW	7.1	6.8	7.6	7.3	7.0	7.6	2.7%	2.6%	1.1%
RM	9.2	9.5	11.0	9.4	9.7	11.1	2.3%	1.9%	1.1%
CALI	13.0	12.5	12.7	13.2	12.6	12.7	1.3%	0.7%	0.0%
Contiguous U.S. Average	9.0	9.0	10.2	9.3	9.2	10.3	3.1%	2.0%	0.9%

Source: EPA's Retail Electricity Price Model, 2011.

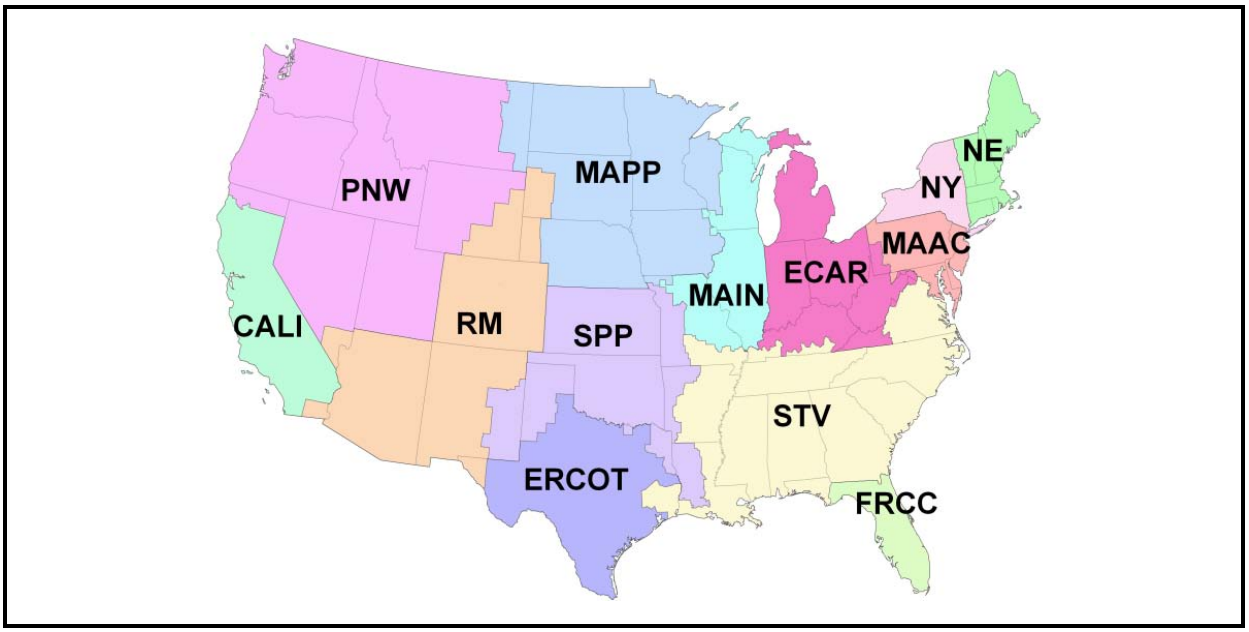

Figure 3-9. Retail Price Model Regions

3.10 Projected Fuel Price Impacts

The impacts of the final Rule on coal and natural gas prices before shipment are shown below in Tables 3-13 and 3-14. Overall, the national average coal price changes are related to changes in demand for a wide variety of coals based upon a number of parameters (e.g., chlorine or mercury content, heat content, proximity to the power plant, etc.), and this national average captures increases and decreases in coal demand and price at the regional level. Generally, total demand for coal decreases slightly under MATS, most notably subbituminous coal, which is by far the least expensive type of coal supplied to the power sector on an MMBtu basis. This is reflected in the projected average minemouth price of coal, which goes up by about 3 percent even though total demand for coal is reduced slightly (1 percent reduction). Notwithstanding the projected "mine-mouth" coal price changes, many units may in fact be realizing overall fuel cost savings by switching to more local coal supplies (which reduces transportation costs) after installing additional pollution control equipment. Gas price changes are directly related the projected increase in natural gas consumption under MATS. This increase in demand is met by producing additional natural gas at some increase in regional costs, resulting over time in a small price increase.

Table 3-13. Average Minemouth and Delivered Coal Prices with the Base Case and with MATS (2007$/MMBtu)

		2015			2030		
	2007	Base Case	MATS	Percent Change from Base	Base Case	MATS	Percent Change from Base
Minemouth	1.27	1.35	1.39	2.8%	1.51	1.56	3.3%
Delivered	1.76	2.11	2.15	1.9%	2.29	2.33	1.7%

Source: Historical data from EIA AEO 2010 Reference Case Table 15 (Coal Supply, Distribution, and Prices); projections from the Integrated Planning Model run by EPA, 2011.

Table 3-14. 2015-2030 Weighted Average Henry Hub (spot) and Delivered Natural Gas Prices with the Base Case and with MATS (2007$/MMBtu)

	Base Case	MATS	Percent Change from Base
Henry Hub	5.29	5.32	0.6%
Delivered - Electric Power	5.56	5.60	0.6%
Delivered - Residential	10.94	10.97	0.3%

Source: Projections from the Integrated Planning Model run by EPA (2011) adjusted to Henry Hub prices using historical data from EIA AEO 2011 reference case to derive residential prices.

IPM modeling of natural gas prices uses both short- and long-term price signals to balance supply of and demand in competitive markets for the fuel across the modeled time horizon. As such, it should be understood that the pattern of IPM natural gas price projections over time is not a forecast of natural gas prices incurred by *end-use consumers* at any particular point in time. The natural gas market in the United States has historically experienced significant price volatility from year to year, between seasons within a year, and even sees major price swings during short-lived weather events (such as cold snaps leading to short-run spikes in heating demand). These short-term price signals are fundamental for allowing the market to successfully align immediate supply and demand needs; however, end-use consumers are typically shielded from experiencing these rapid fluctuations in natural gas prices by retail rate regulation and by hedging through longer-term fuel supply contracts. IPM assumes these longer-term price arrangements take place "outside of the model" and on top of the "real-time" shorter-term price variation necessary to align supply and demand. Therefore, the model's natural gas price projections should not be mistaken for traditionally experienced consumer price impacts related to natural gas, but a reflection of expected average price changes over the time period 2015 to 2030.

For this analysis, in order to represent a natural gas price evolution that end-use consumers can anticipate under retail rate regulation and/or typical hedging behavior, EPA is displaying the weighted average of IPM's natural gas price projections for the 2015-2030 time horizon (see Table 3-14). In that framework, consumer natural gas price impacts are anticipated to range from 0.3% to 0.6% based on consumer class in response to MATS.

3.11 Key Differences in EPA Model Runs for MATS Modeling

In this analysis, we use the Integrated Planning Model (IPM), which is a multiregional, dynamic, deterministic linear programming model of the U.S. electric power sector.[9] The length of time required to conduct emissions and photochemical modeling precluded the use of IPM version 4.10_MATS. Thus the air quality modeling for MATS relied on EGU emission projections from an interim IPM platform that was subsequently updated during the rulemaking process for the base case and policy scenario summarized in this chapter. The 2015 base case EGU emissions projections of mercury, hydrogen chloride, SO_2, and PM used in air quality modeling were obtained from an earlier version of IPM, 4.10_FTransport. IPM version 4.10_FTransport reflects all state rules and consent decrees adopted through December 2010. Units with SO_2 or NO_X advanced controls (e.g., scrubber, SCR) that were not required to run for compliance with Title IV, New Source Review (NSR), state settlements, or state-specific rules were allowed in IPM to decide on the basis of economic efficiency whether to operate those controls. Note that this base case includes CSAPR, which was finalized in July 2011. Further details on the EGU emissions inventory used for this proposal can be found in the IPM Documentation.

The results presented in this chapter, from IPM version 4.10_MATS, reflect updates made to the 4.10_FTransport base case. These revisions are fully documented in the IPM 4.10 Supplemental Documentation for MATS and include: updated assumptions regarding the removal of HCl by alkaline fly ash in subbituminous and lignite coals; an update to the fuel-based mercury emission factor for petroleum coke, which was corrected based on re-examination of the 1999 ICR data; updated capital cost for new nuclear capacity and nuclear life extension costs; corrected variable operating and maintenance cost (VOM) for ACI retrofits; adjusted coal rank availability for some units, consistent with EIA From 923 (2008); updated state rules in Washington and Colorado; and numerous unit-level revisions based on comments received through the notice and comment process. Additionally, IPM v.4.10_MATS does not reflect mercury-specific state regulations (see section 1 above).

[9] http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html

3.12 Projected Primary PM Emissions from Power Plants

IPM does not endogenously model primary PM emissions from power plants. These emissions are calculated as a function of IPM outputs, emission factors and control configuration. IPM-projected fuel use (heat input) is multiplied by PM emission factors (based in part on the presence of PM-relevant pollution control devices) to determine PM emissions. Primary PM emissions are calculated by adding the filterable PM and condensable PM emissions.

Filterable PM emissions for each unit are based on historical information regarding existing emissions controls and types of fuel burned and ash content of the fuel burned, as well as the projected emission controls (e.g., scrubbers and fabric filters).

Condensable PM emissions are based on plant type, sulfur content of the fuel, and SO_2/HCl and PM control configurations. Although EPA's analysis is based on the best available emission factors, these emission factors do not account for the potential changes in condensable PM emissions due to the installation and operation of SCRs. The formation of additional condensable PM (in the form of SO_3 and H_2SO_4) in units with SCRs depends on a number of factors, including coal sulfur content, combustion conditions and characteristics of the catalyst used in the SCR, and is likely to vary widely from unit to unit. SCRs are generally designed and operated to minimize increases in condensable PM. This limitation means that IPM post-processing is potentially underestimating condensable PM emissions for units with SCRs. In contrast, it is possible that IPM post-processing overestimates condensable PM emissions in a case where the unit is combusting a low-sulfur coal in the presence of a scrubber.

EPA plans to continue improving and updating the PM emission factors and calculation methodologies. For a more complete description of the methodologies used to post-process PM emissions from IPM, see "IPM ORL File Generation Methodology" (March, 2011).

3.13 Illustrative Dry Sorbent Injection Sensitivity

Several commenters believe that EPA's IPM modeling assumptions regarding the efficacy and cost of DSI are based on too little data and are too optimistic. Some commenters believe that in practice there will be a need for many more FGD scrubbers for MATS compliance than projected by EPA for effective acid gas control, and at a corresponding higher cost. EPA disagrees with these opinions for several reasons (see the response to comments document in the docket) and believes that EPA's modeling assumptions regarding DSI cost and performance are reasonable.

However, to examine the potential impacts of limited DSI availability, EPA analyzed a scenario that limited total DSI capacity to 35 GW in 2015. In this scenario, which reduces the capacity of DSI by 18 GW compared to the primary MATS scenario, an additional 14 GW of coal capacity chooses to install scrubbers, and an additional 1.3 GW of capacity is projected to withdraw from service.

Limiting total DSI capacity to 35 GW results in a $1.2 billion (2007$) increase in annualized compliance costs in 2015. Additionally, SO_2 is further reduced in 2015 by an additional 62,000 tons (a 4.7% increase in SO_2 reductions and 4.5% increase in health benefits).

3.14 Additional Compliance Costs Analyzed for Covered Units

3.14.1 Compliance Cost for Oil-Fired Units.

As discussed in section 3.1, EPA used IPM to assess impacts of the MATS emission limitations for coal-fired EGUs but did not use IPM to assess the impacts for oil-fired units. IPM, with its power system and fuel cost assumptions, predicts many dual fuel units switch to natural gas and oil-fired units will not operate because IPM focuses on least cost operation of the power system. However, despite their apparent economic disadvantages, many of these units have run during many of the past five years (2006-2010). Therefore, EPA conducted a separate analysis to assess the impacts of the MATS emission limitations for oil-fired units.[10] EPA limited this analysis to oil-fired units in the contiguous U.S. Although there are several oil-fired units in states and territories outside the contiguous U.S., the final MATS emission limitations (shown in Table 3-2) for non-continental units will likely allow these units to continue firing residual fuel oil without additional air pollution controls.

For the base case, EPA categorized units by modeled fuels as listed in NEEDS 4.10 (EPA, December 2010) and assigned each unit the least-cost fuel among its available fuels. For units with natural gas curtailment provisions that might require the firing of residual fuel oil, EPA assigned a mixed fuel ratio based on each unit's 2008-2010 weighted average natural gas-to-fuel oil ratio. For the policy case, EPA assessed three compliance options: (1) switching to natural gas where available, (2) switching to distillate fuel oil, and (3) installing an electrostatic precipitator (ESP) capable of 90% particulate removal efficiency. These compliance options address particulate emissions only. However, there might be additional emission reductions that result from changes to oil-fired units' generation due to changes in relative generating costs.

[10] Additional details and methodology for the analysis are presented in appendix 3A.

Between the base case and policy case, 12 units convert from residual fuel oil to distillate fuel oil at a cost of approximately $12 million annually (2007$) to meet the MATS emission limitations for oil-fired units. An additional 11 units, eight of which are subject to natural gas curtailment, that do not have existing ESP particulate pollution controls install an ESP at a cost of approximately $44 million annually (2007$) to achieve the MATS emission limitations for oil-fired units (see Table 3-15). EPA believes the emission impacts from these potential actions will be relatively small when compared to the full impacts of the MATS emission limitations because particulate emissions from oil-fired units are a small fraction of the total particulate emissions from EGUs.

Table 3-15. Cost Impacts of Compliance Actions for Oil-Fired Units

Compliance option	Number of units affected	Capacity of units affected	Annual cost (2007$)
Switch to distillate fuel oil	12	2,675 MW	$12 million
Install ESP for residual fuel oil	11	4,015 MW	$44 million
Total	**23**	**6,690 MW**	**$56 million**

3.14.2 Monitoring, Reporting and Record-keeping Costs

The annual monitoring, reporting, and record-keeping burden for this collection (averaged over the first 3 years after the effective date of the standards) is estimated to be $158 million. This includes 698,907 labor hours per year at a total labor cost of $49 million per year, and total non-labor capital costs of $108 million per year. This estimate includes initial and annual performance tests, semiannual excess emission reports, developing a monitoring plan, notifications, and record-keeping. Initial capital expenses to purchase monitoring equipment for affected units are estimated at a cost of $231 million. This includes 504,629 labor hours at a total labor cost of $35 million for planning, selection, purchase, installation, configuration, and certification of the new systems and total non-labor capital costs of $196 million. All burden estimates are in 2007 dollars and represent the most cost effective monitoring approach for affected facilities. See Section 7.3, Paperwork Reduction Act.

3.14.3 Total Costs Projected for Covered Units under MATS

EPA used IPM to analyze the compliance cost, and economic and energy impacts of the MATS rule. IPM estimated the costs for coal-fired electric utility steam generating units that burn coal, coal refuse, or solid-oil derived fuel. EPA did not use IPM, however, estimate compliance costs for most oil/gas steam boilers because IPM projection shows least-cost dispatch in an

environment where oil/gas-fired units are primarily selecting natural gas on an economic basis. In the separate analysis summarized above, EPA estimates compliance costs for oil-fired EGUs in a scenario in which these units continue to burn oil as historically observed and thus take compliance measures to remain on oil. This is a reasonable estimate of compliance costs for these units, but does not represent a re-balancing of electricity dispatch where these units combust oil rather than natural gas. Therefore, the summation of IPM-projected compliance costs for least-cost dispatch with the oil-fired compliance costs and the monitoring, reporting, and record-keeping costs is a reasonable approximation of total compliance costs, but does not represent projected compliance costs under an economically efficient dispatch (see Table 3-16).

Table 3-16. Total Costs Projected for Covered Units under MATS, 2015 (billions of 2007$)

	2015
IPM Projection	$9.4
Monitoring/Reporting/Record-keeping	$0.158
Oil-Fired Fleet	$0.056
Total	$9.6

3.15 Limitations of Analysis

EPA's modeling is based on expert judgment of various input assumptions for variables whose outcomes are in fact uncertain. Assumptions for future fuel supplies and electricity demand growth deserve particular attention because of the importance of these two key model inputs to the power sector. As a general matter, the Agency reviews the best available information from engineering studies of air pollution controls to support a reasonable modeling framework for analyzing the cost, emission changes, and other impacts of regulatory actions.

The IPM-projected annualized cost estimates of private compliance costs provided in this analysis are meant to show the increase in production (generating) costs to the power sector in response to the final rule. To estimate these annualized costs, EPA uses a conventional and widely-accepted approach that applies a capital recovery factor (CRF) multiplier to capital investments and adds that to the annual incremental operating expenses. The CRF is derived from estimates of the cost of capital (private discount rate), the amount of insurance coverage required, local property taxes, and the life of capital. The private compliance costs presented earlier are EPA's best estimate of the direct private compliance costs of MATS.

The annualized cost of the final rule, as quantified here, is EPA's best assessment of the cost of implementing the rule. These costs are generated from rigorous economic modeling of

changes in the power sector due to implementation of MATS. This type of analysis using IPM has undergone peer review, and federal courts have upheld regulations covering the power sector that have relied on IPM's cost analysis.

Cost estimates for MATS are based on results from ICF's Integrated Planning Model. The model minimizes the costs of producing electricity (including abatement costs) while meeting load demand and other constraints (full documentation for IPM can be found at http://www.epa.gov/airmarkets/progsregs/epa-ipm and in the IPM 4.10 Supplemental Documentation for MATS. IPM assumes "perfect foresight" of market conditions over the time horizon modeled; to the extent that utilities and/or energy regulators misjudge future conditions affecting the economics of pollution control, costs may be understated as well.

In the policy case modeling, EPA exogenously determines that a subset of covered units might require a retrofit fabric filter (also known as a baghouse) retrofit, or might need to upgrade existing ESP control in order to meet the PM standard. EPA's methodology for assigning these controls to EGUs in policy case modeling is based on historic PM emission rates and reported control efficiencies, and is explained in the IPM 4.10 Supplemental Documentation for MATS.

Additionally, this modeling analysis does not take into account the potential for advancements in the capabilities of pollution control technologies as well as reductions in their costs over time. In addition, EPA modeling cannot anticipate in advance the full spectrum of compliance strategies that the power sector may innovate to achieve the required emission reductions under MATS, which would potentially reduce overall compliance costs. Where possible, EPA designs regulations to assure environmental performance while preserving flexibility for affected sources to design their own solutions for compliance. Industry will employ an array of responses, some of which regulators may not fully anticipate and will generally lead to lower costs associated with the rule than modeled in this analysis. For example, unit operators may find opportunities to improve or upgrade existing pollution control equipment without requiring as many new retrofit devices (i.e., meeting the PM standard with an existing ESP without requiring installation of a new fabric filter).

With that in mind, MATS establishes emission rates on key HAPs, and although this analysis projects a specific set of technologies and behaviors as EPA's judgment of least-cost compliance, the power sector is free to adopt alternative technologies and behaviors to achieve the same environmental outcome EPA has deemed in the public interest as laid out in the Clean Air Act. Such regulation serves to promote innovation and the development of new and

cheaper technologies. As an example, cost estimates of the Acid Rain SO_2 trading program by Resources for the Future (RFF) and MIT's Center for Energy and Environmental Policy Research (CEEPR) have been as much as 83 percent lower than originally projected by the EPA (see Carlson et al., 2000; Ellerman, 2003). It is important to note that the original analysis for the Acid Rain Program done by EPA also relied on an optimization model like IPM. Ex ante, EPA cost estimates of roughly $2.7 to $6.2 billion[11] in 1989 were an overestimate of the costs of the program in part because of the limitation of economic modeling to perfectly anticipate technological improvement of pollution controls and economic improvement of other compliance options such as fuel switching. Ex post estimates of the annual cost of the Acid Rain SO_2 trading program range from $1.0 to $1.4 billion.

In recognition of this historic pattern of overestimated regulatory cost, EPA's mobile source program uses adjusted engineering cost estimates of pollution control equipment and installation costs.[12] To date, and including this analysis, EPA has not incorporated a similar approach into IPM modeling of EGU compliance with environmental constraints. As a result, this analysis may overstate costs where such cost savings from as-yet untapped improvements to pollution control technologies may occur in the future. Considering the broad and complex suite of generating technologies, fuels, and pollution control strategies available to the power sector, as well as the fundamental role of operating cost in electricity dispatch, it is not possible to apply a single technology-improving "discount" transformation to the cost projections in this analysis. The Agency will consider additional methodologies in the future which may inform the amount by which projected compliance costs could be overstated regarding further technological development in analyses of power sector regulations.

As configured in this application, IPM does not take into account demand response (i.e., consumer reaction to electricity prices). The increased retail electricity prices shown in Table 3-13 would prompt end users to increase investment in energy efficiency and/or curtail (to some extent) their use of electricity and encourage them to use substitutes.[13] Those responses would lessen the demand for electricity, resulting in electricity price increases slightly lower than IPM predicts, which would also reduce generation and emissions. Demand response would yield certain unquantified cost savings from requiring less electricity to meet the quantity demanded. To some degree, these saved resource costs will offset the additional costs

[11] 2010 Phase II cost estimate in $1995.

[12] See regulatory impact analysis for the Tier 2 Regulations for passenger vehicles (1999) and Heavy-Duty Diesel Vehicle Rules (2000).

[13] The degree of substitution/curtailment depends on the costs and performance of the goods that substitute for more energy consuming goods, which is reflected in the demand elasticity.

of pollution controls and fuel switching that EPA anticipates from the final rule, although there could be some increase in social cost resulting from any decrease in electricity consumption. Although the reduction in electricity use is likely to be small, the cost savings from such a large industry[14] are not insignificant. EIA analysis examining multi-pollutant legislation in 2003 indicated that the annualized costs of MATS may be overstated substantially by not considering demand response, depending on the magnitude and coverage of the price increases.[15]

EPA's IPM modeling of MATS reflects the Agency's authority to allow facility-level compliance with the HAP emission standards rather than require each affected unit at a given facility to meet the standards separately. This flexibility would offer important cost savings to facility owners in situations where a subset of affected units at a given facility could be controlled more cost-effectively such that their "overperformance" would compensate for any "underperformance" of the rest of the affected units. EPA's modeling in this analysis required the average emission rate across all affected units at a given facility to meet the standard. This averaging flexibility has the potential to offer further cost savings beyond this analysis if particular units find ways to achieve superior pollution control beyond EPA's assumptions of retrofit technology performance at the modeled costs (which could then reduce the need to control other units at the same facility).

Additionally, EPA has chosen to express most of the control requirements here as engineering performance standards (e.g., lbs/MMBtu of heat input), which provide power plant operators goals to meet as they see fit in choosing coals with various pollutant concentrations and pollutant control technologies that they adopt to meet the requirements. Historically, such an approach encourages industry to engineer cheaper solutions over time to achieve the pollution controls requirements.

EPA's IPM modeling is based on retrofit technology cost assumptions which reflect the best available information on current and foreseeable market conditions for pollution control deployment. In the current economic environment, EPA does not anticipate (and thus this analysis does not reflect) significant near-term price increases in retrofit pollution control supply chains in response to MATS. To the extent that such conditions may develop during the

[14] Investor-owned utilities alone accounted for nearly $300 billion in revenue in 2008 (EIA).

[15] See "Analysis of S. 485, the Clear Skies Act of 2003, and S. 843, the Clean Air Planning Act of 2003." Energy Information Administration. September, 2003. EIA modeling indicated that the Clear Skies Act of 2003 (a nationwide cap and trade program for SO_2, NO_X, and mercury), demand response could lower present value costs by as much as 47% below what it would have been without an emission constraint similar to the Transport Rule.

sector's installation of pollution control technologies under the final rule, this analysis may understate the cost of compliance.

3.16 Significant Energy Impact

MATS would have a significant impact according to *E.O. 13211: Actions that Significantly Affect Energy Supply, Distribution, or Use*. Under the provisions of this rule, EPA projects that approximately 4.7 GW of coal-fired generation (less than 2 percent of all coal-fired capacity and 0.5% of total generation capacity in 2015) may be removed from operation by 2015. These units are predominantly smaller and less frequently-used generating units dispersed throughout the area affected by the rule. If current forecasts of either natural gas prices or electricity demand were revised in the future to be higher, that would create a greater incentive to keep these units operational.

EPA also projects fuel price increases resulting from MATS. Average retail electricity price are shown to increase in the contiguous U.S. by 3.1 percent in 2015. This is generally less of an increase than often occurs with fluctuating fuel prices and other market factors. Related to this, the average delivered coal price increases by less than 2 percent in 2015 as a result of shifts within and across coal types. As discussed above in section 8.10, EPA also projects that electric power sector-delivered natural gas prices will increase by about 0.6% percent over the 2015-2030 timeframe and that natural gas use for electricity generation will increase by less than 200 billion cubic feet (BCF) in 2015. These impacts are well within the range of price variability that is regularly experienced in natural gas markets. Finally, the EPA projects coal production for use by the power sector, a large component of total coal production, will decrease by 10 million tons in 2015 from base case levels, which is about 1 percent of total coal produced for the electric power sector in that year. The EPA does not believe that this rule will have any other impacts (e.g., on oil markets) that exceed the significance criteria.

3.17 References

EIA Annual Coal Report 2008. DOE/EIA-0584 (2008). Available at: http://www.eia.doe.gov/cneaf/coal/page/acr/acr_sum.html

EIA Annual Energy Outlook 2003. DOE/EIA-0383 (2003). Available at: http://www.eia.doe.gov/oiaf/archive/aeo03/index.html

EIA Electric Power Annual 2008. DOE/EIA-0348 (2008). Available at: http://www.eia.doe.gov/cneaf/electricity/epa/epa_sum.htm

EIA Electric Power Monthly March 2010 with Data for December 2009. DOE/EIA-0226 (2010/03). Available at: http://www.eia.doe.gov/cneaf/electricity/epm/epm_sum.html

Freme, Fred. 2009. U.S. Coal Supply and Demand: 2008 Review. EIA. Available at: http://www.eia.doe.gov/cneaf/coal/page/special/tbl1.html

Harrington, W., R.D. Morgenstern, and P. Nelson. 2000. "On the Accuracy of Regulatory Cost Estimates." *Journal of Policy Analysis and Management* 19(2):297-322.

Manson, Nelson, and Neumann. 2002. "Assessing the Impact of Progress and Learning Curves on Clean Air Act Compliance Costs." Industrial Economics Incorporated.

APPENDIX 3A
COMPLIANCE COSTS FOR OIL-FIRED ELECTRIC GENERATING UNITS

This appendix highlights the supplemental oil-fired electric generating unit (EGU) compliance cost analysis performed for the Mercury and Air Toxics Standards (MATS). EPA used the Integrated Planning Model (IPM) to assess the cost, economic, and energy impacts of the MATS emission limitations on coal-fired EGUs in the contiguous U.S., but did not use IPM to assess the compliance costs for oil-fired EGUs because IPM focuses on the least cost operation of the power system and, therefore, predicts the oil-fired units will not operate. These oil-fired units, however, do not operate on a purely economic basis. Some oil-fired units may operate as "must run", "black start", or "spinning reserve". In addition, some dual fuel fired units which IPM predicts will fire natural gas may be required to fire fuel oil when subject to mandatory curtailment of natural gas supplies.

When practicable, this supplemental analysis for oil-fired EGUs was based on the data and assumptions used in IPM. Documentation for IPM can be found at http://www.epa.gov/airmarkets/progsregs/epa-ipm.

3A.1 Methodology and Assumptions

3A.1.1 Base Case

EPA developed the base case for oil-fired units listed in the National Electric Energy Data System (hereafter, NEEDS) (EPA, 2010a). NEEDS lists 302 "oil/gas steam" units greater than 25 MW for which distillate fuel oil and/or residual fuel oil are among the modeled fuels (see Table 3A-1).[16] For each of these units, EPA projected 2015 heat input and apportioned the heat input among the NEEDS modeled fuels. EPA used each unit's average annual heat input from 2006-2010[17] as a proxy for 2015 heat input. For units not subject to mandatory natural gas curtailment, EPA assumed the unit fired the least cost fuel available based on regional IPM fuel cost projections for 2015. For units that may be required to fire fuel oil due to mandatory natural gas curtailment, EPA apportioned the heat input based on the unit's weighted average natural gas and fuel oil apportionment from 2008-2010.[18] EPA used the three most recent years because, as a percentage of total heat input, fuel oil heat input has fallen steadily since 2007 (see Figure 3A-1). With increased availability of natural gas in the New York region from new

[16] One unit, Charles Poletti unit 001 (ORIS 2491), was removed because the unit retired in 2010 (EPA, 2011).
[17] Designated representatives for each of the oil-fired units included in this analysis certify and report hourly heat input and emission data to EPA under 40CFR Part 75.
[18] The units subject to mandatory natural gas curtailment report fuel-apportioned heat input to EPA under 40CFR Part 75 (Appendix D). EPA categorized "diesel" as distillate fuel oil and "oil" and "other oil" as residual fuel oil.

gas supplies and new gas pipelines (FERC, 2011), it is likely this trend will continue even in the absence of the MATS. Therefore, using a longer historical period might significantly overestimate the proportion of heat input derived from fuel oil for these units.

Table 3A-1. Oil-fired EGUs by Fuel Type

NEEDS modeled fuel	Number of units	Capacity (MW)
Distillate fuel oil	10	814
Distillate fuel oil, natural gas	99	19,822
Residual fuel oil	17	5,867
Residual fuel oil, distillate fuel oil	15	1,187
Residual fuel oil, natural gas	149	39,913
Residual fuel oil, distillate fuel oil, natural gas	12	3,706

Source: EPA. 2010. National Electricity Energy Data System (NEEDS 4.10). Available at: http://www.epa.gov/airmarkets/progsregs/epa-ipm/toxics.html.

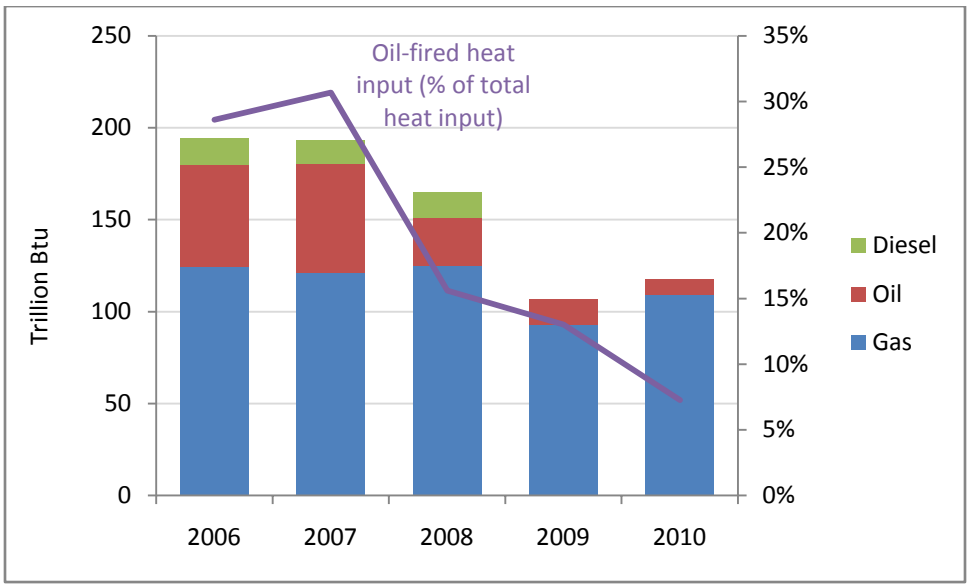

Figure 3A-1. 2006-2010 Heat Input Apportioned by Fuel for Oil-Fired Units Subject to Mandatory Natural Gas Curtailment

Source/Notes: EPA. 2011. Data and Maps. Available at: http://epa.gov/camddataandmaps/

Power companies are responding to fuel prices, natural gas supplies, and other market factors by replacing some oil-gas steam units with new combined cycle plants (Neville, J. 2011). EPA did not, however, factor in the effect of expanded availability of natural gas on the

utilization of these oil-fired units. As a result, this analysis likely overestimates the impact of the MATS emission limitations on oil-fired units.

In the base case, natural gas is the least cost fuel for the majority of units (see Table 3A-2). However, 41 units are expected to continue burning some amount of residual fuel oil because the units are subject to mandatory natural gas curtailment or may not have access to natural gas supplies.[19] Of these 41 units, 14 have existing electrostatic precipitator (ESP) particulate pollution controls.

Table 3A-2. Least Cost NEEDS Modeled Fuels for Oil-fired EGUs

NEEDS modeled fuel	Number of units	Capacity (MW)
Distillate fuel oil	19	1,228
Residual fuel oil	23	6,640
Natural gas	242	57,232
Natural gas with mandatory curtailment	18	6,208

3A.1.2 Policy Case

For the policy case, EPA considered three actions to comply with the MATS emission limitations: (1) switching to natural gas where available, (2) switching to distillate fuel oil, and (3) ESP particulate pollution control capable of 90% particulate removal efficiency. EPA modeled the cost of actions 2 and 3 for each unit in the base case. EPA did not model the cost of converting to natural gas because, for units with natural gas as a NEEDS modeled fuel, it was the least cost fuel and therefore the base case fuel for the unit. The cost of switching a unit's heat input to distillate fuel oil was based on the cost of converting operations, including tank, line, and pump cleaning and burner atomizer assembly replacement, and the unit's 2015 projected heat input from residual fuel oil multiplied by the cost difference between residual fuel oil and distillate fuel oil in the region where the unit is located. Conversion costs were annualized using the methodology described in the IPM documentation (EPA, 2010b).

The cost of installing a flat plate-type ESP on oil-fired model units of various sizes was calculated using the methodology outlined in EPA's Cost Manual (EPA, 2002) and adjusted to 2010 values using the Chemical Engineering Plant Cost Index (CEPCI). EPA developed non-linear

[19] To ensure the analysis was not likely to underestimate compliance costs, EPA assumed units that do not include natural gas as a NEEDS modeled fuel do not have access to a natural gas pipeline. The cost of obtaining pipeline access for these units was assumed to be uneconomical and was not modeled in the analysis.

regression power functions similar to those used for costing air pollution controls in IPM. The cost functions are shown in equations (3A.1)-(3A.3).

$$\text{Capital costs} = 243{,}494.4 \times (\text{MW capacity})^{0.7800} \quad (3A.1)$$

$$\text{Annual fixed costs} = 13{,}883.4 \times (\text{MW capacity})^{0.7294} \quad (3A.2)$$

$$\text{Annual variable costs} = 8{,}108.6 \times (\text{MWh generation})^{0.8632} \quad (3A.3)$$

Capital costs were annualized using the capital cost recovery factor used in the IPM documentation (EPA, 2010b). Annual variable costs were calculated using the predicted 2015 generation from residual fuel oil based on the unit's base case 2015 residual fuel oil heat input and the unit's heat rate listed in NEEDS (EPA, 2010a).

3A.1.3 Cost Sensitivities Related to Mandatory Natural Gas Curtailment

There are 18 dual fuel fired units (i.e., units capable of firing both gas and oil) that are subject to mandatory natural gas curtailment. Of these units, six have existing ESP particulate pollution controls installed. For the remaining 12 units, nine fired natural gas for more than 90 percent of their total heat input (see Table 3A-3). Because the MATS emission limits do not apply to units that fire coal or oil for less than 10 percent of total heat input averaged over three years or 15 percent in a single year, EPA analyzed historical oil-fired heat input between 2006 and 2010 at these units and found that four dual fuel fired units subject to mandatory natural gas curtailment did not exceed 15 percent in any single year and averaged less than 10 percent across all three year periods between 2006 and 2010. EPA did not include the cost of control on these units in the summary results. If these four units were to install ESPs, however, the annual compliance cost of the MATS emission limits would increase $13 million (2007$).

As noted in 3A.1.1, natural gas supplies to the region are increasing and operating data for dual fuel fired units subject to mandatory natural gas curtailment indicate that their proportion of heat input from residual oil is declining. There are four units in addition to those described in the paragraph above that exceeded 15 percent oil-fired heat input in 2006 and/or 2007, but between 2008 and 2010 did not exceed 15 percent oil-fired heat input in a single year and averaged below 10 percent across all three years. These units were assigned ESP particulate pollution controls in this analysis. However, if these four dual fuel fired units do not install ESPs, the annual compliance cost of the MATS emission limits would decline $16 million (2007$).

Table 3A-3. Percentage of Total Heat Input Derived from Oil for Oil-Fired Units Subject to Mandatory Natural Gas Curtailment (2008-2010)

Percentage	Number of units
< 1.0%	4
1.0% to 4.9%	1
5.0% to 9.9%	4
10.0% to 15.0%	3

3A.2 Results

For the purpose of estimating the impacts of the MATS emission limitations for oil-fired units, EPA had to make assumptions about the compliance actions oil-fired units will take. Table 3A-4 lists those assumptions based on differences between the base and policy cases. EPA assumed that the least cost compliance option for 12 residual fuel oil-fired units would be converting to distillate fuel oil at an annual cost of approximately $12 million (2007$). An additional 11 units would likely continue to burn residual fuel oil following the installation of an ESP at a cost of approximately $44 million annually (2007$).

Table 3A-4. Costs to Achieve the MATS Emission Limitations for Oil-Fired Units

Unit	Compliance action	Annual cost (2007$)
Cleary Flood, Unit 8	Distillate fuel oil	$ 308,000
Jefferies, Unit 1	Distillate fuel oil	$ 642,000
Jefferies, Unit 2	Distillate fuel oil	$ 673,000
McManus, Unit 1	Distillate fuel oil	$ 391,000
McManus, Unit 2	Distillate fuel oil	$ 512,000
Montville Station, Unit 6	Distillate fuel oil	$ 3,968,000
Possum Point, Unit 5	Distillate fuel oil	$ 119,000
Schuylkill Generating Station, Unit 1	Distillate fuel oil	$ 2,113,000
Vienna Operations, Unit 8	Distillate fuel oil	$ 1,741,000
William F Wyman, Unit 1	Distillate fuel oil	$ 783,000
William F Wyman, Unit 2	Distillate fuel oil	$ 646,000
Yorktown, Unit 3	Distillate fuel oil	$ 119,000
Astoria Generating Station, Unit 30	ESP	$ 4,214,000
Astoria Generating Station, Unit 40	ESP	$ 4,132,000
Astoria Generating Station, Unit 50	ESP	$ 4,202,000
B L England, Unit 3	ESP	$ 2,155,000
East River, Unit 60	ESP	$ 1,844,000
East River, Unit 70	ESP	$ 2,336,000
Herbert A Wagner, Unit 4	ESP	$ 4,352,000
Middletown, Unit 4	ESP	$ 4,391,000
Ravenswood, Unit 10	ESP	$ 3,904,000
Ravenswood, Unit 20	ESP	$ 3,898,000
Ravenswood, Unit 30	ESP	$ 8,322,000

3A.3 References

EPA. 2002. EPA Air Pollution Control Cost Manual. Sixth Edition. EPA/452/B-02-001. Available at: http://www.epa.gov/ttn/catc/dir1/c_allchs.pdf

EPA. 2010a. National Electric Energy Data System (NEEDS 4.10). Available at: http://www.epa.gov/airmarkets/progsregs/epa-ipm/toxics.html

EPA. 2010b. Documentation for EPA Base Case v. 4.10. Chapter 8: Financial Assumptions. Available at: http://www.epa.gov/airmarkets/progsregs/epa-ipm/docs/v410/Chapter8.pdf

EPA. 2011. Data and Maps. Available at: http://epa.gov/camddataandmaps/

FERC. 2011. Major Pipeline Projects Pending (Onshore). Available at: http://www.ferc.gov/industries/gas/indus-act/pipelines/pending-projects.asp

Neville, J. 2011. "Top Plant: Astoria II Combined Cycle Plant, Queens, New York," *Power Magazine*. September.

CHAPTER 4
MERCURY AND OTHER HAP BENEFITS ANALYSIS

4.1 Introduction

This chapter provides an analysis of the benefits of the proposed Toxics Rule from mercury and reductions of other HAP. Our efforts at quantifying the toxics benefits of this rule focus on quantifying and estimating the welfare benefits of reducing mercury emissions because mercury is the only HAP controlled by this rule for which there are sufficient available analytic tools to conduct a national-scale benefits assessment.

This analysis of the benefits of reduced mercury exposure from EGUs as a result of the rule is not changed from that provided for the proposed rule. It uses the same baseline and control cases for mercury deposition as was used to estimate mercury benefits in the Mercury and Air Toxics Rule proposal. EPA determined that it was reasonable to not update the mercury benefits assessment for the final rule because of the small magnitude of the quantified mercury benefits in the proposal, and the small difference (approximately 2 tons) in mercury emissions reductions between the proposed and final rules. It is not expected that mercury benefits would be substantially changed, and given the small magnitude of the benefits, any changes would not meaningfully affect the overall benefits of the rule, nor impact the benefit-cost comparison. An assessment of how forecast EGU mercury emissions changed between the baseline used at proposal and the baseline used for the costs and co-benefits analysis, and between the regulation as proposed and the regulation as finalized, is described in Appendix 5A.

This analysis builds on the methodologies developed previously for the 2005 Clean Air Mercury Rule (CAMR). This is a national scale assessment which focuses on the exposures to methylmercury in populations who consume self-caught freshwater fish (recreational fishers and their families). While there are other routes of exposure, including self-caught saltwater fish and commercially purchased fresh and saltwater fish, these exposures are not evaluated because (1) for self-caught saltwater fish, we are unable to estimate the reduction in fish tissue methylmercury that would be associated with reductions in mercury deposition from U.S. EGUs, and (2) for commercially purchased ocean fish, it is nearly impossible to determine the source of the methylmercury in those fish, and thus we could not attribute mercury levels to U.S. EGUs.

This benefits analysis focuses on reductions in lost IQ points in the population, because of the discrete nature of the effect, and because we are able to assign an economic value to IQ

points. There are other neurological effects associated with exposures to methylmercury, including impacts on motor skills and attention/behavior and therefore, risk estimates based on IQ will not cover these additional endpoints and therefore could lead to an underestimate of overall neurodevelopmental impacts. In addition, the NRC (2001) noted that "there remains some uncertainty about the possibility of other health effects at low levels of exposure. In particular, there are indications of immune and cardiovascular effects, as well as neurological effects emerging later in life, that have not been adequately studied." These limitations suggest that the benefits of mercury reductions are understated by our analysis; however, the magnitude of the additional benefits is highly uncertain.

In Section 4.2, we discuss the potential health effects of mercury. Section 4.3 provides a discussion of mercury in the environment, including potential impacts on wildlife. Section 4.4 describes the resulting change in mercury deposition from air quality modeling of the proposed Toxics rule. Section 4.5 presents information on key data and assumptions used in conducting the benefits analysis. Section 4.6 presents information on a dose-response function that relates mercury consumption in women of childbearing with changes in IQ seen in children that were exposed prenatally. IQ is used as a surrogate for the neurobehavioral endpoints that EPA relied upon for setting the methylmercury reference dose (RfD). Section 4.7 presents exposure modeling and benefit methodologies applied to a no-threshold model (i.e., a model that assumes no threshold in effects at low doses of mercury exposure). Section 4.8 presents the final benefits and risk estimates for recreational freshwater anglers and selected high-risk subpopulations. Section 4.9 presents a qualitative description of the benefits from reductions in HAPs other than mercury that will take place as a result of the Toxics Rule.

For this benefits assessment, EPA chose to focus on quantification of intelligence quotient (IQ) decrements associated with prenatal mercury exposure as the endpoint for quantification and valuation of mercury health benefits. Reasons for this focus on IQ included the availability of thoroughly-reviewed, high-quality epidemiological studies assessing IQ or related cognitive outcomes suitable for IQ estimation, and the availability of well-established methods and data for economic valuation of avoided IQ deficits, as applied in EPA's previous benefits analyses for childhood lead exposure.

The quantitative estimates of human health benefits and risk levels provided in Section 4.2 is a national-scale assessment of economic benefits associated with avoided IQ loss due to reduced methylmercury (MeHg) exposure among recreational freshwater anglers. Modeled risk levels, in terms of IQ loss, for six high-risk subpopulations as a means of estimating potential

disproportionate impacts on demographic groups with traditionally subsistence or near-subsistence rates of fish consumption are presented in Chapter 7 Section 7.11.

The first analysis (Section 4.2.1) estimates benefits from avoided IQ loss under various regulatory scenarios for all recreational freshwater anglers in the 48 contiguous U.S. states. The average effect on individual avoided IQ loss in 2016 is 0.00209 IQ points, with total nationwide benefits estimated between $0.5 and $6.1 million.[1] In contrast, the subpopulations analyses (Section 7.12.2) focus on specific demographic groups with relatively high levels of fish consumption. For example, an African-American child in the Southeast born in 2016 to a mother consuming fish at the 90th percentile of published subsistence-like levels is estimated to experience a loss of 7.711 IQ points as a result of in-utero MeHg exposure from all sources in the absence of a Toxics Rule.[2] The implementation of the Toxics Rule would reduce the expected IQ loss for this child by an estimated 0.176 IQ points.

4.2 Impact of Mercury on Human Health

4.2.1 Introduction

Mercury is a persistent, bioaccumulative toxic metal that is emitted from power plants in three forms: gaseous elemental Hg (Hg^0), oxidized Hg compounds (Hg^{+2}), and particle-bound Hg (Hg_P). Elemental Hg does not quickly deposit or chemically react in the atmosphere, resulting in residence times that are long enough to contribute to global scale deposition. Oxidized Hg and HgP deposit quickly from the atmosphere impacting local and regional areas in proximity to sources. Methylmercury (MeHg) is formed by microbial action in the top layers of sediment and soils, after Hg has precipitated from the air and deposited into waterbodies or land. Once formed, MeHg is taken up by aquatic organisms and bioaccumulates up the aquatic food web. Larger predatory fish may have MeHg concentrations many times, typically on the order of one million times, that of the concentrations in the freshwater body in which they live.

[1] Monetized benefits estimates are for an immediate change in MeHg levels in fish. If a lag in the response of MeHg levels in fish was accounted for, the monetized benefits could be significantly lower, depending on the length of the lag and the discount rate used. As noted in the discussion of the Mercury Maps modeling, the relationship between deposition and fish tissue MeHg is proportional in equilibrium, but the MMaps approach does not provide any information on the time lag of response. Depending on the watershed studied, the lag time between changes in mercury deposition and changes in the MeHg levels in fish has been shown to range from XX

[2] We do note that overall confidence in IQ loss estimates above approximately 7 points decreases because we begin to apply the underlying IQ loss function at exposure levels (ppm hair levels) above those reflected in epidemiological studies used to derive those functions. The 39.1 ppm was the highest measured ppm level in the Faroes Island study, while ~86 was the highest value in the New Zealand study (USEPA, 2005) (a 7 IQ points loss is approximately associated with a 40 ppm hair level given the concentration-response function we are using).

Although Hg is toxic to humans when it is inhaled or ingested, we focus in this rulemaking on exposure to MeHg through ingestion of fish, as it is the primary route for human exposures in the U.S., and potential health risks do not likely result from Hg inhalation exposures associated with Hg emissions from utilities.

In 2000, the National Research Council (NRC) of the NAS issued the NAS Study, which provides a thorough review of the effects of MeHg on human health. There are numerous studies that have been published more recently that report effects on neurologic and other endpoints.

4.2.2 Neurologic Effects

In its review of the literature, the NAS found neurodevelopmental effects to be the most sensitive and best documented endpoints and appropriate for establishing an RfD (NRC, 2000); in particular NAS supported the use of results from neurobehavioral or neuropsychological tests. The NAS report (NRC, 2000) noted that studies in animals reported sensory effects as well as effects on brain development and memory functions and support the conclusions based on epidemiology studies. The NAS noted that their recommended endpoints for an RfD are associated with the ability of children to learn and to succeed in school. They concluded the following: "The population at highest risk is the children of women who consumed large amounts of fish and seafood during pregnancy. The committee concludes that the risk to that population is likely to be sufficient to result in an increase in the number of children who have to struggle to keep up in school."

4.2.3 Cardiovascular Impacts

The NAS summarized data on cardiovascular effects available up to 2000. Based on these and other studies, the NRC (2000) concluded that "Although the data base is not as extensive for cardiovascular effects as it is for other end points (i.e. neurologic effects) the cardiovascular system appears to be a target for MeHg toxicity in humans and animals." The NRC also stated that "additional studies are needed to better characterize the effect of methylmercury exposure on blood pressure and cardiovascular function at various stages of life."

Additional cardiovascular studies have been published since 2000. EPA did not to develop a quantitative dose-response assessment for cardiovascular effects associated with MeHg exposures, as EPA finds there is no consensus among scientists on the dose-response

functions for these effects. In addition, there is inconsistency among available studies as to the association between MeHg exposure and various cardiovascular system effects. The pharmacokinetics of some of the exposure measures (such as toenail Hg levels) are not well understood. The studies have not yet received the review and scrutiny of neurotoxicity studies.

4.2.4 Genotoxic Effects

The Mercury Study noted that MeHg is not a potent mutagen but is capable of causing chromosomal damage in a number of experimental systems. The NAS concluded that evidence that human exposure to MeHg caused genetic damage is inconclusive; they note that some earlier studies showing chromosomal damage in lymphocytes may not have controlled sufficiently for potential confounders. One study of adults living in the Tapajós River region in Brazil (Amorim et al., 2000) reported a direct relationship between MeHg concentration in hair and DNA damage in lymphocytes; as well as effects on chromosomes. Long-term MeHg exposures in this population were believed to occur through consumption of fish, suggesting that genotoxic effects (largely chromosomal aberrations) may result from dietary, chronic MeHg exposures similar to and above those seen in the Faroes and Seychelles populations.

4.2.5 Immunotoxic Effects

Although exposure to some forms of Hg can result in a decrease in immune activity or an autoimmune response (ATSDR, 1999), evidence for immunotoxic effects of MeHg is limited (NRC, 2000).

4.2.6 Other Human Toxicity Data

Based on limited human and animal data, MeHg is classified as a “possible” human carcinogen by the International Agency for Research on Cancer (IARC, 1994) and in IRIS (USEPA, 2002). The existing evidence supporting the possibility of carcinogenic effects in humans from low-dose chronic exposures is tenuous. Multiple human epidemiological studies have found no significant association between Hg exposure and overall cancer incidence, although a few studies have shown an association between Hg exposure and specific types of cancer incidence (e.g., acute leukemia and liver cancer) (NAS, 2000).

4.3 Impact of Mercury on Ecosystems and Wildlife

4.3.1 Introduction

Deposition of mercury to waterbodies can also have an impact on ecosystems and wildlife. Mercury contamination is present in all environmental media with aquatic systems experiencing the greatest exposures due to bioaccumulation. Bioaccumulation refers to the net uptake of a contaminant from all possible pathways and includes the accumulation that may occur by direct exposure to contaminated media as well as uptake from food.

Atmospheric mercury enters freshwater ecosystems by direct deposition and through runoff from terrestrial watersheds. Once mercury deposits, it may be converted to organic methylmercury mediated primarily by sulfate-reducing bacteria. Methylation is enhanced in anaerobic and acidic environments, greatly increasing mercury toxicity and potential to bioaccumulate in aquatic foodwebs. A number of key biogeochemical controls influence the production of methylmercury in aquatic ecosystems. These include sulfur, pH, organic matter, iron, mercury "aging," and bacteria type and activity (Munthe et al.2007).

Wet and dry deposition of oxidized mercury is a dominant pathway for bringing mercury to terrestrial surfaces. In forest ecosystems, elemental mercury may also be absorbed by plants stomatally, incorporated by foliar tissues and released in litterfall (Ericksen et al., 2003). Mercury in throughfall, direct deposition in precipitation, and uptake of dissolved mercury by roots (Rea et al., 2002) are also important in mercury accumulation in terrestrial ecosystems.

Soils have significant capacity to store large quantities of atmospherically deposited mercury where it can leach into groundwater and surface waters. The risk of mercury exposure extends to insectivorous terrestrial species such as songbirds, bats, spiders, and amphibians that receive mercury deposition or from aquatic systems near the forest areas they inhabit (Bergeron et al., 2010a, b; Cristol et al., 2008; Rimmer et al., 2005; Wada et al., 2009 & 2010).

Numerous studies have generated field data on the levels of mercury in a variety of wild species. Many of the data from these environmental studies are anecdotal in nature rather than representative or statistically designed studies. The body of work examining the effects of these exposures is growing but still incomplete given the complexities of the natural world. A large portion of the adverse effect research conducted to date has been carried out in the laboratory setting rather than in the wild; thus, conclusions about overarching ecosystem health and

population effects are difficult to make at this time. In the sections that follow numerous effects have been identified at differing exposure levels.

4.3.2 Effects on Fish

A review of the literature on effects of mercury on fish (Crump and Trudeau, 2009) reports results for numerous species including trout, bass (large and smallmouth), northern pike, carp, walleye, salmon and others from laboratory and field studies. The effects studied are reproductive and include deficits in sperm and egg formation, histopathological changes in testes and ovaries, and disruption of reproductive hormone synthesis. These studies were conducted in areas from New York to Washington and while many were conducted by adding MeHg to water or diet many were conducted at current environmental levels.

The Integrated Science Assessment for Oxides of Nitrogen and Sulfur—Ecological Criteria (EPA, 2008) presents information regarding the possible complementary effects of sulfur and mercury deposition. The ISA has concluded that there is a causal relationship between sulfur deposition and increased mercury methylation in wetlands and aquatic environments. This suggests that lowering the rate of sulfur deposition would also reduce mercury methylation thus alleviating the effects of aquatic acidification as well as the effects of mercury on fish.

4.3.3 Effects on Birds

In addition to effects on fish, mercury also affects avian species. In previous reports (EPA, 1997 and EPA, 2005) much of the focus has been on large piscivorous species, in particular the common loon. The loon is most visible to the public during the summer breeding season on northern lakes and they have become an important symbol of wilderness in these areas (McIntyre and Barr, 1997). A multitude of loon watch, preservation, and protection groups have formed over the past few decades and have been instrumental in promoting conservation, education, monitoring, and research of breeding loons (McIntyre and Evers, 2000, Evers, 2006). Significant adverse effects on breeding loons from mercury have been found to occur, including behavioral (reduced nest-sitting), physiological (flight feather asymmetry), and reproductive (chicks fledged/territorial pair) effects (Evers, 2008, Burgess, 2008) and reduced survival (Mitro et al., 2008). Additionally Evers et al. (2008) report that they believe that results from their study integrating the effects on the endpoints listed above and evidence from other studies the weight of evidence indicates that population-level effects negatively impacting population viability occur in parts of Maine and New Hampshire, and potentially in broad areas of the loon's range.

Recently attention has turned to other piscivorous species such as the white ibis and great snowy egret. While considered to be fish-eating generally these wading birds have a diverse diet including crayfish, crabs, snails, insects and frogs. These species are experiencing a range of adverse effects due to exposure to mercury. The white ibis has been observed to have decreased foraging efficiency (Adams and Frederick, 2008). Additionally ibises have been shown to exhibit decreased reproductive success and altered pair behavior at chronic exposure to levels of dietary MeHg commonly encountered by wild birds (Frederick and Jayasena, 2010). These effects include significantly more unproductive nests, male/male pairing, reduced courtship behavior (head bobbing and pair bowing) and lower nestling production by exposed males. In this study a worst-case scenario suggested by the results could involve up to a 50% reduction in fledglings due to MeHg in diet. These estimates may be conservative if male/male pairing in the wild resulted in a shortage of partners for females and the effect of homosexual breeding were magnified. In egrets mercury has been implicated in the decline of the species in south Florida (Sepulveda et al., 1999) and Hoffman (2010) has shown that egrets experience liver and possibly kidney effects. While ibises and egrets are most abundant in coastal areas and these studies were conducted in south Florida and Nevada, the ranges of ibises and egrets extend to a large portion of the United States. Ibis territory can range inland to Oklahoma, Arkansas and Tennessee. Egret range covers virtually the entire United States except the mountain west.

Insectivorous birds have also been shown to suffer adverse effects due to current levels of mercury exposure. These songbirds such as Bicknell's thrush, tree swallows and the great tit have shown reduced reproduction, survival, and changes in singing behavior. Exposed tree swallows produced fewer fledglings (Brasso, 2008), lower survival (Hallinger, 2010) and had compromised immune competence (Hawley, 2009). The great tit has exhibited reduced singing behavior and smaller song repertoire in an area of high contamination in the vicinity of a metallurgic smelter in Flanders (Gorissen, 2005). While these effects were small and would likely have little effect on population viability in such a short-lived species.

4.3.4 Effects on Mammals

In mammals adverse effects of methylmercury exposure have been observed in mink and river otter, both fish eating species, collected in the wild in the northeast where atmospheric deposition from municipal waste incinerators and electric utilities are the largest sources (USEPA, 1999). For otter from Maine and Vermont maximum concentrations of Hg in fur nearly equal or exceed a concentration associated with mortality. Concentrations of Hg in liver for mink in Massachusetts/ Connecticut and the levels in fur from mink in Maine exceed

concentrations associated with acute mortality (Yates, 2005). Adverse sub-lethal effects may be associated with lower Hg concentrations and consequently be more widespread than potential acute effects. These effects may include increased activity, poorer maze performance, abnormal startle reflex, and impaired escape and avoidance behavior (Scheuhammer et al., 2007).

The studies cited here provide a glimpse of the scope of mercury effects on wildlife particularly reproductive and survival effects at current exposure levels. These effects range across species from fish to mammals and spatially across a wide area of the United States. The literature is far from complete however. Much more research is required to establish a link between the ecological effects on wildlife and the effect on ecosystem services (services that the environment provides to people) such as recreational fishing, bird watching and wildlife viewing. EPA is not, however, currently able to quantify or monetize the benefits of reducing mercury exposures affecting provision of ecosystem services adversely affected by mercury depostion.

4.4 Mercury Risk and Exposure Analyses—Data Inputs and Assumptions

4.4.1 Introduction

This section provides information regarding key data inputs and assumptions used in this assessment. The section begins with a description of the populations modeled in this assessment, follows with information about the data used to estimate MeHg concentrations in fish, and closes with a summary of the science and related assumptions used in this assessment to link changes in modeled mercury deposition to changes in fish tissue concentrations.

4.4.2 Data Inputs

4.4.2.1 Populations Assessed For the National Aggregate Estimates of Exposed Populations in Freshwater Fishing Households

The main source of data for identifying the size and location of the potentially exposed populations is the Census 2000 data, summarized at the tract-level. There are roughly 64,500 tracts in the continental United States, with populations generally ranging between 1,500 and 8,000 inhabitants. For the national aggregate analysis of exposure levels, the specific population of interest drawn from these data is the number of women aged 15 to 44 (i.e., childbearing age) in each tract. To predict populations in later years (2005 and 2016), we applied county-level population growth projections for the corresponding population category (Woods and Poole, 2008) to the 2000 tract-level data. To specifically estimate the portion of

these populations that are pregnant in any given year, we applied state-level 2006 fertility rate (live births per 1,000 women aged 15 to 44 years) data from U.S. Vital Statistics (DHHS, 2009).

Two main sources of national-level recreation activity data are available and suitable for estimating the size and spatial distribution of freshwater recreational angler populations and activities in the United States:

- the National Survey of Fishing, Hunting, and Wildlife-Associated Recreation (FHWAR), maintained by the Department of the Interior (DOI) (DOI and DOC, 1992, 1997, 2002, 2007) and
- the National Survey of Recreation and the Environment (USDA, 1994).

FHWAR Angler Data. The FHWAR, conducted by the U.S. Census Bureau about every 5 years since 1955, includes data on the number and characteristics of participants as well as time and money spent on hunting, fishing, and wildlife watching. The most recent survey and report are for recreational activities conducted in 2006 (DOI and DOC, 2007). Data from this report were used to provide the most recent estimate of the percentage of the resident population in each state (16 years old or older) that engaged in freshwater fishing during the year. As shown in Table 4-1, these percentages vary from 3% (New Jersey) to 27% (Minnesota).

The methodology for assessing mercury exposures also requires a further breakdown of freshwater fishing activities into two categories: rivers (including rivers and streams) and lakes (including lakes, ponds, reservoirs, and other flat water). Data at this level of detail are not reported in the summary national reports for the FHWAR; however, they are available from the FHWAR survey household-level data. For this analysis, data from a previous analysis and summary of the 2001 FHWAR household-level survey data (EPA, 2005) were used to provide estimates of the percentage of freshwater fishing days by residents in each state that were to either the lake or river category[3]. As shown in Table 4-1, the highest percentage going to lakes is in Minnesota (89%) and the highest to rivers is in Oregon (61%).

[3]Although the total *number* of fishing trips varies from year to year, there is little reason to expect that the *ratio* of river trips to lake trips would have changed significantly since 2001. For this reason, despite information on the type of waterbody visited being collected on the 2006 FWHAR survey, given resource and timetable limitations we did not update this input to the analysis.

Table 4-1. Summary of FWHAR State-Level Recreational Fishing Characteristics

State	Freshwater Anglers as Percentage of State Population[a]	Percentage of Freshwater Fishing Trips[b]	
		Lakes	Rivers
Alabama	15.7%	59.9%	40.1%
Arizona	7.0%	79.2%	20.8%
Arkansas	19.5%	81.1%	18.9%
California	4.1%	53.5%	46.5%
Colorado	13.2%	63.7%	36.3%
Connecticut	6.4%	58.7%	41.3%
Delaware	5.0%	52.8%	47.2%
Florida	7.9%	67.4%	32.6%
Georgia	12.6%	70.4%	29.6%
Idaho	18.4%	44.4%	55.6%
Illinois	7.3%	76.4%	23.6%
Indiana	12.3%	77.8%	22.2%
Iowa	16.8%	55.1%	44.9%
Kansas	14.8%	84.7%	15.3%
Kentucky	17.5%	80.0%	20.0%
Louisiana	14.2%	71.2%	28.8%
Maine	19.4%	73.7%	26.3%
Maryland	5.5%	40.7%	59.3%
Massachusetts	5.1%	75.5%	24.5%
Michigan	14.2%	85.6%	14.4%
Minnesota	26.9%	89.0%	11.0%
Mississippi	19.6%	79.0%	21.0%
Missouri	18.9%	80.2%	19.8%
Montana	22.8%	46.8%	53.2%
Nebraska	12.3%	80.6%	19.4%
Nevada	5.9%	80.5%	19.5%
New Hampshire	8.9%	67.9%	32.1%
New Jersey	3.1%	68.9%	31.1%

(continued)

Table 4-1. Summary of FWHAR State-Level Recreational Fishing Characteristics (continued)

State	Freshwater Anglers as Percentage of State Population[a]	Percentage of Freshwater Fishing Trips[b]	
		Lakes	Rivers
New Mexico	10.9%	56.1%	43.9%
New York	4.7%	67.2%	32.8%
North Carolina	10.7%	68.7%	31.3%
North Dakota	17.3%	87.2%	12.8%
Ohio	11.8%	78.8%	21.2%
Oklahoma	18.8%	83.1%	16.9%
Oregon	13.6%	39.0%	61.0%
Pennsylvania	8.1%	44.0%	56.0%
Rhode Island	4.4%	73.5%	26.5%
South Carolina	14.2%	75.6%	24.4%
South Dakota	14.6%	69.7%	30.3%
Tennessee	13.8%	68.6%	31.4%
Texas	9.7%	79.3%	20.7%
Utah	15.6%	68.0%	32.0%
Vermont	12.6%	71.1%	28.9%
Virginia	7.5%	70.4%	29.6%
Washington	9.5%	50.0%	50.0%
West Virginia	19.7%	50.1%	49.9%
Wisconsin	22.8%	79.5%	20.5%
Wyoming	23.5%	64.0%	36.0%

[a] Based on FHWAR 2006 data for residents 16 years and older.

[b] Based on FHWAR 2001 data for residents 16 years and older.

NSRE Angler Data. The NSRE, formerly known as the National Recreation Survey (NRS), is a nationally administered survey, which has been conducted periodically since 1962. It is designed to assess outdoor recreation participation in the United States and elicit information regarding people's opinions about their natural environment. The NSRE sample of freshwater anglers is smaller than the FHWAR sample, but it is nonetheless a useful resource because it provides a wide variety of information about fishing activities. Importantly, it includes relatively detailed information about the nature and location of recent freshwater trips. Because the

sampling procedure is designed to be representative, inferences may be drawn about the relative popularity of particular types of freshwater bodies (e.g., lakes, rivers) among the general public and the average distance traveled to reach these sites. Although more recent NSRE surveys have been conducted in 2000 and 2009, data from 1994 survey (NSRE, 1994) is used for this analysis because it contains the most detailed information regarding fishing trip destinations.

The NSRE 1994 elicited information from respondents about *the most recent* fishing trip. One of the main advantages of NSRE 1994 is that it includes geocoded data for reported fishing destinations. To specify the location of the last fishing trip, respondents were asked to provide the name of the waterbody, the nearest town to the waterbody, and an estimate of the distance and direction from their home to the waterbody. Appendix B describes how these data were used in this analysis to estimate the percentage of freshwater fishing trips that were in different distance intervals from respondents' homes. Using the demographic data from the NSRE, these estimates were further differentiated according to the income level and urban versus nonurban location of the respondents.

4.4.3 Mercury Concentrations in Freshwater Fish

4.4.3.1 Data Sources for Fish Tissue Concentrations

To characterize the spatial distribution of mercury concentration estimates in freshwater fish across the country, we compiled data from three main sources, which are described below.

National Listing of Fish Advisory (NLFA) database. The NLFA, managed by EPA (http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/), collects and compiles fish tissue sample data from all 50 states and from tribes across the United States. In particular, it contains data for over 43,000 mercury fish tissue samples collected from 1995 to 2007.

U.S. Geologic Survey (USGS) compilation of mercury datasets. As part of its Environmental Mercury Mapping and Analysis (EMMA) program, USGS compiled mercury fish tissue sample data from a wide variety of sources (including the NLFA) and has posted these data at http://emmma.usgs.gov/datasets.aspx. The compilation includes (1) state-agency collected and reported data (including Delaware, Iowa, Indiana, Louisiana, Minnesota, Ohio, South Carolina, Virginia, Wisconsin, and West Virginia) from over 40,000 fish tissue samples, covering the period 1995 to 2007 and (2) over 10,000 fish tissue samples from several other sources, including the National Fish Tissue Survey, the National Pesticide Monitoring Program

(NPMP), the National Contaminant Biomonitoring Program (NCBP), the Biomonitoring of Environmental Status and Trends (BEST) datasets of the USFWS and USGS (http://www.cerc.cr.usgs.gov/data/data.htm), and the Environmental Monitoring and Analysis Program (EMAP) (http://www.epa.gov/emap/).

EPA's National River and Stream Assessment (NRSA) study data. These data include nearly 600 fish tissue mercury samples collected at randomly selected freshwater sites across the United States during the period 2008 to 2009.

4.4.3.2 Approach for Compiling Fish Tissue Dataset for Exposure Analysis

Data from these three datasets were combined into a single master fish tissue dataset covering the period 1995 to 2009. One problem encountered in combining these datasets is the potential duplication of samples in the NLFA and USGS state-collected data. Unfortunately, these two datasets do not contain directly comparable and unique identifiers that allow duplicate samples to be easily identified and removed. Therefore, as an alternative, the samples from these two datasets were subdivided into data groups according to the year and state in which they were collected. If both datasets contained a data group for the same year and the same state, then the data group with the fewer number of observations was excluded from the master data.

The following criteria were also applied to exclude data from the master fish tissue dataset to be used in the analysis. Samples were excluded if they:

- did not include useable latitude-longitude coordinates for spatial identification;
- were located at sites outside the tidal boundaries of the continental United States (i.e., if they were not sampled from freshwater sites);
- did not come from fish species found in freshwater; or
- did not come from sampled fish that were at least 7 inches in length (i.e., unlikely to be consumed).

Each remaining sample was then categorized as either a river or lake sample based on information about the sampling site location. First, specific character strings in the site names (e.g., "river," "creek," "lake," "pond," and "reservoir") were used to classify sites. Second, remaining sites were categorized based on a GIS analysis that linked the sites' latitude-longitude coordinates to the nearest waterbody and its category.

- The resulting master fish tissue mercury concentration dataset contains 26,940 sample concentration estimates from 3,876 river sites and 23,206 estimates from 2,167 lake sites.

- A new dataset was then created by spatially grouping and averaging the river and lake concentration estimates at the HUC-12 sub-watershed level. First, all of the mercury sampling sites included in the master data were mapped and matched to the HUC-12 sub-watersheds in which they are located. A total of 3,884 HUC-12s in the continental United States (4.6%) contain at least one river or lake mercury sample.[4] Second, site-specific average mercury concentration values were generated by computing the mean concentration estimate at each site. Third, HUC-level average lake concentration estimates were computed as the mean of the site-specific average lake concentration estimates for each HUC containing at least one lake sampling site (1,396 HUCs). Fourth, HUC-level average river concentration estimates were computed as the mean of the site-specific average river concentration estimates for each HUC containing at least one river sampling site (2,655 HUCs).

4.4.3.3 Summary of Fish Tissue Mercury Concentration Estimates Used in the Exposure Analysis

The resulting HUC-level mercury concentration dataset is summarized in Table 4-2. The average HUC-level mercury concentration estimate for lakes is 0.29 ppm and for rivers is 0.26 ppm. The large standard deviations and ranges reported in the table also reflect the considerable spatial variation in lake and river concentration estimates across samples. As described below, the analysis uses this inter-watershed spatial variation (rather than just the average point estimate across watersheds) to estimate mercury exposures However, in this analysis, exposure estimates were only generated for populations linked to these HUCs containing at least one river or lake mercury fish tissue sample.

[4]This number excludes 15 HUC-12s containing mercury samples. These HUC-12s were excluded from the analysis due to their proximity to potentially significant non-air sources of mercury, including gold mines or non-EGU mercury sources included in the 2008 Toxic Release Inventory.

Table 4-2. Summary of HUC-level Average Mercury Fish Tissue Concentration Estimates

	N[a]	Mean	Std. Dev.	Min	Max
Lake Fish Tissue Concentrations					
HUC-level average mercury concentration (ppm)	1,396	0.286	0.231	0.000	3.56
Number of lake samples per HUC	1,396	16.62	31.61	1	458
Number of lake sampling sites per HUC	1,396	1.55	1.97	1	33
River Fish Tissue Concentrations					
HUC-level average mercury concentration (ppm)	2,655	0.261	0.259	0.006	4.97
Number of river samples per HUC	2,655	10.15	22.45	1	288
Number of river sampling sites per HUC	2,655	1.46	1.10	1	16

[a] Number of HUC-12s with at least one river or lake sampling site.

4.5 Linking Changes in Modeled Mercury Deposition to Changes in Fish Tissue Concentrations

4.5.1 Introduction

In the United States, humans are exposed to MeHg mainly by consuming fish that contain MeHg. Accordingly, to estimate changes in human exposure EPA must analyze how changes in Hg deposition from U.S. coal-fired power plants translate into changes in MeHg concentrations in fish. Quantifying the linkage between different levels of Hg deposition and fish tissue MeHg concentration is an important step in the risk assessment process and the focus of the material described in this section.

To effectively estimate fish MeHg concentrations in a given ecosystem, it is important to understand that the behavior of Hg in aquatic ecosystems is a complex function of the chemistry, biology, and physical dynamics of different ecosystems. The majority (95 to 97 percent) of the Hg that enters lakes, rivers, and estuaries from direct atmospheric deposition is in the inorganic form (Lin and Pehkonen, 1999). Microbes convert a small fraction of the pool of inorganic Hg in the water and sediments of these ecosystems into the organic form of Hg (MeHg). MeHg is the only form of Hg that biomagnifies in organisms (Bloom, 1992). Ecosystem-specific factors that affect both the bioavailability of inorganic Hg to methylating microbes (e.g., sulfide, dissolved organic carbon) and the activity of the microbes themselves (e.g., temperature, organic carbon, redox status) determine the rate of MeHg production and subsequent accumulation in fish (Benoit et al., 2003). The extent of MeHg bioaccumulation is also affected by the number of trophic levels in the food web (e.g., piscivorous fish populations)

because MeHg biomagnifies as large piscivorous fish eat smaller organisms (Watras and Bloom, 1992; Wren and MacCrimmon, 1986). These and other factors can result in considerable variability in fish MeHg levels among ecosystems at the regional and local scale.

4.5.2 Use of Mercury Maps to Project Changes in Fish Tissue Concentrations

To analyze the relationship between Hg deposition and MeHg concentrations in fish in freshwater aquatic ecosystems across the U.S. for the national scale benefits assessment, EPA applied EPA's Office of Water's Mercury Maps (MMaps) approach (US EPA, 2001a). MMaps implements a simplified form of the IEM-2M model applied in EPA's Mercury Study Report to Congress (USEPA, 1997). By simplifying the assumptions inherent in the freshwater ecosystem models that were described in the Report to Congress, the MMaps model showed that these models converge at a steady-state solution for MeHg concentrations in fish that are proportional to changes in Hg inputs from atmospheric deposition (i.e., over the long term, fish concentrations are expected to decline proportionally to declines in atmospheric loading to a waterbody).

MMaps has several limitations:

1. The MMaps approach is based on the assumption of a linear, steady-state relationship between concentrations of MeHg in fish and present day air deposition mercury inputs. We expect that this condition will likely not be met in many waterbodies because of recent changes in mercury inputs and other environmental variables that affect mercury bioaccumulation. For example, the US has recently reduced human-caused emissions while international emissions have increased.

2. The requirement that environmental conditions remain constant over the time required to reach steady state inherent in the MMaps methodology may not be met, particularly in systems that respond slowly to changes in mercury inputs.

3. Many water bodies, particularly in areas of historic gold and mercury mining, contain significant non-air sources of mercury. The MMaps methodology will yield biased results when applied to such waterbodies. As a simple illustrative example, if we have mercury deposition of 100 at a given location and a MeHg fish concentration of 6 in a local fish tissue sample, and a new emissions rule reduces deposition by half to 50, then, in the absence of other non-air deposition sources, we would assume that the MeHg fish concentration is reduced by the same proportion, to 3 ((50 / 100) × 6). However, if total pre-control mercury loading to the system is actually 100 *plus* another unaccounted for source (for example, an additional 100 due to area gold mining), then the MeHg fish concentration of 6 is actually due to 200 in total mercury loading. In this case, reducing mercury air deposition from 100 to 50 would only reduce the total loading by 25%, to 150, which, based on the MMaps

methodology, would result in a MeHg fish concentration of 4.5 ((150 / 200) × 6) rather than 3. In areas where non-air sources of mercury load are unaccounted for, MMaps-based estimates of changes in MeHg fish tissue concentrations due to reduced mercury air emissions would therefore be biased high.

4. Finally, MMaps does not account for a calculation of the time lag between a reduction in mercury deposition and a reduction in the MeHg concentrations in fish and, as noted earlier, depending on the nature of the watersheds and waterbodies involved, the temporal response time for fish tissue MeHg levels following a change in mercury deposition can range from years to decades depending on the attributes of the watershed and waterbody involved[5] Research has suggested that fish tissue MeHg levels in some locations may display a multi-phase response following a discrete change in mercury deposition, with the first phase lasting a few years to a decade or more and primarily involving changes in aerial loading directly to the waterbody and the second phase lasting decade (to a century or more) and reflecting longer-term changes in watershed erosion and runoff to the waterbody (Knights et al., 2009, Harris et al., 2007).

This methodology therefore applies only to situations where air deposition is the sole significant source of Hg to a water body, and where the physical, chemical, and biological characteristics of the ecosystem remain constant over time. EPA recognizes that concentrations of MeHg in fish across all ecosystems may not reach steady state and that ecosystem conditions affecting mercury dynamics are unlikely to remain constant over time. EPA further recognizes that many water bodies, particularly in areas of historic gold and Hg mining in western states, contain significant non-air sources of Hg. Finally, EPA recognizes that MMaps does not account for the time lag between a reduction in Hg deposition and a reduction in the MeHg concentrations in fish. While acknowledging these limitations, EPA is unaware of any other tool for performing a national-scale assessment of the change in fish MeHg concentrations resulting from reductions in atmospheric deposition of Hg. The following paragraphs provide additional details on the above limitations, as well as a brief assessment of the degree to which conditions match those assumptions.

The MMaps model represents a reduced form of the IEM-2M and MCM models used in the Mercury Study Report to Congress (USEPA, 1997), as well as the subsequent Dynamic MCM (D-MCM) model (Harris et al., 1996). That is, the equations of these mercury fate and transport

[5]As noted in footnote 1 of this chapter,monetized benefits estimates are for an immediate change in MeHg levels in fish (i.e., the potential lag period associated with fully realizing fish tissue MeHg levels was not reflected in benefits modeling). If a lag in the response of MeHg levels in fish were assumed, the monetized benefits could be significantly lower, depending on the length of the lag and the discount rate used. MMaps approach does not account for the time lag of response.

models are reduced to steady state and consolidated into a single equilibrium equation equating the ratio of future/current air deposition rates to future/current fish tissue concentrations.

Though plainly stated, the steady-state assumption is a compilation of a number of individual conditions. For example, fish tissue data may not represent average, steady-state concentrations for two major reasons:

- Fish tissue and deposition rate data for the base period are not at steady state. Where deposition rates have recently changed, the watershed or waterbody may not have had sufficient time to fully respond. The pool of mercury in different media could be sufficiently large relative to release rates, and thus needs more time to achieve a new equilibrium. This is more likely to occur in deeper lakes and lakes with large catchments where turnover rates are longer and where the watershed provides significant inputs of mercury.

- Fish tissue data do not represent average conditions (or conditions of interest for forecast fish levels). Methylation and bioaccumulation are variable and dynamic processes. If fish are sampled during a period of high or low methylation or bioaccumulation, they would not be representative of the average, steady-state or dynamic equilibrium conditions of the waterbody. This effect is significantly more pronounced in small and juvenile fish. Examples include tissue data collected during a drought or during conditions of fish starvation. Other examples include areas in which seasonal fluctuations in fish mercury levels are significant due, for example, from seasonal runoff of contaminated soils from abandoned gold and mercury mines or areas geologically rich in mercury. In such a case, MMaps predictions would be valid for similar conditions (e.g. wet year/dry year, or season) in the future, rather than typical or average conditions. Alternatively, sufficient fish tissue would need to be collected to get an average concentration that represents a baseline dynamic equilibrium.

Other ecosystem conditions might cause projections from the MMaps approach to be inaccurate for a particular ecosystem. Watershed and waterbody conditions can undergo significant changes in capacity to transport, methylate, and bioaccumulate mercury. Examples of this include regions where sulfate and/or acid deposition rates are changing (in turn affecting MeHg production independently of total mercury loading), and where the trophic status of a waterbody is changing. A number of other water quality parameters have been correlated with increased fish tissue concentrations (e.g. low pH, high DOC, lower algal concentrations), but these relationships are highly variable among different waterbodies. MMaps will be biased when waterbody characteristics change between when fish were initially sampled, and the new conditions of the waterbody.

As stated above, the relationship between the change in mercury deposition from air to the change in fish tissue concentration holds only when air deposition is the predominant source of the mercury load to a waterbody. Due to this requirement in the model, the national application of the MMaps approach screened out those watersheds that either contained active gold mines or had other substantial non-US EGU anthropogenic releases of mercury. Identification of watersheds with gold mines was based on a 2005 USGS data set characterizing mineral and metal operations in the United States. The data represent commodities monitored by the National Minerals Information Center of the USGS, and the operations included are those considered active in 2003 (online link: http://tin.er.usgs.gov/mineplant/). EPA considered the 25th percentile US-EGU emission level to be a reasonable screen for additional substantial non-US EGU releases to a given watershed. The identification of watersheds with substantial non-EGU anthropogenic loadings was based on a TRI-net query for 2008 of non-EGU mercury sources with total annual on-site Hg loading (all media) of 39.7 pounds or more. This threshold value corresponds to the 25th percentile annual US-EGU mercury emission value as characterized in the 2005 NATA. It should be noted that MMaps was designed to address an important, but very specific issue—that of eventual response of fish tissue to air deposition reductions. As such it responds to a need to understand how mercury reductions, independent of other changes in the environment, will impact fish contamination and human health. More complex models are required in cases where more complete descriptions are needed. A dynamic model is essential for modeling waterbody recovery during the period in which waterbody response lags reductions in mercury loads. A dynamic model is also essential for understanding seasonal fluctuations, as well as year-to-year fluctuations due to meteorological variability. Finally, a more complex model would be essential for assessing the impact of other watershed and water quality changes (e.g., erosion, wetlands coverage, and acid deposition) that might affect mercury bioaccumulation in fish. These complex models are used to derive the MMaps approach, and are themselves based on a number of assumptions. While these assumptions are considered reasonable given the state of the science of environmental modeling and mercury in the environment, the validity of assumptions inherent in both the MMaps approach and dynamic ecosystem scale models will need to be reevaluated as the science of mercury fate and transport evolves.

The MMaps methodology was peer reviewed by a set of national experts in the fate and transport of mercury in watersheds (US EPA, 2001a). While two reviewers felt it could be used to predict future fish tissue concentrations, a third cautioned it should not be considered a robust predictor until scientific data can be generated to validate the approach. Reviewers systematically identified a set of implicit assumptions that compose the steady state

assumption in the MMaps approach. They pointed out that due to evolving and complex nature of the science of mercury, some features of the complex models are assumptions themselves, and thus cannot be wholly relied upon as ultimate predictors of mercury fate and transport. The reviewers pointed out that there is limited scientific information to directly verify this approach, and that some scientific data appears to refute individual components of the overall steady state assumption. One reviewer did perform a D-MCM and MMaps comparison, and found that, under these assumptions, MMaps model did produce comparable steady-state results as the D-MCM model. There was considerable discussion about how best to aggregate the data, to scale up to a deposition reduction requirement, from fish-specific and waterbody specific information. The description of the approach and the methodologies as applied in this analysis are largely consistent with the peer review recommendations.

The MMaps report (US EPA, 2001a) presented a national-scale application of Mercury Maps to determine the percent reductions in air deposition that would be needed in watersheds across the country for average fish tissue concentrations to achieve the national MeHg criterion. In this national-scale assessment, fish tissue concentrations were aggregated at the scale of large watersheds, thus presenting average results for each watershed. The use of other scales of aggregation, e.g., waterbody specific, is consistent with the MMaps approach to the degree to which different mercury loads can be discerned.

4.5.3 The Science of Mercury Processes and Variability in Aquatic Ecosystems

The set of physical, chemical, and biological processes controlling mercury fate in watersheds and water bodies can be grouped into specific categories: mercury cycle chemistry; mercury processes in the atmosphere, soils and water; bioavailability of mercury in water; and mercury accumulation in the food web. The following is a review of these categories, discussing the related scientific developments that have added to our understanding of mercury processes. This review builds upon the work previously summarized in EPA's Mercury Report to Congress (USEPA, 1997).

4.5.3.1 Mercury Cycle Chemistry

Mercury occurs naturally in the environment as several different chemical species. The majority of mercury in the atmosphere (95-97%) is present in a neutral, elemental state (Hg^0) (Lin and Pehkonen, 1999), while in water, sediments and soils the majority of mercury is found in the oxidized, divalent state (Hg(II)) (Morel et al., 1998). A small fraction (percent) of this pool of divalent mercury is transformed by microbes into MeHg (CH3Hg(II)/ MeHg) (Jackson, 1998). MeHg is retained in fish tissue and is the only form of mercury that biomagnifies in aquatic food

webs (Kidd et al., 1995). As a result, MeHg concentrations in higher trophic level organisms such as piscivorous fish, birds and wildlife are often 104-106 times higher than aqueous MeHg concentrations (Jackson, 1998). Transformations among mercury species within and between environmental media result in a complicated chemical cycle. Mercury emissions from both natural and anthropogenic sources are predominantly as Hg(II) species and Hg^0 (Landis and Keeler, 2002; Seigneur et al., 2004). Anthropogenic point sources of mercury consist of combustion (e.g., utility boilers, municipal waste combustors, commercial/industrial boilers, medical waste incinerators) and manufacturing sources (e.g., chlor-alkali, cement, pulp and paper manufacturing) (USEPA, 1997). Natural sources of mercury arise from geothermic emissions such as crustal degassing in the deep ocean and volcanoes as well as dissolution of mercury from geologic sources (Rasmussen, 1994).

4.5.3.2 Mercury Processes in the Atmosphere

The relative contributions of local, regional and long range sources of mercury to fish mercury levels in a given water body are strongly affected by the speciation of natural and anthropogenic emissions sources. Elemental mercury is oxidized in the atmosphere to form the more soluble mercuric ion (Hg(II)) (Schroeder et al., 1989). Particulate and reactive gaseous phases of Hg(II) are the principle forms of mercury deposited onto terrestrial and aquatic systems because they are more efficiently scavenged from the atmosphere through wet and dry deposition than Hg0 (Lindberg and Stratton, 1998). Because Hg(II) species or reactive gaseous mercury (RGM) and particulate mercury (Hg(p)) in the atmosphere tend to be deposited more locally than Hg^0, differences in the species of mercury emitted affect whether it is deposited locally or travels longer distances in the atmosphere (Landis et al., 2004).

4.5.3.3 Mercury Processes in Soils

A portion of the mercury deposited in terrestrial systems is re-emitted to the atmosphere. On soil surfaces, sunlight may reduce deposited Hg(II) to Hg^0, which may then evade back to the atmosphere (Carpi and Lindberg, 1997; Frescholtz and Gustin, 2004; Scholtz et al., 2003). Significant amounts of mercury can be co-deposited to soil surfaces in throughfall and litterfall of forested ecosystems (St. Louis et al., 2001), and exchange of gaseous Hg^0 by vegetation has been observed (e.g., (Gustin et al., 2004).

Hg(II) has a strong affinity for organic compounds such that inorganic Hg in soils and wetlands is predominantly bound to dissolved organic matter (Mierle and Ingram, 1991). MeHg likewise forms stable complexes with solid and dissolved organic matter (Hintelmann and Evans, 1997). These complexes can dominate MeHg speciation under aerobic conditions

(Karlsson and Skyllberg, 2003). Truly dissolved and dissolved organic carbon (DOC)-complexed Hg(II) and MeHg are transported by percolation to shallow groundwater, and by runoff to adjacent surface waters (Ravichandran, 2004). Sorbed Hg(II) and MeHg are transported by erosion fluxes to depositional areas on the watershed and to adjacent surface waters (e.g., (Hurley et al., 1998).

Concentrations of MeHg in soils are generally very low. In contrast, wetlands are areas of enhanced MeHg production and account for a significant fraction of the external MeHg inputs to surface waters that have watersheds with a large portion of wetland coverage (e.g., St. Louis et al., 2001). Accordingly, there is a positive relationship between MeHg yield and percent wetland coverage (Hurley et al., 1995). Hydrology exerts an important control on the magnitude and flux of MeHg in wetland ecosystems (Branfireun and Roulet, 2002), as well as the transport of inorganic mercury deposited in a given watershed to surface waters (Babiarz et al., 2001).

4.5.3.4 Mercury Processes in Water

In a water body, deposited Hg(II) is reduced to Hg^0 by ultraviolet and visible wavelengths of sunlight as well as microbially mediated reduction pathways (Amyot et al., 2000; Mason et al., 1995). In turn, Hg^0 is oxidized back to Hg(II), driven by sunlight as well as by "dark" chemical or biochemical processes (Lalonde et al., 2001; Zhang and Lindberg, 2001). Driven by wind and water currents, dissolved Hg^0 in the water column is volatilized, which can be a significant removal mechanism for mercury in surface waters and a net source of mercury to the atmosphere (Siciliano et al., 2002).

In the water column and sediments, Hg(II) partitions strongly to silts and biotic solids, sorbs weakly to sands, and complexes strongly with dissolved and particulate organic material. The abundance of various inorganic ligands (e.g., OH^-, Cl^-, S^2-, DOC) in freshwater and saltwater ecosystems plays an important role in both oxidation and reduction of inorganic mercury as well as its bioavailability to methylating microbes. For example, reduction of Hg(II) is hypothesized to be a function of the predominance of $Hg(OH)_2$, which is inversely correlated with pH (Mason et al., 1995). Reduction of Hg(II) to Hg^0 and subsequent volatilization from the water column is important because it effectively reduces the pool of inorganic mercury that could potentially undergo conversion to MeHg.

Hg(II) and MeHg sorbed to solids settle out of the water column and accumulate on the surface of the benthic sediment layer. Surficial sediments interact with the water column via resuspension and bioturbation. The burial of sediments below the surficial zone can be a

significant removal mechanism for contaminants in surface sediments (e.g., Gobas et al., 1998; Gobas et al., 1995). The depth of the active sediment layer is a highly sensitive parameter for predicting the temporal response of different ecosystems to changes in mercury loading in environmental fate models. This is because the reservoir of Hg(II) potentially available for conversion to MeHg in the sediments is a function of the depth and volume of the active sediment layer. The compartment conducive for methylation is similarly affected (Harris and Hutchison, 2003; Sunderland et al., 2004). Physical characteristics of different ecosystem types affect estuarine mixing and sediment resuspension, which also affect the production of MeHg in the water and sediments (Rolfhus et al., 2003; Sunderland et al., 2004; Tseng et al., 2001).

4.5.3.5 Bioavailability of Inorganic Mercury to Methylating Microbes

The amount of bioavailable MeHg in water and sediments of aquatic systems is a function of the relative rates of mercury methylation and demethylation. In the water, MeHg is degraded by two microbial processes and sunlight (Barkay et al., 2003; Sellers et al., 1996). Recent research has shown that demethylating Hg-resistant bacteria may adapt to systems that are highly contaminated with total mercury, helping to explain the paradox of low MeHg and fish Hg levels in these systems (Schaefer et al., 2004).

Mass balances for a variety of lakes and coastal ecosystems show that in situ production of MeHg is often one of the main sources of MeHg in the water and sediments (Benoit et al., 1998; Bigham and Vandal, 1994; Gbundgo-Tugbawa and Driscoll, 1998; Gilmour et al., 1998; Mason et al., 1999). Sulfate-reducing bacteria (SRB) are thought to be the principle agents responsible for the majority of MeHg production in aquatic systems (Beyers et al., 1999; Compeau and Bartha, 1987; Gilmour and Henry, 1991). SRB thrive in the redoxocline, where the maximum gradient between oxic and anoxic conditions exists (Hintelmann et al., 2000). Thus, in addition to the presence of bioavailable Hg(II), MeHg production and accumulation in aquatic systems is a function of the geochemical parameters that enhance or inhibit the activity of methylating microbes, especially sulfur concentrations, redox potential (Eh) and the composition and availability of organic carbon.

A number of factors affect the bioavailabilty of Hg(II). A strong inverse relationship between complexation of Hg(II) by sulfides and MeHg production has been demonstrated in a number of studies (Benoit et al., 1999a; Benoit et al., 1999b; Craig and Bartlett, 1978; Craig and Moreton, 1986). Passive diffusion of dissolved, neutral inorganic mercury species is hypothesized as one of the main modes of entry across the cell membranes of methylating microbes (Benoit et al., 1999a; Benoit et al., 2003; Benoit et al., 1999b). Thus, the formation of

neutral, dissolved mercury species such as $HgCl_2$, $Hg(OH)_2$, HgClOH, and HgS^0(aq.), which depend on the availability of constituent ligands in the surface and interstitial waters, may strongly influence the availability of inorganic mercury to SRB, although our understanding of the forms of mercury that are bioavailable to methylating microbes is currently incomplete (Benoit et al., 2001; Benoit et al., 1999a; King et al., 2001). Additional detail is provided below on the relationship between sulfur deposition and mercury methylation.

Changes in the bioavailability of inorganic mercury and the activity of methylating microbes as a function of sulfur, carbon and ecosystem specific characteristics mean that ecosystem changes and anthropogenic "stresses" that do not result in a direct increase in mercury loading to the ecosystem but alter the rate of MeHg formation may also affect mercury levels in organisms (Grieb et al., 1990). Because mercury concentrations in fish can increase even when there has been no change in the total amount of mercury deposited in the ecosystem, environmental changes such as eutrophication, which may alter microbial activity and the chemical dynamics of mercury within an ecosystem, must be considered together with emission control strategies to effectively manage mercury accumulation in the food web.

Recent research indicates that the bioavailability or reactivity of newly deposited Hg(II) may be greater than older "legacy" mercury in the system (Hintelmann et al., 2002). These results suggest that lakes receiving the bulk of their mercury directly from deposition to the lake surface (e.g., some seepage lakes) would see fish mercury concentrations respond more rapidly to changes in atmospheric deposition than lakes receiving most of their mercury from watershed runoff. The implications of these data are also that systems with a greater surface area to watershed area ratio that receive most of their inputs directly from the atmosphere (e.g., seepage lakes) may respond more rapidly to changes in emissions and deposition of mercury than those receiving significant inputs of mercury from the catchment area.

Sulfur and Mercury Methylation. EPA's *2008 Integrated Science Assessment (ISA) for Oxides of Nitrogen and Sulfur–Ecological Criteria (Final Report)* concluded that evidence is sufficient to infer a casual relationship between sulfur deposition and increased mercury methylation in wetlands and aquatic environments. Specifically, there appears to be a relationship between SO_4^{2-} deposition and mercury methylation; however, the rate of mercury methylation varies according to several spatial and biogeochemical factors whose influence has not been fully quantified (see Figure 4-1). Therefore, the correlation between SO_4^{2-} deposition

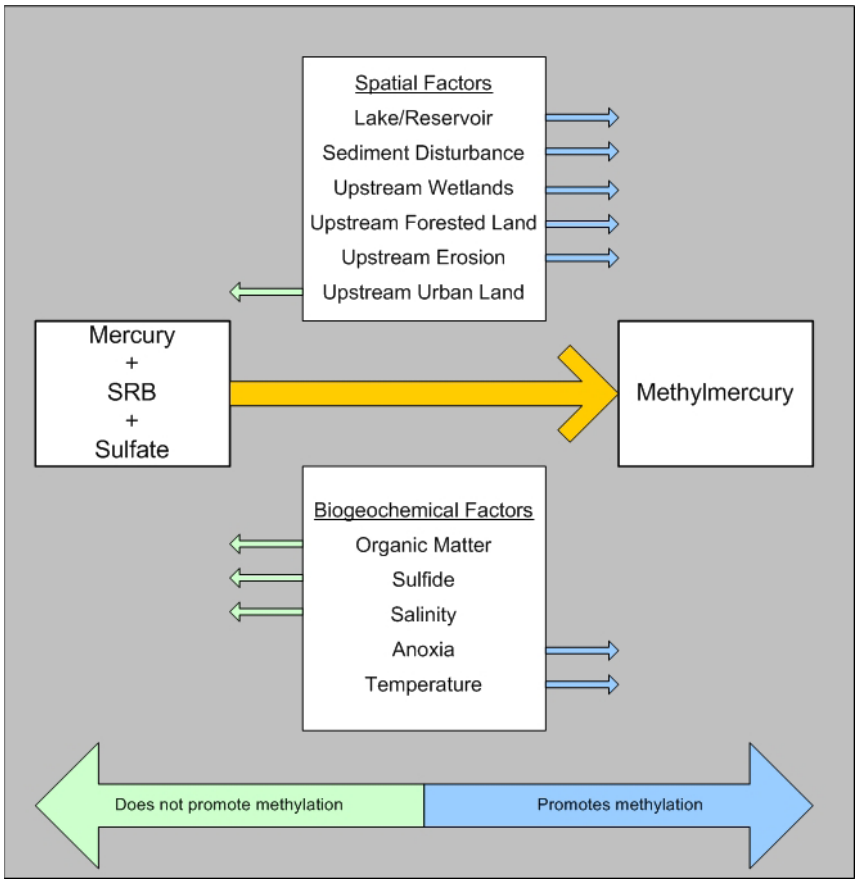

Figure 4-1. Spatial and Biogeochemical Factors Influencing MeHg Production

and MeHg could not be quantified for the purpose of interpolating the association across waterbodies or regions. Nevertheless, because changes in MeHg in ecosystems represent changes in significant human and ecological health risks, the association between sulfur and mercury cannot be neglected (EPA, 2008, Sections 4.4.1 and 4.5).

As research evolves and the computational capacity of models expands to meet the complexity of mercury methylation processes in ecosystems, the role of interacting factors may be better parsed out to identify ecosystems or regions that are more likely to generate higher concentrations of MeHg. Figure 4-2 illustrates the type of current and forward-looking research being developed by the U.S. Geological Survey (USGS) to synthesize the contributing factors of mercury and to develop a map of sensitive watersheds. The mercury score referenced in Figure 4-3 is based on SO_4^{2-} concentrations, acid neutralizing capacity (ANC), levels of dissolved organic carbon and pH, mercury species concentrations, and soil types to gauge the methylation sensitivity (Myers et al., 2007).

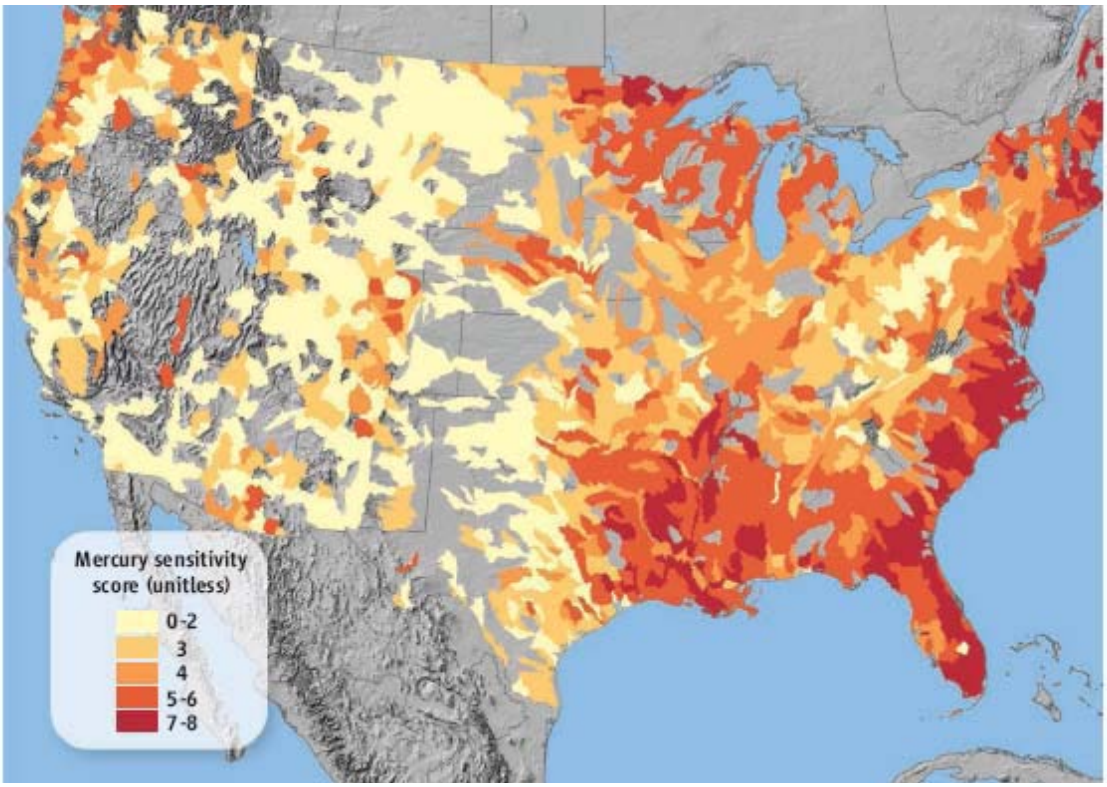

Figure 4-2. Preliminary USGS Map of Mercury Methylation–Sensitive Watersheds Derived from More Than 55,000 Water Quality Sites aqnd 2,500 Watersheds (Myers et al., 2007)

Interdependent biogeochemical factors preclude the existence of simple sulfate-related mercury methylation models (see Figure 4-2). It is clear that decreasing sulfate deposition is likely to result in decreased MeHg concentrations. Future research may allow for the characterization of a usable sulfate-MeHg response curve; however, no regional or classification calculation scale can be created at this time because of the number of confounding factors.

Decreases in SO_4^{2-} deposition have already shown promising reductions in MeHg. Observed decreases in MeHg fish tissue concentrations have been linked to decreased acidification and declining SO_4^{2-} and mercury deposition in Little Rock Lake, WI (Hrabik and Watras, 2002), and to decreased SO_4^{2-} deposition in Isle Royale in Lake Superior, MI (Drevnick et al., 2007). Although the possibility exists that reductions in SO_4^{2-} emissions could generate a pulse in MeHg production because of decreased sulfide inhibition in sulfate-saturated waters, this effect would likely involve a limited number of U.S. waters (Harmon et al., 2007). Also, because of the diffusion and outward flow of both mercurysulfide complexes and SO_4^{2-},

increased mercury methylation downstream may still occur in sulfate-enriched ecosystems with increased organic matter and/or downstream transport capabilities.

Remediation of sediments heavily contaminated with mercury has yielded significant reductions of MeHg in biotic tissues. Establishing quantitative relations in biotic responses to MeHg levels as a result of changes in atmospheric mercury deposition, however, presents difficulties because direct associations can be confounded by all of the factors discussed in this section. Current research does suggest that the levels of MeHg and total mercury in ecosystems are positively correlated, so that reductions in mercury deposited into ecosystems would also eventually lead to reductions in MeHg in biotic tissues. Ultimately, an integrated approach that involves the reduction of both sulfur and mercury emissions may be most efficient because of the variability in ecosystem responses. Reducing SO_X emissions could have a beneficial effect on levels of MeHg in many waters of the United States.

4.5.3.6 Mercury Accumulation in the Food Web

Dissolved Hg(II) and MeHg accumulate in aquatic vegetation, phytoplankton, and benthic invertebrates. Unlike Hg(II), MeHg biomagnifies though each successive trophic level in both benthic and pelagic food chains such that mercury in predatory, freshwater fish is found almost exclusively as MeHg (Bloom, 1992; Watras et al., 1998). Thus, trophic position and food-chain complexity plays an important role in MeHg bioaccumulation (Kidd et al., 1995). The chemical and physical characteristics of different ecosystems affect MeHg uptake at the base of the food chain, driving bioaccumulation at higher trophic levels. At the base of pelagic freshwater food-webs, MeHg uptake by plankton is thought to be a combination of passive diffusion and facilitated transport (Laporte et al., 2002; Watras et al., 1998). Uptake of MeHg by plankton can be enhanced or inhibited by the presence of different ligands bound to MeHg (Lawson and Mason, 1998). Similarly, the assimilation efficiency of MeHg at the base of the food chain is also affected by the type of dissolved MeHg-complexes in the water and sediments. This may be a function of differences in the ability of organisms to solubilize MeHg through digestive processes with different MeHg complexes (Lawrence and Mason, 2001; Leaner and Mason, 2002). The presence of organic ligands and high concentrations of DOC in aquatic ecosystems are generally thought to limit MeHg uptake by biota (Driscoll et al., 1995; Sunda and Huntsman, 1998; Watras et al., 1998).

In fish, MeHg bioaccumulation is a function of several uptake (diet, gills) and elimination pathways (excretion, growth dilution) (Gilmour et al., 1998; Greenfield et al., 2001). As a result, the highest mercury concentrations for a given fish species correspond to smaller, long-lived

fish that accumulate MeHg over their life span with minimal growth dilution (e.g., (Doyon et al., 1998). In general, higher mercury concentrations are expected in top predators, which are often large fish relative to other species in a waterbody.

4.5.4 Summary

In the United States, humans are exposed to MeHg mainly by consuming fish that contain MeHg. Aquatic ecosystems respond to changes in mercury deposition in a highly variable manner as a function of differences in their chemical, biological and physical properties. Depending on the characteristics of a given ecosystem, methylating microbes convert a small but variable fraction of the inorganic mercury in the sediments and water derived from human activities and natural sources into MeHg. MeHg is the only form of mercury that biomagnifies in the food web. Concentrations of MeHg in fish are generally on the order of a million times the MeHg concentration in water. In addition to mercury deposition, key factors affecting MeHg production and accumulation in fish include the amount and forms of sulfur and carbon species present in a given waterbody. Thus, two adjoining water bodies receiving the same deposition can have significantly different fish mercury concentrations.

For this analysis, EPA used the Mercury Maps (MMaps) model to estimate changes in freshwater fish mercury concentrations resulting from changes in mercury deposition after regulation of mercury emissions from U.S. coal-fired power plants. MMaps, a simplified form of the IEM-2M model applied in EPA's 1997 Mercury Study Report to Congress, is a static model that assumes a proportional relationship between declines in atmospheric mercury deposition and concentrations in fish at steady state. This means, for example, that a 50% decrease in mercury deposition rates is projected to lead to a 50% decrease in mercury concentrations in fish. MMaps does not consider the dynamics of relevant ecosystem specific factors that can affect the methylation and bioaccumulation in fish in different water bodies over time, nor does it consider the inputs of non-air sources to the watershed. In all cases, the MMaps model does not address the lag time of different ecosystems to reach steady state (i.e., when fish mercury concentrations reflect changes in atmospheric deposition). In addition, applying the MMaps model assumes that atmospheric deposition is the principle source of mercury to the waterbodies being investigated and environmental factors that affect MeHg production and accumulation in organisms will remain constant, allowing each ecosystem to reach steady state. While MMaps has several limitations, EPA knows of no alternative tool for performing a national-scale assessment of such changes.

4.6 Analysis of the Dose-Response Relationship Between Maternal Mercury Body Burden and Childhood IQ

4.6.1 Introduction

In considering possible health endpoints for quantification and monetization, EPA reviewed the scientific literature on the health effects of mercury, including the "Toxicological Effects of Methylmercury," published by the National Research Council (NRC) in 2000 (NRC, 2000).

EPA chose to focus on quantification of intelligence quotient (IQ) decrements associated with prenatal mercury exposure as the initial endpoint for quantification and valuation of mercury health benefits. Reasons for this initial focus on IQ included the availability of thoroughly-reviewed, high-quality epidemiological studies assessing IQ or related cognitive outcomes suitable for IQ estimation, and the availability of well-established methods and data for economic valuation of avoided IQ deficits, as applied in EPA's previous benefits analyses for childhood lead exposure. In the "Peer Review of EPA's Draft National-Scale Mercury Risk Assessment" (SAB, 2011 available at: http://yosemite.epa.gov/sab/sabproduct.nsf/ea5d9a9b55cc319285256cbd005a472e/aaf67ae4dd199409852578cb006bcb04!OpenDocument) the Science Advisory Board noted that a number of measures of potential neurodevelopmental effects of methylmercury exist, some of which have greater sensitivity than IQ loss. However, none were viewed by the Panel as suitable for quantitative risk estimation with a reasonable degree of scientific certainty at the present time, and none were recommended for incorporation into the analysis. IQ score has not been the most sensitive indicator of methylmercury's neurotoxicity in the populations studied. The Faroe Island study the most sensitive indicators were in the domains of language (Boston Naming), attention (continuous performance) and memory (California Verbal Learning Test), neuropsychological tests that are not subtests of IQ tests and are not highly correlated with global IQ. In the Seychelles study, the Psychomotor Development Index has been most sensitive measure and, while this is a component of the Bailey Scales of Infant Development, it is not highly correlated with cognitive measures. While the Panel agreed that the concentration-response function for IQ loss used in the risk assessment is appropriate, IQ loss is not a sensitive response to methylmercury and its use likely underestimates the impact of reducing methylmercury in water bodies.

Epidemiological studies of prenatal mercury exposure conducted in the Faroe Islands (Grandjean et al., 1997), New Zealand (Kjellstrom et al., 1989; Crump et al., 1998), and the Seychelles Islands (Davidson et al., 1998; Myers et al., 2003) have examined

neurodevelopmental outcomes through the administration of tests of cognitive functioning. Each of these studies included some but not all of the following tests: full-scale IQ, performance IQ, problem solving, social and adaptive behavior, language functions, motor skills, attention, memory and other functions. The NRC reviewed the studies and determined that "Each of the studies was well designed and carefully conducted, and each examined prenatal MeHg exposures within the range of the general U.S. population exposures" (NRC, 2000).

As part of previous analyses, EPA attempted to identify the appropriate dose-response coefficients from the Faroe Islands, New Zealand, and Seychelles Islands studies, and devised a statistical approach for combining those coefficients to provide an integrated estimate of the IQ dose-response coefficient.

For this assessment, EPA used a more recently revised estimate of the IQ dose-response function, based on a peer-reviewed study by Axelrad et al. (2007) ("the Axelrad study"). The Axelrad study estimated a dose-response relationship between maternal mercury body burden and subsequent childhood decrements in IQ using a Bayesian hierarchical model to integrate data from the Faroe Islands, New Zealand, and Seychelles Islands studies.

The Axelrad study used a linear model that goes through the origin to fit population-level dose-response relationships to the pooled data from the three studies. The application of a linear model should not be interpreted to suggest that any of the three studies used have data showing health effects from MeHg exposure at or below the RfD. The RfD is an estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 2002). EPA believes that exposures at or below the RfD are unlikely to be associated with appreciable risk of deleterious effects. It is important to note, however, that the RfD does not define an exposure level corresponding to zero risk; mercury exposure near or below the RfD could pose a very low level of risk which EPA deems to be non-appreciable. It is also important to note that the RfD does not define a bright line, above which individuals are necessarily at risk of adverse effect. Use of a linear model that goes through the origin, rather than one that reflects a threshold effect is technically more simple and practical. It associates an increment of IQ benefit with a given reduction in exposure. A linear model allows us to estimate the benefits of reductions in exposure due to power plants without a complete assessment of other sources of exposure. Other models would require information on the joint distribution of exposure from power plants and other sources to estimate the benefits of reducing the exposure due to power plants, which would require much more precise information about consumption patterns.

4.6.2 Epidemiological Studies of Mercury and Neurodevelopmental Effects

The IQ dose-response estimate is based on data from three major prospective studies investigating potential neurotoxicity of low-level, chronic mercury exposure: the Faroe Islands study, the New Zealand study, and the Seychelles Child Development Study.

In assembling the New Zealand sample, Kjellstrom et al. (1989) ascertained the fish consumption of 10,930 of 16,293 pregnant women in the study area. They identified 935 women who reportedly consumed fish at least 3 times per week. Hair samples were obtained from these women, and 73 were found to have a hair mercury level of 6 parts per million (ppm) or greater. In this group, the mean was 8.3 ppm, with a range of 6 to 86 ppm, although only one woman had a level greater than 20 ppm. Each woman with 6 ppm hair mercury or greater was matched to 3 controls—one with hair mercury between 3–6 ppm, one with hair mercury less than 3 ppm and high fish consumption, and one with hair mercury less than 3 ppm and low fish consumption. Ethnic group, age, smoking, residence time in New Zealand, and child sex were also used to select controls. The final study group included 237 children, including 57 fully matched sets of 4 children. Although children were assessed at 4 and 6 years of age, only the data collected at the older age is considered in this analysis, as the reliability and validity of neurodevelopmental testing generally increases with child age.

The Faroe Islands investigators assembled a birth cohort of 1,353 newborns recruited from three hospitals over a 21-month period in 1986–1987. In 1,022 women, two biomarkers of prenatal mercury exposure were collected: cord-blood mercury, and maternal hair mercury at delivery. Neurodevelopmental assessments of 917 children were conducted at age 7 (Grandjean et al., 1997). For these 917 children, the geometric mean concentration of mercury in cord-blood was 22.6 parts per billion (ppb) (inter-quartile range 13.1–40.5 ppb, full range 0.9–351 ppb). The geometric mean concentration of mercury in maternal hair was 4.2 ppm (inter-quartile range: 2.5–7.7 ppm, full range 0.2–39.1 ppm) (Budtz-Jorgensen et al., 2004a). Neurodevelopmental assessments of the children were conducted at age 7 years (Grandjean et al., 1997).

In assembling the Seychelles Child Development Study sample, investigators obtained hair samples from 779 pregnant women and ultimately enrolled a study sample consisting of 740 newborns. The mean maternal hair mercury level was 6.8 ppm (range 0.9–25.8 ppm) (Davidson et al., 1998). Neurodevelopmental assessments were conducted when the children were 6.5, 19, 29, and 66 months, and at 9 years. The mean maternal hair mercury level for the

643 children who participated in the assessment at age 9 years was 6.9 ppm (standard deviation 4.5 ppm) (Myers et al., 2003).

4.6.3 Statistical Analysis

Previous statistical analysis conducted by Ryan (2005) produced a dose-response relationship, integrating data from all three studies, with a central estimate of an IQ change of −0.13 IQ points (95% confidence interval −0.28, −0.03) for every ppm of mercury in maternal hair. Axelrad et al. (2007) conducted a more recent statistical analysis integrating data from the Faroe Islands, New Zealand, and Seychelles Islands studies to produce a single estimate of the IQ dose-response relationship, which is used in this RIA. Additional details of the analysis are reported in the Axelrad study and in its Supplemental Material (available at http://www.ehponline.org/docs/2007/9303/suppl.pdf). The information is summarized below.

The Axelrad study used a Bayesian hierarchical statistical model to estimate the integrated dose-response coefficient. This is similar to the approach used by the NRC panel to calculate a benchmark dose value integrating data from all three studies (NRC, 2000). The model makes use of dose-response coefficients for IQ, and also considered all other cognitive endpoints reported in the three studies in an effort to obtain more robust estimates of the IQ relationship that account for within-study (endpoint-to-endpoint) variability as well as variability across studies.

The Axelrad study assumed a linear relationship between mercury body burdens and neurodevelopmental outcomes, in keeping with the recommendation of the NRC committee (NRC, 2000). In the New Zealand and Seychelles Islands studies, all information necessary for the model was obtained from the published papers, including linear regression coefficients (Crump et al., 1998; Myers et al., 2003). The Faroe Islands publications, however, reported results with cord blood and maternal hair mercury transformed to the log scale and provided no results of linear models (Grandjean et al., 1997, 1999). A report by the Faroe Islands investigators (Budtz-Jorgensen et al., 2005) provided the additional details needed for the analysis.

The Wechsler Intelligence Scales for Children (WISC) is a standard test of childhood IQ that was used in each of the three studies. The version of the test administered in the Seychelles Islands (3rd ed.; WISC-III) was different from the earlier version used in New Zealand and the Faroe Islands (revised ed.; WISC-R). In a sample of approximately 200 children, the correlation between the Full-Scale IQ scores for the two versions was 0.89; thus the WISC-R and

WISC-III appear to measure the same constructs and generate scores with similar dispersion (Wechsler, 1991).

The WISC-R includes 10 core subtests and three supplementary subtests. For the Faroe Islands study, the investigators administered only three subtests of the WISC-R: Digit Span and Similarities (core subtests) and Block Design (a supplementary subtest). The Axelrad study used data for these three subtests to estimate an IQ–mercury coefficient for the Faroe Islands cohort. The Faroe Islands investigators fit data for these three subtests in a structural equation model (SEM) to estimate a standardized coefficient for a hypothetical Full-Scale IQ (Budtz-Jorgensen et al., 2005). In the SEM analysis of IQ, the three WISC-R subtests are viewed as representative of an underlying latent IQ variable.

To estimate the association between mercury and IQ using information from the three studies, the Axelrad study used a hierarchical random-effects model that includes study-to-study as well as endpoint–to–endpoint variability. Axelrad et al. (2007) implemented the model with a Bayesian approach, using WinBUGS version 1.4 (http://www.mrc-bsu.cam.ac.uk/bugs/). Although the Axelrad study's Bayesian analysis yields highest posterior density (HPD) intervals, the authors refer to these as confidence intervals to aid in the interpretation of results (Axelrad et al., 2007).

The integrated analysis produced a central estimate of -0.18 (95% CI, −0.378 to −0.009) IQ points for each part per million maternal hair mercury, similar to the results found for both the Faroe Islands and Seychelles studies, and lower than the estimate found in the New Zealand study. This central estimate was used as the basis for estimating IQ loss associated with prenatal MeHg exposure in this assessment.

4.6.4 Strengths and Limitations of the IQ Dose-Response Analysis

The Axelrad study produced an estimate of the relationship between maternal mercury body burdens during pregnancy and childhood IQs that incorporates data from all three epidemiologic studies judged by the NRC to be of high quality and suitable for risk assessment. The statistical approach makes use of all the available data (including information on results for related tests of cognitive function), and can be used to produce population-based estimates of a health outcome that can be readily monetized for use in benefit-cost analysis.[6]

[6]There is limited evidence directly linking IQ and methylmercury exposure in the three large epidemiological studies that were evaluated by the NAS and EPA. Based on its evaluation of the three studies, EPA believes that children who are prenatally exposed to low concentrations of methylmercury may be at increased risk of poor

There are several aspects of IQ as a metric for neurodevelopmental effects in this benefit-cost analysis that are important to recognize. Full-Scale IQ is a composite index that averages a child's performance across many functional domains, providing a good overall picture of cognitive health. An extensive body of data documents the predictive validity of full-scale IQ, as measured at school age, and late outcomes such as academic and occupational success (Neisser et al., 1996). In addition, methods are readily available for valuing shifts in IQ and thus conducting a benefits analysis of interventions that shift the IQ distribution in a population. Methods for monetization of the other tests administered in the three studies have not been developed.

It is important to recognize, however, that full-scale IQ might not be the cognitive endpoint that is most sensitive to prenatal mercury exposure. Significant inverse associations were found, in both the New Zealand and Faroe Islands studies, between prenatal mercury levels and neurobehavioral endpoints other than IQ. If the effects of mercury are highly focal, affecting only specific cognitive functions, taking full-scale IQ as the primary endpoint for a benefits analysis might underestimate the impacts. In averaging performance over diverse functions in order to compute full-scale IQ, the specific effects of mercury on only certain of these functions would be "diluted," and the estimated magnitude of the change in performance per unit change in the mercury biomarker would be underestimated.

Moreover, it is well known that there may be substantial deficits in cognitive wellbeing even in individuals with normal or above average IQ. The criterion most frequently used to identify children with learning disabilities for the purposes of assignment to special education services is a discrepancy between IQ and achievement. Specifically, the child's achievement in reading, math, or other academic areas is significantly lower than what would be expected, given his or her full-scale IQ. Thus, there are deficits in cognitive functioning that are not captured by IQ scores. For example, two of the most sensitive endpoints in the Faroe Islands study were the Boston Naming Test, which assesses word retrieval, and the California Verbal Learning Test-Children, which assesses the acquisition and retention of information presented verbally. Depending on the severity of the deficits, a child who has deficits in either of these skills could be at a considerable disadvantage in the classroom setting and at substantial educational risk. Neither of these abilities is directly assessed by the WISC-R or WISC-III, however, and so do not explicitly contribute to a child's IQ score. Therefore, benefits

---

performance on neurobehavioral tests, such as those measuring attention, fine motor function, language skills, visual-spatial abilities (like drawing), and verbal memory. For this analysis, EPA is adopting IQ as a surrogate for the neurobehavioral endpoints that NAS and EPA relied upon for the RfD.

calculations relying solely on IQ decrements are likely to underestimate the benefits to cognitive functioning of reduced mercury exposures. In additions, impacts on other neurological domains (such as motor skills and attention/behavior) are not represented by IQ scores and thus are also excluded from the benefits analysis.

As discussed above, the Faroe Islands study did not include testing for full-scale IQ. For the Axelrad study, an estimate of a dose-response coefficient for full-scale IQ was estimated using the three subtests. While this extrapolation introduces some uncertainty, information has been presented that demonstrates a high correlation between the subtests and full-scale IQ scores.

While the Seychelles and New Zealand studies use maternal hair mercury as the exposure biomarker, the Faroe Islands study uses cord blood mercury. For purposes of the integrated analysis, it was necessary to express results from all three studies in the same terms. Several studies have examined the relationship between hair mercury and blood mercury, and have reported hair:blood ratios typically in the range of 200 to 300 (see ATSDR, 1999, pages 249–252 for a review). However, these studies generally do not use cord blood mercury, which is the exposure metric in the Faroe Islands study. One analysis found that mercury concentrations in cord blood are, on average, 70 percent higher than those in maternal blood (Stern and Smith, 2003). For conversion of Faroe Islands data from cord blood mercury to maternal hair mercury, the Axelrad study used data specific to this population, indicating a median maternal hair:cord blood mercury ratio of 200 (Budtz-Jorgensen et al., 2004a).

One uncertainty concerning the New Zealand study is the strong influence of one child in the study population with a particularly high maternal hair mercury level. Published analyses of the New Zealand study presented results with data for this child both included and excluded (Crump et al., 1998). In keeping with the conclusions of the NRC (2000), the integrated dose-response analysis in the Axelrad study made use of the dose-response coefficients calculated with this child omitted. A sensitivity analysis using the New Zealand coefficient with this child included results in an integrated dose-response coefficient that is reduced in magnitude by 25 percent (−0.125 versus a primary central estimate of −0.18).

Some uncertainty is also associated with the Seychelles study due to the exclusion of some members of the cohort from the data reported by Myers et al. (2003) and used as input to this integrated dose-response analysis. The Seychelles researchers did not include a small number of outliers (defined as observations with model residuals exceeding 3 standard deviation units), and no results are available for the full cohort. However, the authors report

that “In all cases, the association between prenatal MeHg exposure and the endpoint was the same, irrespective of whether outliers were included” (Myers et al., 2003).

Finally, the integrated dose-response analysis assumes the exposures assigned to each study subject are accurate representations of true exposure. In reality, there is likely to be some discrepancy between measured and actual exposures, for example, due to variation in hair length. Alternatively, the true exposure of interest may have been during the first trimester of pregnancy, whereas exposures in maternal hair and cord blood measured at birth reflect exposures later in pregnancy. Presence of exposure measurement error could introduce a bias in the results, most likely towards the null (Budtz-Jorgensen et al., 2004b).

4.6.5 Possible Confounding from Long-Chained Polyunsaturated Fatty Acids

Maternal consumption of fish during pregnancy exposes the fetus to long-chain polyunsaturated fatty acids (LCPUFAs), believed to be beneficial for fetal brain development, and to the neurotoxicant MeHg (Helland et al., 2003; Daniels et al., 2004; Dunstan et al., 2006; Judge et al., 2007). Reports from the Seychelles Islands study cohort have suggested a negative impact of MeHg exposure, accompanied by a simultaneous beneficial effect of omega-3 LCPUFAs on children’s development (Davidson et al., 2008; Strain et al., 2008). It is unclear whether this result was evidence for independent influences of MeHg and LCPUFAs or effect modification. A recent study by Lynch et al. (2010) used varying coefficient models to characterize the interaction of mercury and nutritional covariates (Hastie and Tibshirani, 1993), including omega-3 LCPUFAs, using data from the Seychelles Islands study.

The Seychelles Islands study cohort of mother-child pairs had fish consumption averaging 9 meals per week. Lynch et al., (2010) assessed maternal nutritional status for five different nutritional covariates known to be present in fish (n-3 LCPUFA, n-6 LCPUFA, iron status, iodine status, and choline) and associated with children’s neurological development. The study also included prenatal MeHg exposure (measured in maternal hair).

Lynch et al., (2010) examined two child neurodevelopmental outcomes (Bayley Scales Infant Development-II (BSID-II) Mental Developmental Index (MDI) and Psychomotor Developmental Index (PDI)), each administered at 9 and at 30 months. The varying coefficient models allowed the possible interactions between each nutritional component and MeHg to be modeled as a smoothly varying function of MeHg as an effect modifier. Iron, iodine, choline, and omega-6 LCPUFAs had little or no observable modulation at different MeHg exposures. In contrast the omega-3 LCPUFA docosahexaenoic acid had beneficial effects on the BSID-II PDI that were reduced or absent at higher MeHg exposures. The results from Lynch et al. (2010)

suggest a potentially useful modeling method that could shed further light on the issue of interactions between nutritional covariates.

A recent study by Rice et al. (2010) considered possible confounding in a probabilistic assessment of the health benefits of reducing MeHg exposure in the United States. In deciding on a dose-response relationship between MeHg exposure and effects on IQ loss, the authors chose to use the central estimate from the Axelrad study, noting however that Axelrad et al. (2007) did not explicitly consider possible confounding of the MeHg-IQ relationship by the concurrent consumption of LCPUFAs that might enhance cognitive development and bias downward the observed regression coefficient estimates from the Faroe Islands, New Zealand, and Seychelles Islands studies. Rice et al. (2010) therefore multiplied the central estimate from Axelrad et al. (2010) by an adjustment factor to offset the possible downward bias from inadequate confounder control. A factor of 1.5 was selected "to acknowledge the recent argument of Budtz-Jorgensen et al. (2007) that the parameter estimates from the three epidemiological studies may be biased downward by a factor of approximately 2 because of failure to adequately control for confounding" (Rice et al., 2010).

There remains uncertainty with respect to the nature and magnitude of potential confounding between LCPUFAs and MeHg, and the associated effects on childhood neurodevelopment due to maternal ingestion during pregnancy. Additional research is needed to provide further clarity on this issue, but recent studies such as those referenced above reinforce the view that fish consumption during pregnancy should be approached as a case of multiple exposures to nutrients and to MeHg, with a complex and potentially interactive set of risks and benefits related to infant development. Due to the remaining uncertainty regarding the potential confounding between LCPUFAs and MeHg exposure, we have not incorporated any factors or other quantitative adjustments into this assessment.

4.7 Mercury Benefits Analysis Modeling Methodology

4.7.1 Introduction

This section describes the methodology used to model fishing behavior and associated MeHg exposure levels. The methodology incorporates data, assumptions, and analytical techniques already described in previous sections. Sections 4.7.2 and 4.7.3 below describe elements of the methodology applied to develop a national-scale estimate of benefits associated with avoided IQ loss among freshwater recreational anglers. Chapter 7 section 7.11 describes a variation of the methodology used to estimate risk levels (as measured by IQ loss) among modeled high-risk subpopulations.

4.7.2 Estimation of Exposed Populations and Fishing Behaviors

This section describes the methodology used to estimate the average daily ingestion of mercury (g/day) through noncommercial freshwater fish consumption (HgI) for selected populations of interest. Because the primary measurable health effect of concern—developmental neurological abnormalities in children—occurs as a result of in-utero exposures to mercury, the specific population of interest in this case is prenatally exposed children. To identify and estimate the size of this exposed population, the benefits analysis focuses on pregnant women in freshwater recreational angler households.

Generally speaking, estimating mercury exposures for this exposure pathway and population of interest requires three main components:

N_i = size of the exposed population of interest i (annual number of pregnant women in freshwater angler households during the year),

CHg_i = average concentration (ppm) of methyl mercury in noncommercial freshwater fish filets consumed by population i, and

C_i = average daily consumption rate (gm/day) of noncommercial freshwater fish by population i.

The flow diagram in Figure 4-3 illustrates the approach used to estimate the first two components of this equation—N_i and CHg_i. It shows the spatial scale of the data used to estimate these components and describes how these components are interrelated. For the third component—C_i—recommendations from EPA's *Environmental Exposure Factors Handbook* (EPA, 1997) were used to estimate an average consumption rate estimate for recreationally caught freshwater fish.

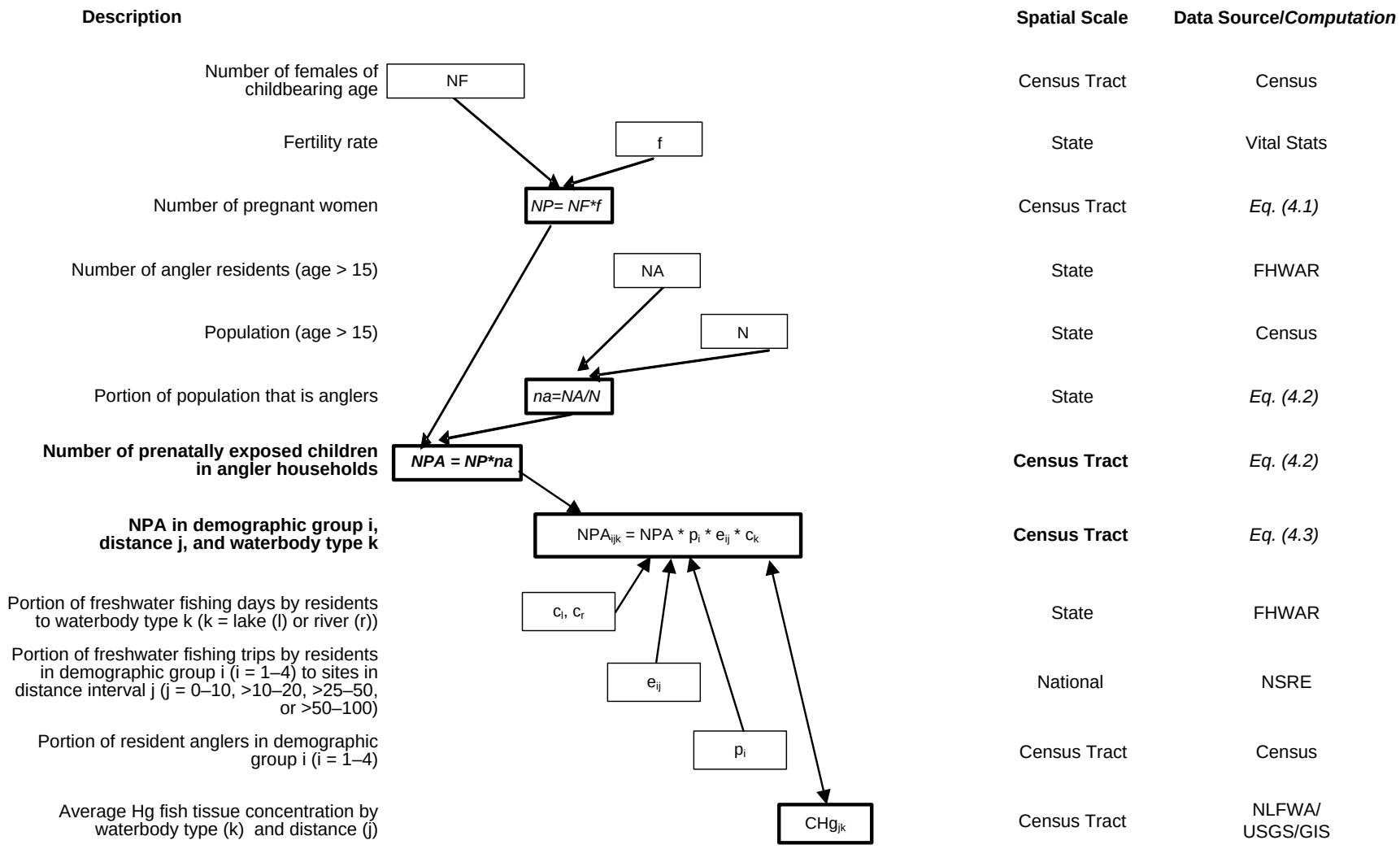

Figure 4-3. Methodology for Estimating and Linking Exposed Populations and Levels of Mercury Exposure

First, 2000 Census data (U.S. Census Bureau, Census 2000 Summary File 3, Detailed Tables, United States) were used to define the size, age, gender distribution, and income of the populations within each census tract in the 48 contiguous U.S. states.

1. Estimating the number of pregnant women (NP) living in the census tract as

$$NP = NF * f_s, \quad (4.1)$$

where

NF = number of females aged 15 to 44 in the tract (Census 2000) and

f_s = state-level general fertility rate (average number of live births in a year per 1,000 women aged 15 to 44) (2006 Vital Statistics).

2. Estimating the annual number of prenatally exposed children in angler households (NPA) as

$$NPA = NP*(NA_s/N_s), \quad (4.2)$$

where

NA_s = state-level number of angler residents (FHWAR) and

N_s = adult population of state s (Census).

Using Eq. (4.2) to estimate NPA implies that (1) the fraction of pregnant women in a state who are in freshwater angler households is equal to the fraction of households in the state that include freshwater anglers (i.e., pregnant women are no more or less likely than the rest of the state population to live in households with freshwater anglers) and (2) the fraction of households in the state that includes freshwater anglers is equal to the fraction of adult residents in the state who are freshwater anglers.

To estimate NPA for years after 2000, it was assumed that state-level fertility rates (f_s) and angler participation rates (NA_s/N_s) would remain constant; however, the number of women of childbearing age in each block (NF) was increased based on county-level population growth projections (Woods and Poole, 2008). In other words, for the period 2000 to 2016, the estimated NPA for each census tract was assumed to increase at the same rate as the projected annual population growth rates for females 15 to 44 in their corresponding counties.

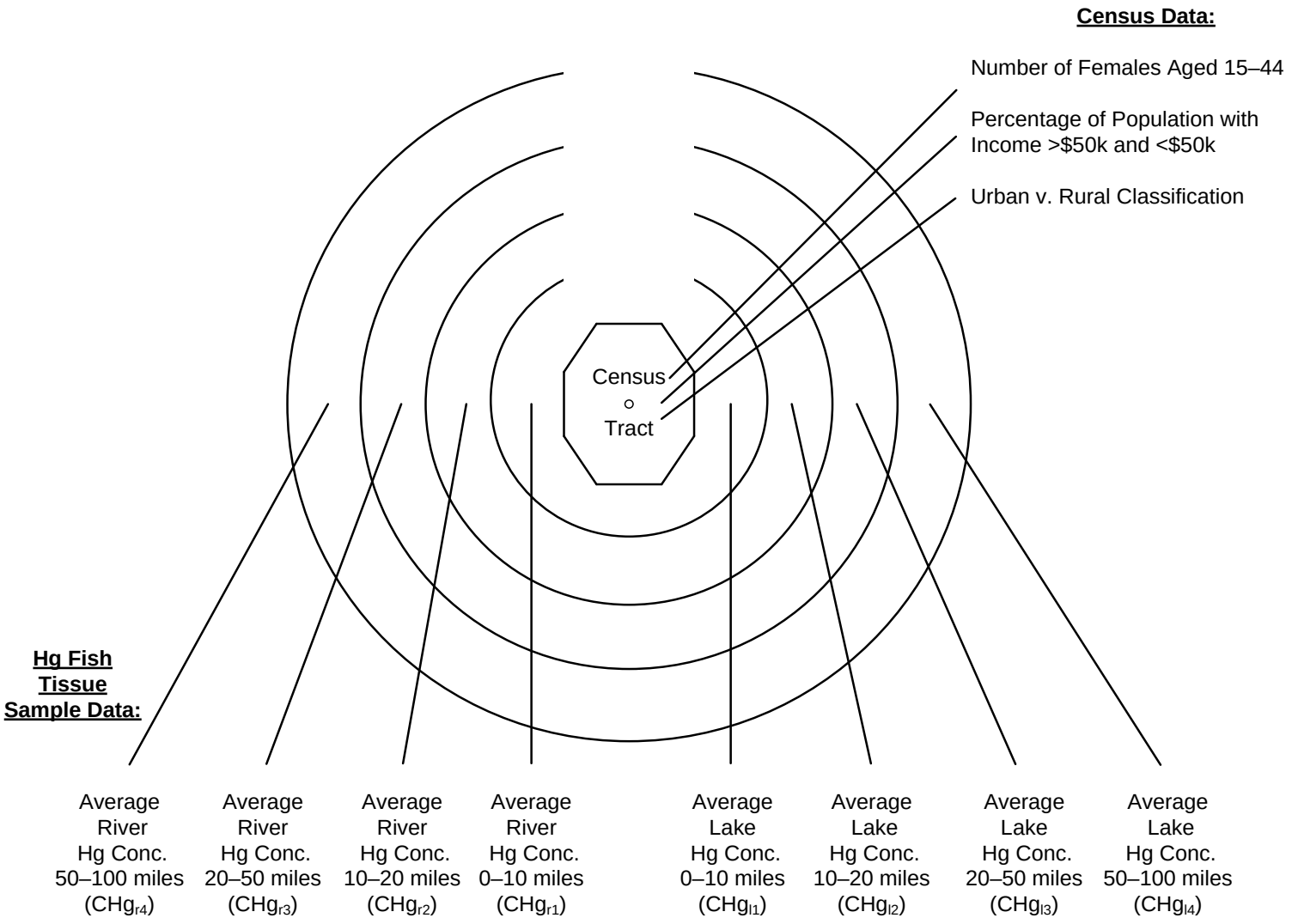

Figure 4-4. Linking Census Tracts to Demographic Data and Mercury Fish Tissue Samples

Fourth, to match exposed populations in each tract with mercury concentrations, we first divided the exposed population into four distinct demographic groups (i = 1 – 4): urban/low income, urban/high income, nonurban/low income, and nonurban/high income. To estimate the portion of households in each demographic group (p_i for i = 1 – 4), tract-level Census data were used to specify (1) the percentage of the population in each tract that resides in an urban area and (2) the percentage with household income less than $50,000 (i.e., the portion in the low-income group).

In addition, it was assumed that

1. each exposed individual in a census tract is associated with freshwater fishing in a single distance interval and a single waterbody type (i.e., all the fish they consume comes from the same distance and type of waterbody), [7] and

2. the exposed populations in each census tract (rather than just the fishing trips) are distributed across the distance intervals and waterbody types according to the estimated proportions (i.e., parameters c, e, and p shown in Figure 4-4).

More specifically, a maximum of 32 separate exposed subpopulations were defined for each census tract:

$$NPA_{ijk} = NPA * p_i * e_{ij} * c_k \text{ (for all i, j, and k)} \qquad (4.3)$$

for

i = 1 – 4 demographic subgroup in the census tract,

j = 1 – 4 distance interval, and

k = lake or river.

(See Figure 4-3 for definitions of p_i, e_{ij}, and c_k).

Using this approach, we were able to separately match each subpopulation NPA_{ijk} with the census tract's average mercury concentration for the corresponding distance and waterbody category (CHg_{jk}).

[7] An alternative would be to assume that all anglers in the census tract have the same distribution of trips across distance intervals and water types. This assumption would imply no variation in per-capita mercury exposures within a census tract, but it would not affect the estimates of *total* exposure and *total* IQ losses in the tract.

To approximate the percentage freshwater fishing trips (and exposed individuals) from each census tract matched to each waterbody type (c_l or c_r), we used state-level averages. These averages were calculated for each state, based on the portion of residents' freshwater fishing trips that are to each waterbody type, based on 2001 FHWAR data.

Data from NSRE 1994 were used to approximate the percentage of freshwater fishing trips (and exposed individuals) matched to different distances from anglers' residential location. Four distance intervals were defined as 0–10 miles, >10–20 miles, >20–50 miles, and >50–100 miles. Based on self-reported trip distance information from nearly 2,000 respondents (see Appendix B for details), each of these distance categories was associated with roughly 20% of the reported trips in the NSRE sample. Four distinct demographic groups were also found to have significantly different average travel distances for freshwater fishing in the NSRE sample: high-income urban, high-income rural, low-income urban, and low-income rural. An annual household income threshold of $50,000 (in 2000 dollars) was used to define high and low income, because it is close to the median value for both the NSRE sample and the U.S. population. The portion of trips for each demographic group (i = 1 – 4) to each distance interval (j = 1 – 4) is defined as e_{ij}. The estimated values for e_{ij} are reported in Appendix B.

To estimate average daily mercury ingestion rates for each exposed subpopulation n=ijk, we applied the following equation:

$$HgI_n = CHgFC_n * C_n = (CHg_n * CCF) * C_n \quad (4.4)$$

where

HgI = average daily mercury ingestion rate (µg/day);

CHg = average mercury concentration in uncooked freshwater fish (ppm);

CCF = cooking conversion factor: ratio of mercury concentration in cooked fish to mercury concentration in uncooked fish (= 1.5);

CHgFC = average mercury concentration in cooked freshwater fish (ppm); and

C = average daily self-caught freshwater cooked fish consumption rate (gm/day) = 8 gm/day.

To determine an appropriate daily fish consumption rate (C) for the analysis, EPA conducted an extensive review of existing literature characterizing self-caught freshwater fish consumption. Based on this review, it was decided that the ingestion rates for recreational

freshwater fishers, specified as "recommended" in EPA's *Environmental Exposure Factors Handbook* (EPA, 1997) (mean of 8 gm/day and 95th percentile of 25 gm/day), represented the most appropriate values to use in this analysis. These recommended values were derived based on ingestion rates from four studies conducted in Maine, Michigan, and Lake Ontario (Ebert et al., 1992; Connelly et al., 1996; West et al., 1989; West et al., 1993), which measured annual average daily intake rates for self-caught freshwater fish by all recreational fishers including consumers and non-consumers of fish. The mean values presented in these four studies ranged from 5 to 17 gm/day, while the 95th percent values ranged from 13 to 39 gm/day (Note: the 39 gm/day value actually represents a 96th percent value). The EPA "recommended values" were developed by considering the range and spread of means and 95th percentile values presented in the four studies. EPA recognizes that using mean and 95th percentile consumption rates based on these four studies may not be representative of fishing behavior across the entire 48-state study area and that regional trends in consumption may differ from the values used in this analysis. Moreover, rates of consumption by pregnant women in freshwater angler households may be different from those of the recreational fishers themselves. However, EPA believes that these four studies do represent the best available data for developing recreational fisher ingestion rates in the United States.

Because the consumption rate estimate C is for cooked fish and the mercury concentrations are estimated for uncooked filet, a conversion factor (CCF) was applied to estimate mercury concentrations in cooked fish. Cooking fish tends to reduce the overall weight of fish by approximately one-third (Great Lakes Sport Fish Advisory Task Force, 1993). Because volatilization of mercury is unlikely to occur during cooking, the overall amount of mercury will stay unchanged during cooking, and the concentration of mercury will increase by a factor of roughly 1.5 (Morgan, Berry, and Graves, 1997).

4.7.3 Estimation of Lost Future Earnings

Estimating the IQ decrements in children that result from mothers' ingestion of mercury required two steps. First, based on the estimated average daily maternal ingestion rate, the expected mercury concentration in the hair of exposed pregnant women was estimated as follows:

$$CHgH_n = (0.08)^{-1} * (HgI_n/W), \quad (4.5)$$

where

CHgH = average mercury concentration in maternal hair (ppm) and

W = average body weight for female adults below age 45 (= 64 kg).

This conversion rate between average daily ingestion rate and maternal hair concentration is based on the one compartment model developed by Swartout and Rice (2000). The 2002 EPA Workshop on Methylmercury Neurotoxicity recommended that this one compartment model might be better suited than the PBPK model in modeling dose-response (EPA, 2002). The average body weight estimate (W) was based on EPA's Exposure Factor Handbook (EPA, 1997).

Second, to estimate the expected IQ decrement in offspring resulting from in-utero exposure to mercury through mothers' fish consumption, the following dose-response relationship was applied:

$$dIQ_n = 0.18 * CHgH_n, \quad (4.6)$$

where

dIQ = IQ decrement in exposed mother/child (IQ pts).

The 0.18 dose-response coefficient in this equation is based on the summary findings reported in Axelrad et al. (2007).

The valuation approach used to assess monetary losses due to IQ decrements is based on an approach applied in previous EPA analyses (EPA, 2008). The approach expresses the loss to an affected individual resulting from IQ decrements in terms of foregone future earnings (net of changes in education costs) for that individual. These losses were estimated using the following equation:

$$V_n = VIQ * dIQ_i, \quad (4.7)$$

where

V = present value of net loss per exposed mother/child (2006 dollars) and

VIQ = net loss per change in IQ point.

The net loss per IQ point decrement is estimated based on the following relationship:

$$VIQ = (z * PVY) – (s * PVS), \quad (4.8)$$

where

PVY = median present value of lifetime earnings,

PVS = present value of education costs per additional year of schooling,

z = percentage change in PVY per 1-point change in IQ, and

s = years of additional schooling per 1-point increase in IQ.

The estimate for PVY is derived using earnings and labor force participation rate data from the 2006 Current Population Survey (CPS) and assuming (1) an individual born today would begin working at age 16 and retire at age 67; (2) the growth rate of wages is 1% per year, adjusted for survival probabilities and labor force participation by age; and (3) lifetime earnings are discounted back to the year of birth. Using a 3% discount rate, the resulting present value of median lifetime earnings is $555,427 in 2006 dollars.

Estimates of the average effect of a 1-point increase in IQ on lifetime earnings (z) range from a 1.76% increase (Schwartz, 1994) to a 2.379% increase (Salkever, 1995). The percentage increases in the two studies reflect both the direct impact of IQ on hourly wages and indirect effects on annual earnings as the result of additional schooling and increased labor force participation. The estimate for *s* is based on Schwartz (1994) who reports an increase of 0.131 years of schooling per IQ point.

In addition to this positive net effect on earnings, an increase in IQ is also assumed to have a positive effect on the amount of time spent in school (s) and on associated costs (PVS). The range of estimate for *s* is based on Schwartz (1994) who reports an increase of 0.131 years of schooling per IQ point and Salkever (1995) who reports an increase of 0.1007 years.

The estimate for PVS is derived using an estimate of $16,425 per additional year of schooling in 1992 dollars (EPA, 2005), which is based on U.S. Department of Education data reflecting both direct annual expenditures per student and annual average opportunity cost (i.e., lost income from being in school). We assume these costs are incurred when an individual born today turns 19, based on an average 12.9 years of education among people aged 25 and over in the United States. Discounting at a 3% rate to the year of birth results in an estimate of $13,453 per additional year of schooling in 2006 dollars.

To incorporate (1) uncertainty regarding the size of z and (2) different assumptions regarding the discount rate, the resulting value estimates for the average net loss per IQ point

decrement (VIQ) are expressed as a range. Assuming a 3% discount rate, VIQ ranges from $8,013 (using the Schwartz estimate for z and s) to $11,859 (using the Salkever estimates). With a 7% discount rate assumption, the VIQ estimates range from $893 to $1,958.

4.8 Mercury Benefits and Risk Analysis Results

4.8.1 Baseline Incidence

Applying the methodology described in Section 4.7, we first used GIS to link census tract centroids in the continental United States with HUC-12 watersheds containing mercury fish tissue sample data for 1995 to 2007. We found that, out of the 64,500 tracts in the 48-state area, almost all of them are located within 100 miles of at least one HUC-12 with freshwater mercury fish tissue sampling data. Therefore, very few tracts were entirely excluded from the analysis due to a lack of sampling data within 100 miles. Table 4-4 reports the number of tracts linked to HUC-level river or lake mercury concentration estimates within each distance interval. As expected, this number decreases as the size of the distance interval decreases. For example, 33% are within 10 miles of a HUC-12 containing a lake sample, and 52% are within 10 miles of a HUC-12 containing a river sample.

Table 4-4 also reports the average river and lake HUC-level fish tissue mercury concentrations found within each distance interval. Assuming that the 1995 to 2007 samples are representative of baseline conditions in 2005, the distance-specific mean lake concentrations range from 0.26 to 0.3 ppm, and the mean river concentrations vary from 0.25 to 0.27 ppm.

Table 4-4 also reports corresponding river and lake mercury concentration estimates for a 2016 base case scenario. This scenario represents total mercury deposition from all global natural and anthropogenic sources based on projected 2016 conditions, including future anticipated regulations (e.g., Transport Rule). As described in Section 4.4, CMAQ air quality modeling runs were used to estimate average mercury deposition levels by HUC-12 sub-watershed under both the 2005 base case and the 2016 base case scenarios. For this analysis, it is assumed that HUC-level fish tissue mercury concentrations would change (between the two scenarios) by the same percentage as the change in modeled deposition levels. Overall, the mean concentrations decline by 6% to 9% in the 2005 base case compared with the 2016 base case scenarios.

With these tract-level mercury concentration estimates, we then estimated the size of the exposed populations (NPA) in 2005 and 2016. These estimates are reported in Table 4-5. As

described in Section 4.7.2, a separate exposed population (NPA_{jk}) was estimated for each distance interval (j = 1 – 4) and waterbody (k = lake or river) combination at each tract. If mercury concentration data were not available for a specific distance-waterbody combination, then the corresponding exposed population for the tract (NPA_{jk}) was not included in the analysis. Consequently, the exposed population estimates reported in Table 4-5 are best interpreted as lower-bound estimates of the total exposed population. Excluding potentially exposed populations from the analysis because of missing/unavailable mercury concentration data reduced the total exposed population estimate by roughly 44%. These excluded populations include the portions of the tract-level exposed populations that were matched with fishing trip travel distances that either (1) did not overlap with at least one HUC-12 with sampling data or (2) were greater than 100 miles (see Appendix C). For 2005, there were estimated to be 239,174 prenatally exposed children, and for 2016 the estimate is 244,286 prenatally exposed children.

Table 4-4. Summary of Baseline Mercury Fish Tissue Concentrations

Distance from Tract Centroid	N[a]	2005 Base Case				2016 Base Case			
		Min (ppm)	Mean (ppm)	Max (ppm)	Median (ppm)	Min (ppm)	Mean (ppm)	Max (ppm)	Median (ppm)
Lake Sampling Sites									
0–10 miles	20,998	0.000	0.297	3.561	0.198	0.000	0.276	3.420	0.178
>10–20 miles	35,149	0.000	0.285	3.561	0.209	0.000	0.264	3.420	0.187
>20–50 miles	55,885	0.000	0.289	3.561	0.223	0.000	0.270	3.420	0.202
>50–100 miles	61,820	0.000	0.264	2.333	0.241	0.000	0.247	2.251	0.227
River Sampling Sites									
0–10 miles	33,342	0.006	0.246	4.967	0.185	0.005	0.224	4.924	0.168
>10–20 miles	44,493	0.006	0.269	4.967	0.195	0.005	0.247	4.924	0.174
>20–50 miles	54,970	0.019	0.270	4.480	0.203	0.019	0.251	4.441	0.183
>50–100 miles	62,868	0.023	0.267	4.967	0.214	0.022	0.251	4.924	0.192

[a] Number of tracts (out of 64,419) with at least one HUC-12 with sample data in the distance interval.

Table 4-5. Baseline Levels of Mercury Exposure and IQ Impacts Due to Freshwater Self-Caught Fish Consumption

State	Number of Census Tracts with Hg Samples w/in 100 Miles	2005 Base Case					2016 Base Case				
		Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses	Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses
		Mean per Tract	Total in State				Mean per Tract	Total in State			
Total	**63,978**	**3.74**	**239,174**	**3.04**	**0.11**	**25,544.9**	**3.82**	**244,286**	**2.84**	**0.10**	**24,419.4**
AL	1,081	5.51	5,956	3.28	0.12	685.9	5.53	5,981	3.04	0.11	638.3
AR	623	6.45	4,017	3.80	0.13	537.1	6.55	4,084	3.66	0.13	525.9
AZ	1,097	3.17	3,476	2.21	0.08	269.8	3.75	4,117	2.18	0.08	316.3
CA	6,801	1.19	8,089	6.04	0.21	1,716.4	1.26	8,599	5.74	0.20	1,734.0
CO	1,045	3.53	3,693	1.20	0.04	155.3	3.92	4,101	1.18	0.04	169.8
CT	812	2.47	2,003	4.58	0.16	322.2	2.38	1,929	4.29	0.15	291.3
DC	181	2.23	404	1.67	0.06	23.7	2.03	367	1.35	0.05	17.4
DE	196	1.77	348	1.98	0.07	24.2	1.79	352	1.71	0.06	21.2
FL	3,144	3.28	10,299	5.24	0.18	1,897.5	3.71	11,651	5.17	0.18	2,118.9
GA	1,614	8.38	13,525	3.14	0.11	1,494.8	8.74	14,111	2.88	0.10	1,431.0
IA	791	6.39	5,052	1.21	0.04	215.3	6.18	4,888	1.15	0.04	197.5
ID	280	6.30	1,765	2.43	0.09	150.9	7.13	1,996	2.31	0.08	162.3
IL	2,950	2.33	6,884	1.83	0.06	442.3	2.32	6,831	1.49	0.05	356.9
IN	1,409	5.47	7,711	2.20	0.08	596.7	5.51	7,759	1.90	0.07	519.2
KS	716	2.08	1,490	2.38	0.08	124.8	2.06	1,478	2.34	0.08	121.8
KY	993	4.99	4,954	2.19	0.08	381.9	4.92	4,889	1.90	0.07	326.1

(continued)

Table 4-5. Baseline Levels of Mercury Exposure and IQ Impacts Due to Freshwater Self-Caught Fish Consumption (continued)

State	Number of Census Tracts with Hg Samples w/in 100 Miles	2005 Base Case: Number of Prenatally Exposed Children (NPA)		2005 Base Case: Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	2005 Base Case: Average IQ Loss per Exposed Child (dIQ)	2005 Base Case: Total IQ Point Losses	2016 Base Case: Number of Prenatally Exposed Children (NPA)		2016 Base Case: Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	2016 Base Case: Average IQ Loss per Exposed Child (dIQ)	2016 Base Case: Total IQ Point Losses
		Mean per Tract	Total in State				Mean per Tract	Total in State			
LA	1,103	6.91	7,623	3.82	0.13	1,022.9	6.59	7,269	3.77	0.13	962.6
MA	1,357	1.81	2,456	5.40	0.19	466.0	1.74	2,359	5.04	0.18	417.7
MD	1,210	2.23	2,703	2.16	0.08	204.8	2.35	2,840	1.76	0.06	176.2
ME	344	4.66	1,602	5.12	0.18	288.3	4.31	1,484	5.05	0.18	263.4
MI	2,701	3.89	10,520	2.72	0.10	1,005.0	3.79	10,234	2.37	0.08	854.0
MN	1,294	11.53	14,915	2.86	0.10	1,501.2	11.71	15,157	2.77	0.10	1,474.7
MO	1,311	3.66	4,796	1.80	0.06	302.7	3.75	4,911	1.70	0.06	294.2
MS	604	9.18	5,546	5.11	0.18	996.2	9.32	5,632	4.98	0.18	986.9
MT	267	3.62	965	2.40	0.08	81.5	3.68	984	2.38	0.08	82.3
NC	1,554	5.13	7,976	3.29	0.12	921.5	5.33	8,280	2.95	0.10	859.1
ND	224	2.89	647	3.43	0.12	78.1	2.79	626	3.41	0.12	74.9
NE	500	3.97	1,984	1.60	0.06	111.9	4.03	2,014	1.56	0.05	110.5
NH	272	3.68	1,001	5.53	0.19	194.5	3.71	1,010	5.39	0.19	191.2
NJ	1,930	1.02	1,965	3.28	0.12	226.5	1.00	1,936	2.98	0.10	202.7
NM	244	1.75	426	1.74	0.06	26.0	1.89	461	1.77	0.06	28.6
NV	471	1.70	803	3.78	0.13	106.8	2.09	985	3.60	0.13	124.8
NY	4,791	1.41	6,770	3.86	0.14	918.4	1.35	6,486	3.54	0.12	807.0

(continued)

Table 4-5. Baseline Levels of Mercury Exposure and IQ Impacts Due to Freshwater Self-Caught Fish Consumption (continued)

State	Number of Census Tracts with Hg Samples w/in 100 Miles	2005 Base Case					2016 Base Case				
		Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses	Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses
		Mean per Tract	Total in State				Mean per Tract	Total in State			
OH	2,923	4.11	12,015	1.61	0.06	678.8	3.93	11,489	1.30	0.05	527.0
OK	987	5.65	5,580	3.07	0.11	602.9	5.73	5,653	3.03	0.11	601.4
OR	754	5.14	3,877	2.80	0.10	382.1	5.43	4,095	2.81	0.10	404.3
PA	3,116	2.40	7,485	2.30	0.08	605.9	2.31	7,194	1.91	0.07	482.2
RI	233	1.55	361	6.01	0.21	76.2	1.53	356	5.15	0.18	64.5
SC	864	7.39	6,388	4.43	0.16	995.4	7.59	6,559	4.08	0.14	941.0
SD	225	3.29	740	1.77	0.06	45.9	3.20	719	1.72	0.06	43.6
TN	1,253	4.95	6,204	3.01	0.11	656.7	5.06	6,335	2.76	0.10	615.5
TX	4,310	3.97	17,127	2.83	0.10	1,701.2	4.32	18,633	2.67	0.09	1,748.9
UT	482	3.95	1,905	2.05	0.07	137.3	4.68	2,254	2.06	0.07	163.5
VA	1,524	3.66	5,580	2.61	0.09	512.7	3.82	5,820	2.19	0.08	448.7
VT	179	3.50	627	3.85	0.14	84.8	3.37	604	3.70	0.13	78.6
WA	1,315	3.67	4,823	1.69	0.06	287.2	3.90	5,133	1.68	0.06	302.8
WI	1,313	8.03	10,543	2.77	0.10	1,026.2	7.85	10,309	2.59	0.09	938.1
WV	466	6.53	3,042	2.10	0.07	224.3	6.10	2,840	1.66	0.06	166.1
WY	124	4.13	512	1.97	0.07	35.5	3.99	495	1.97	0.07	34.3

For each exposed population, we then estimated their average mercury ingestion rate (HgI) using Equation (4.4) and the IQ loss associated with this exposure level. As reported in Table 4-5, in 2005, the average estimated mercury ingestion rate for the population of exposed pregnant women was 3.04 ug/day. For 2016, the ingestion rate was estimated to be 2.84 ug/day (6.6% lower). The corresponding average IQ loss per prenatally exposed child was 0.11 in 2005 and 0.10 in 2016. Multiplying these average IQ losses by the size of the exposed population, the total loss in IQ points due to mercury exposures through consumption of self-caught freshwater fish was estimated to be 25,545 in 2005. For the 2016 base case, the total decrease in IQ points was estimated to be 24,419 (4.4% lower).

4.8.2 IQ Loss and Economic Valuation Estimates

In addition to the base case scenarios described above, CMAQ air quality modeling runs were used to estimate average mercury deposition levels for three emissions control scenarios:

- **2005 EGU Zero-Out.** This scenario represents total mercury deposition from all global natural and anthropogenic sources *except for U.S. EGUs* based on current-day conditions.

- **2016 EGU Zero-Out.** This scenario represents total mercury deposition from all global natural and anthropogenic sources *except for U.S. EGUs* based on projected 2016 conditions, including future anticipated regulations (e.g., Transport Rule).

- **2016 Toxics Rule.** This scenario represents total mercury deposition from all global natural and anthropogenic sources based on projected 2016 conditions, including future anticipated regulations (e.g., Transport Rule) *and* the Toxics Rule.

For these three scenarios, it was again assumed that the HUC-level fish tissue mercury concentrations would change (relative to the 2005 base case) by the same percentage as the change in modeled deposition levels.

Mercury exposure and IQ loss estimates were then derived for these three scenarios, using the exposed population estimates for the relevant year (2005 or 2016) and the corresponding mercury concentration estimates for the relevant emission scenario (zero-out or Toxics Rule). In addition, the valuation methodology summarized in Section 4.7.2 (in particular, Equation [4.7]) was applied to estimate the present value of IQ loss estimates for the two base case and three emissions control scenarios.

To assess the aggregate benefits of reductions in EGU emissions, we evaluated five *emission reduction scenarios*.

- 2005 EGU zero-out (relative to 2005 base case)
- 2016 base case (relative to 2005 base case)
- 2016 EGU zero-out (relative to 2016 base case)
- 2016 Toxics Rule (relative to 2005 base case)
- 2016 Toxics Rule (relative to 2016 base case)

The benefits of each emission reduction scenario are calculated as the difference (i.e., decrease) in total present value of IQ losses between the selected emission control scenario and the selected base case scenario.

4.8.3 Primary Results for National Analysis of Exposures from Recreational Freshwater Fish Consumption

Table 4-6 summarizes the aggregate national IQ and present-value loss estimates for the two base case and three emission control scenarios. The highest losses are estimated for the 2005 base case. For the population of prenatally exposed children included in the analysis (almost 240,000, as reported in Table 4-5), mercury exposures under baseline conditions during the year 2005 are estimated to have resulted in more than 25,500 IQ points lost. Assuming a 3% discount rate, the present value of these losses ranges from $210 million to $290 million.[8] This range of total loss estimates is based on the range of per-IQ-point value (VIQ) estimates summarized in Section 4.7.3. These losses represent expected present value of declines in future net earnings over the entire lifetimes of the children who are prenatally exposed during the year 2005. With a 7% discount rate, the present value range is considerably lower: $23 million to $51 million.

The lowest losses are estimated to result from the 2016 zero-out scenario, with total IQ losses of less than 24,000 among roughly 244,000 prenatally exposed children and present values of these losses ranging from $200 to $290 million (3% discount rate).

For the five emission reduction scenarios described above, Table 4-7 reports estimates of aggregate nationwide benefits associated with reductions in mercury exposures and resulting reductions in IQ losses. Most importantly, the benefits of the 2016 Toxics Rule

[8]Monetized benefits estimates are for an immediate change in MeHg levels in fish. If a lag in the response of MeHg levels in fish were assumed, the monetized benefits could be significantly lower, depending on the length of the lag and the discount rate used. As noted in the discussion of the Mercury Maps modeling, the relationship between deposition and fish tissue MeHg is proportional in equilibrium, but the MMaps approach does not provide any information on the time lag of response.

Table 4-6. Summary Estimates of the Aggregate Size and Present Value of IQ Losses Under Alternative Base Case and Emissions Control Scenarios

Scenario	Average IQ Loss per Prenatally Exposed Child (dIQ)	Total IQ Losses from One Year of Exposure	Value of Total IQ Losses in 2016 (millions of 2007$)	
			3% Discount Rate	7% Discount Rate
2005 base case	0.1068	25,545	$210 to $310	$23 to $51
2005 EGU zero-out	0.0985	23,561	$190 to $290	$22 to $47
2016 base case	0.1000	24,419	$200 to $300	$22 to $49
2016 EGU zero-out	0.0971	23,722	$200 to $290	$22 to $48
2016 Toxics Rule	0.0979	23,909	$200 to $290	$22 to $48

Table 4-7. Aggregate Benefit Estimates for Reductions IQ Losses Associated with Alternative Emissions Reduction Scenarios

Emission Reduction Scenario	Decrease in Average IQ Loss per Prenatally Exposed Child (dIQ)[a]	Decrease in Total IQ Losses from One Year of Exposure	Value of Total IQ Losses in 2016 (millions of 2007$)	
			3% Discount Rate	7% Discount Rate
2005 EGU zero-out (relative to 2005 base case)	0.00829	1,983	$16 to $24	$1.8 to $4.0
2016 base case (relative to 2005 base case)	0.00684	1,126	$9.3 to $14	$1.0 to $2.3
2016 EGU zero-out (relative to 2016 base case)	0.00285	697	$5.7 to $8.5	$0.6 to $1.4
2016 Toxics Rule (relative to 2005 base case)	0.00893	1,636	$13 to $20	$1.5 to $3.3
2016 Toxics Rule (relative to 2016 base case)	0.00209	511	$4.2 to $6.2	$0.47 to $1.0

[a] As reported in Table 4-5, the estimated number of prenatally exposed children is 239,174 in 2005 and 244,286 in 2016.

scenario (relative to the 2016 base case) are estimated to range between $4.2 million and $6.2 million (assuming a 3% discount rate), because of an estimated 511 point reduction in IQ losses. These benefits are 73% as large as the benefits of the 2016 zero-out scenario (relative to the

same 2016 base case). Relative to the *2005* base case, the benefits of the 2016 Toxics Rule scenario range from $13 million to $20 million (3% discount). Despite growth in the exposed population from 2005 to 2016, the changes from the 2005 base case to the 2016 base case account for 69% of these benefits, while the changes from the 2016 base case to the 2016 Toxics Rule account for 31%.

4.8.5 Discussion of Assumptions, Limitations, and Uncertainties

Uncertainty regarding the model results and estimates reported in Section 4.8 can arise from several sources. Some of the uncertainty can be attributed to model uncertainty. For example, to estimate exposures a number of different modeling approaches have been selected and combined. The separate model components are summarized in Figure 4-4 and equations (4.) to (4.8), each of which simplifies potentially complex processes. The results, therefore, depend importantly on how these models are selected, specified, and combined.

Another important source of uncertainty can be characterized as input or parameter uncertainties. Each of the modeling components discussed in this report requires summary data and estimates of key model parameters. For example, estimating IQ losses associated with consumption of freshwater fish requires estimates of the size of the exposed population of interest, the average mercury concentrations in consumed fish, the freshwater fish consumption rate for the exposed population, and the concentration-response relationship between mercury ingestion and IQ loss. All of these inputs are measured with some degree of uncertainty and can affect, to differing degrees, the confidence range of our summary results. The discussion below identifies and highlights some of the key model parameters, characterizes the source and extent of uncertainties associated with them, and characterizes the potential effects of these uncertainties on the model results.

To organize this discussion, we discuss different components of the modeling framework separately. This section first discusses issues related to estimating the mercury concentrations and then those related to estimating the exposed population. After that, it discusses issues related to matching these two components and then concludes by discussing the estimation of mercury ingestion through fish consumption.

4.8.5.1 Mercury Concentration Estimates

As described in Section 4.2.2, the mercury concentration estimates for the analysis come from several different sources, including fish tissue sample data from the National Listing of Fish Advisories (NLFA) and several other state- and national-level sources. These estimates

were then used to approximate mercury concentrations across the study area. Some of the key assumptions, limitations, and uncertainties associated with these estimates are the following:

- The fish tissue sampling data from various sources are subject to measurement and reporting error and variability. The NLFA is the largest and most detailed source of data on mercury in fish; however, even this system was not centrally designed (e.g., by EPA) using a common set of sampling and analytical methods. Rather, states collected the data primarily to support the development of advisories, and the data are submitted voluntarily to EPA. Each state uses different methods and criteria for sampling and allocates different levels of resources to their monitoring programs. In addition, there are uncertainties regarding the precise locations (lat/long coordinates) of some of the samples. The heterogeneity and potential errors across state sampling programs can bias the results in any direction and contribute to uncertainty.

- The fish tissue sampling data were assigned as either lake or river samples, based on the site name and/or the location coordinates mapped to the nearest type of waterbody. This process also involves measurement error and may have resulted in misclassifications for some of the samples. These errors are not expected to bias results, but they contribute to uncertainty.

- The mercury concentration estimates used in the model were based on simple temporal and spatial averages of reported fish tissue samples. This approach assumes that the mercury samples are representative of "local" conditions (i.e., within the same HUC-12) in similar waterbodies (i.e., rivers or lakes). However, even though states use a variety of approaches to monitor and sample fish tissue contaminants, in some cases, the sampling sites are selected to target areas with high levels of angler activity and/or a high level of pollution potential. To the extent that sample selection procedures favor areas with relatively high mercury, the spatial extrapolation methods used in this report will tend to overstate exposures. These approaches also implicitly assume that mercury concentration estimates are strongly spatially correlated, such that closer sampling sites (i.e., from the same HUC or distance interval) provide more information about mercury concentrations than more distant sites. To the extent that spatial correlation is weaker than assumed, this will increase the degree of uncertainty in the modeling results.

- To generate average mercury fish tissue concentration estimates, all available samples from the three main data sources (1995–2009) and from freshwater fish larger the 7 inches were included in the analysis. Smaller fish were excluded to better approximate concentrations in the types of fish that are more likely to be consumed, and samples from years before 1995 were excluded to better represent more recent conditions. Even with these sample selection procedures, average concentration estimates from the retained samples may still under or overestimate actual concentrations in currently consumed fish.

4.8.5.2 Exposed Population Estimates

The methods described in Section 4.7 to estimate the total exposed population of interest in 2005 and 2016 involve the following key assumptions, limitations, and uncertainties:

- The approach relies on data from the FHWAR to estimate state-level freshwater angler activity levels, including freshwater fishing participation rates and lake-to-river trip ratios. Each of these data elements is measured with some error in the FHWAR, but they are based on a relatively large sample. More importantly the state-level averages are applied to each modeled census tract in the state; therefore, the model fails to capture within-state variation in these factors, which contributes to uncertainty in the model estimates.

- The analysis also uses state-level fertility rate data to approximate the rate of pregnancy among women of childbearing age in angler households for a smaller geographic area. The state-level fertility rates from the National Vital Statistics are estimated with relatively little error; however, applying these rates to specific census tracts (and specifically to women in angler households) does involve considerably more uncertainty.

- The approach assumes that, in each census tract, the percentage of women who live in freshwater angler households (i.e., households with at least one freshwater angler) is equal to the percentage of the state adult population that fishes. Applying the state-level participation rate to approximate the conditions at a block level creates uncertainty. More importantly, however, using individual-based fishing participation rates to approximate household rates is likely to underestimate the percentage of women living in freshwater angler households.[9] Unfortunately, data on household participation levels in freshwater fishing are not readily available.

- Census tract populations are only included in the model if they are matched to distance intervals and waterbody types that have spatial overlap with at least one HUC-12 sub-watershed containing a mercury concentrations estimate for that waterbody type. By design, this approach undercounts the exposed population (by roughly 40 to 45%) and, therefore, leads to underestimates of national aggregate baseline exposures and risks and underestimates of the risk reductions and benefits resulting from mercury emission reductions.

- All of the tract-level population estimates are based on Census 2000 data, which are projected forward to 2005 and 2016 using county-level growth projections for the subpopulations of interest from Woods and Poole (2008). Therefore, the 2005 and 2016 population estimates incorporate uncertainty from both the growth

[9]For example, hypothetically if one out of every three members in each household fished, the population rate would be 33%, but the household rate would be 100%.

projections themselves and from transferring the county-level growth estimates to the tract level.

The purpose of the analysis of potentially high risk subpopulations is not to estimate the size of the exposed population but rather to characterize the distribution of individual-level risks in the subpopulations of interest. Nevertheless, the size and spatial distribution of the total population in each group was used as a proxy for characterizing the spatial distribution of pregnant women in freshwater fishing households in each group.

The main assumption underlying this approach is that the expected proportion of the subgroup's population in each Census tract that consists of pregnant women in fishing households is the same across the selected census tracts. The main limitation of this assumption is that it does not allow or account for spatial variation in (1) the percentage of the subpopulation that are women of childbearing age, (2) the percentage of these women that are pregnant (i.e., fertility rate) and (3) the freshwater angler participation rates for the subgroups of interest. Unfortunately, spatially varying data for the last component (fishing participation rates among the subpopulations of interest) are not readily available. This assumption is not expected to bias the results but it does contribute to uncertainty in the estimated distributions of individual-level risks.

4.8.5.3 Matching of Exposed Populations to Mercury Concentrations

The methods described in Section 4.7 to match the exposed population estimates with the corresponding mercury concentration estimates involve the following key assumptions, limitations, and uncertainties:

- For the aggregate benefits analysis, tract-level exposed populations are assigned to waterbody types based on state-level ratios of lake-to-river fishing days (from the FHWAR). They are further assigned to distance intervals based on observed travel distance patterns in national fishing data (NSRE, 1994). Both of these assignment methods involve uncertainty, but particularly the second method because it is based on much more aggregate data and on a much smaller and more dated sample of anglers. This approach does not take into account the physical characteristics of the area in which the population is located. In particular, the allocation of exposures to lakes or rivers at different distances from each census tract does not take into account the presence or number of these waterbodies in each distance interval. Using these state and national level estimates to represent conditions at a local (i.e., census tract) level increases uncertainty in the model results, but it is not expected to bias the results in either direction.

- For the analysis of potentially high-risk populations, these methods and assumptions were slightly modified. In particular, because these analyses focus on low-income and/or subsistence fishing populations, all trips were assumed to occur within 20 miles of the census tract. Unfortunately, it is difficult to evaluate the accuracy of this restriction due to limited data on travel distances for the subgroups of interest.

One potentially important factor that is not included for matching populations and mercury concentrations is the effect of fish consumption advisories on fishing behavior. Evidence summarized in Jakus, McGuinness, and Krupnick (2002) suggests that awareness of advisories by anglers is relatively low (less than 50%), and even those who are aware do not always alter their fishing behavior. Nonetheless, anglers are less likely to fish in areas with advisories. Unfortunately, we were not able to reliably quantify the reduction and redistribution of fishing trips in either model to account for fish advisories. By excluding these effects, the model estimates are likely to overstate mercury exposures.

4.8.5.4 Fish Consumption Estimates

One of the most influential variables in both modeling approaches is the rate of self-caught freshwater fish consumption. The following key assumptions, limitation, and uncertainties are associated with the methods used:

- For the aggregate analysis we have assumed 8 g/day for the general population in freshwater angler households (based on recommendations in EPA's EFH). Unfortunately, data are not available to reliably vary this rate with respect to characteristics of the population across the entire study area. Uncertainty regarding the true average fish consumption rate has a direct effect on uncertainty for the aggregate exposure and benefit estimates. Because a single consumption rate is applied uniformly across the entire exposed population and because it is a multiplicative factor in the model, the two uncertainties are directly proportional to one another. The recommended 8 g/day rate is based on four studies with mean estimates ranging from 5 g/day (37% less than 8) to 17 g/day (113% more than 8). If it is assumed that this range of estimates represents the uncertainty in the *mean* freshwater fish consumption rate for the study population, then the resulting uncertainty range for the estimated *mean* mercury ingestion level (and resulting IQ loss) will also be between −37% and +113% of the mean mercury ingestion level.

- To analyze the distributions of individual-level risks in potentially high risk subpopulations, we applied empirical distributions of fish consumption rates for specific subpopulations. One of the main limitations of this approach is that these empirical distributions are based on relatively small and localized samples. In particular, the estimated distribution of consumption rates for low-income African American subsistence/recreational fishers in the Southeastern U.S. (see Table 4-3) is based on a very small sample (N=39) drawn from one location (Columbia, SC). The

sample sizes for the other groups, particularly the Hispanic (N= 45) and Laotian (N=54) populations are also small; therefore, there is considerable uncertainty regarding how well these empirical consumption rate distributions reflect actual rates of consumption in the subpopulations of interest.

Another related and potentially influential variable in the modeling approach is the assumed conversion factor for mercury concentrations between uncooked and cooked fish. Studies have found that cooking fish tends to reduce the overall weight of fish by approximately one-third (Great Lakes Sport Fish Advisory Task Force, 1993) without affecting the overall amount of mercury. But these conversion rates depend on cooking practices and types of fish. Uncertainty regarding this conversion factor also has a proportionate effect on the modeling results.

4.8.5.5 Measurement and Valuation of IQ Related Effects

The models for estimating and valuing IQ effects involve three main steps. The first step is translating maternal mercury ingestion rates to mercury levels in hair. The second step is translating differences in hair mercury concentrations during pregnancy to IQ changes in offspring. The third step is translating IQ losses into expected reductions in lifetime earnings. As discussed below, each of these steps also involves the following assumptions, limiations, and uncertainties:.

- The conversion of mercury ingestion rate to mercury concentration in hair is based on uncertainty analysis of a toxicokinetic model for estimating reference dose (Swartout and Rice, 2000). The conversion factor was estimated by considering the variability and uncertainty in various inputs used in deriving the dose including body weight, hair-to-blood mercury ratio, half-life of MeHg in blood, and others. Therefore, there is uncertainty regarding the conversion factor between hair mercury concentration and mercury ingestion rate. Although, the median conversion factor (0.08 µg/kg-day/hair-ppm) is used, the 90% confidence interval is from 0.037 to 0.16 µg/kg day/hair-ppm. Any change in the conversion factor will proportionately affect the benefits results because of the linearity of the model.

- The dose-response model used to estimate neurological effects on children because of maternal mercury body burden is susceptible to various uncertainties. In particular, there are three main concerns. First, there are other cognitive end-points that have stronger association with MeHg than IQ point losses. Therefore, using IQ points as a primary end point in the benefits assessment may underestimate the impacts. Second, blood-to-hair ratio for mercury is uncertain, which can cause the results from analyses based on mercury concentration in blood to be uncertain. Third, uncertainty is associated with the epidemiological studies used in deriving the dose-response models.

- With regard to the relationship between prenatal methylmercury exposure and childhood IQ loss, we expect greater uncertainty in associated estimates of IQ loss as exposure levels increase beyond those observed in the primary studies (i.e., Faroe Islands, New Zealand, Seychelles Islands studies) used to derive the dose-response function. In particular, high-end total exposure estimates for some of the subsistence-level fishing subpopulations included in this assessment likely exceed levels observed in the three primary studies.

- To parameterize the dose-response relationship between maternal hair concentrations and IQ loss for this analysis, we applied the results of an integrative study by Axelrad et al. (2007). The implications of applying this study include the following:

  – This approach may confound potentially positive cognitive effects of fish consumption and, more specifically, omega-3 fatty acids. Results from Rice (2010) offer a reasonable, but highly uncertain, estimate for offsetting the possible downward bias resulting from the positive confounding effects of fatty acids. Rice's high coefficient reflects the central estimate of Axelrad but adjusted upwards by a factor of 1.5 to "acknowledge the recent argument of Budtz-Jorgensen (2007) that the parameter estimates from these three epidemiological studies (Faroe Islands, Seychelles Islands, New Zealand) may be biased downward by a factor of approximately 2 because of failure to adequately control for confounding." A third study, Oken (2008), analyzes a cohort in Massachusetts and also seems to support a higher "Axelrad-plus" coefficient range due to evidence of fatty acid confounding (i.e., positive cognitive effects of fatty acids in fish may have previously led to underestimates of mercury-attributable IQ loss). This study offers further qualitative support for a higher-end estimate but is limited by the fact that it did not control for the children's home environment, which is generally a significant factor in early cognitive development.

  – The dose-response coefficient from the Axelrad et al. study is sensitive to the exclusion of one outlier data point from the Seychelles study. Including the outlier would reduce the effect size by about 25 percent. If this outlier actually reflects the true response for a subset of the populations, then risks (as modeled) could be biased high specifically for this subpopulation

  – Because the dose-response coefficient is applied uniformly across the entire exposed population and is a multiplicative factor in the model, the uncertainty in this parameter has a directly proportional effect on the reported risk and benefit estimates. In other words, adjusting the absolute value of the dose-response coefficient upward by a factor of 1.5 (i.e., based on Rice, 2010) would yield reductions in IQ losses and benefits from mercury emission reductions that are also greater by a factor of 1.5.

- The valuation of IQ losses is based on a unit-value approach developed by EPA, which estimates that the average effect of a 1-point reduction in IQ is to reduce the present value of net future earnings. Three key assumptions of this unit-value approach are that (1) there is a linear relationship between IQ changes and net earnings losses, (2) the unit value applies to even very small changes in IQ, and (3) the unit value will remain constant (in real present value terms) for several years into the future. Each of these assumptions contributes to uncertainty in the result. In particular the unit value estimate is itself subject to two main sources of uncertainty.
  - The first source is directly related to uncertainties regarding the average reductions in future earnings and years in school as a result of IQ changes. The average percentage change estimates are subject to statistical error, modeling uncertainties, and variability across the population. To address these uncertainties we have included in the analysis and reported results a range of values for this parameter, based on statistical analyses by Salkever (1995) and Schwartz (1994).
  - The second main source of uncertainty is the estimates of average lifetime earnings and costs of schooling. Both of these estimates are derived from national statistics from the early 1990s, but they are also subject to statistical error, modeling uncertainties, and variability across the population. It is also worth noting that the lost future earnings estimates do not include present value estimates for nonwage/nonsalary earnings (i.e., fringe benefits) and household (nonmarket) production. Based on the results of Grosse et al. (2009), including these factors would increase the present value of median earnings (both explicit and implicit) by a factor of roughly 1.9. However, it is not known whether IQ changes have a similar effect on these other (implicit) earnings.

4.8.5.6 Unquantified Benefits

In addition to the uncertainties discussed above associated with the benefit analysis of reducing exposures to MeHg from recreational freshwater angling, we are unable to quantify several additional benefits, which adds to the uncertainties in the final estimate of benefits.

Table 4-20 displays the health and ecosystem effects associated with MeHg exposure that are discussed in Section 4.2.2 for which we are currently unable to quantify. We note that specifically with regard to health effects, the NRC (2000) provided the following observation: "Neurodevelopmental effects are the most extensively studied sensitive end point for MeHg exposure, but there remains some uncertainty about the possibility of other health effects at low levels of exposure. In particular, there are indications of immune and cardiovascular effects, as well as neurological effects emerging later in life, that have not been adequately studied."

Table 4-8. Unquantified Health and Ecosystem Effects Associated with Exposure to Mercury

Category of Health or Ecosystem Effect	Potential Health or Ecosystem Outcomes
Neurologic Effects	Impaired cognitive development
	Problems with language
	Abnormal social development
Other Health Effects[a]	Associations with genetic, autoimmune and cardiovascular effects
Ecological Effects[a]	Survival, reproductive, behavioral, and neurological effects in wildlife (birds, fish, and mammals)

[a] These are potential effects and are not quantified because the literature is either contradictory or incomplete.

In addition to the health and ecosystem effects that we are not able to quantify, we are currently unable to quantify exposures to other segments of the U.S. population including consumption of commercial seafood and freshwater fish (produced domestically as well as imported from foreign sources) and consumption of recreationally caught seafood from estuaries, coastal waters, and the deep ocean. These consumption pathways impact additional recreational anglers who are not modeled in our benefits analysis as well as the general U.S. population. Reductions in domestic fish tissue concentrations can also impact the health of foreign consumers (consuming U.S. exports). Because of technical/theoretical limitations in the science, EPA is unable to quantify the benefits associated with several of these fish consumption pathways. For example, reductions in U.S. power plant emissions will result in a lowering of the global burden of elemental mercury, which will likely produce some degree of reduction in mercury concentrations for fish sourced from the open ocean and freshwater and estuarine waterbodies in foreign countries. In the case of mercury reductions for fish in the open ocean, complexities associated with modeling the linkage between changes in air deposition of mercury and reductions in biomagnification and bioaccumulation up the food chain (including open ocean dilution and the extensive migration patterns of certain high-consumption fish such as tuna) prevent the modeling of fish obtained from the open ocean. In the case of commercial fish obtained from foreign freshwater and estuarine waterbodies, although technical challenges are associated with modeling long-range transport of elemental mercury and the subsequent impacts to fish in these distant locations, additional complexities such as accurately modeling patterns of harvesting and their linkages to commercial consumption in the United States prevent inclusion of foreign-sourced freshwater and estuarine fish in the primary benefits analysis.

Finally, with regard to commercially-produced freshwater fish sourced in the United States (i.e., fish from catfish, bass, and trout farms), we are unable to accurately quantify

effects from this consumption pathway because many of the fish farms operating in the United States use feed that is not part of the aquatic food web of the waterbody containing the fish farm (e.g., use of agricultural-based supplemental feed). In addition, many of the farms involve artificial "constructed" waterbody environments that are atypical of aquatic environments found in the regions where those farms are located, thereby limiting the applicability of Mercury Maps' assumption in linking changes to mercury deposition to changes in mercury fish tissue concentrations (e.g., waterbodies may have restricted or absent watersheds and modified aquatic chemistry, which can effect methylation rates and impact time scales for reaching steady-state mercury fish tissue concentrations following reductions in mercury deposition). Some research indicates that the recycling of water at fish farms can magnify the mercury concentration because the system does not remove mercury as it is recycled, while newly deposited mercury is added to the system. Thus, additional research on aquaculture farms is necessary before a benefits analysis can be conducted.

Exclusion of these commercial pathways means that this benefits analysis, although covering an important source of exposure to domestic mercury emissions (recreational freshwater anglers), excludes a large and potentially important group of individuals. Recreational freshwater consumption accounts for approximately 10 to 17% of total U.S. fish consumption, and 90% is derived from commercial sources (domestic seafood, aquaculture, and imports) (EPA, 2005).

In conclusion, several unquantified benefits associated with this analysis add to the overall uncertainty in estimating total benefits. To the extent that the proposed rule will reduce mercury deposition from power plants over estuarine areas, coastal, and open ocean waters, there would be a subsequent reduction in mercury fish tissue concentrations in these different waterbodies and an associated benefit from avoided decrements in IQ and other known health and ecosystem effects.

4.8.6 Overall Conclusions

4.8.6.1 Total Baseline Incidence of IQ Loss: Self-Caught Fish Consumption among Recreational Freshwater Anglers

- Out of 64,500 census tracts in the continental U.S., 63,978 are located within 100 miles of at least one HUC-12 watershed with freshwater mercury fish tissue sampling data, and therefore were included in the modeling of IQ loss among recreational freshwater anglers.

- Approximately 240,000 prenatally exposed children were modeled, with an average IQ loss of 0.11 and 0.10 IQ points, respectively, from self-caught freshwater fish consumption for the 2005 and 2016 base case scenarios.

- The highest estimated state-specific average IQ loss among children of freshwater recreational anglers is 0.21 IQ points under the 2005 base case scenario, in both California and Rhode Island.

- Total estimated IQ loss from self-caught freshwater fish consumption among children of recreational anglers is estimated at 25,555 and 24,419 IQ points, respectively, for the 2005 and 2016 base case scenarios.

- The present economic value of baseline IQ loss for 2005 ranges from $210 million to $310 million, assuming a 3% discount rate, and from $23 million to $51 million, assuming a 7% discount rate.

- The present economic value of baseline IQ loss for 2016 ranges from $200 million to $300 million, assuming a 3% discount rate, and from $22 million to $49 million, assuming a 7% discount rate.

4.8.6.2 Avoided IQ Loss and Economic Benefits due to Regulatory Action: Self-Caught Fish Consumption among Recreational Freshwater Anglers

- Eliminating all mercury air emissions from U.S. EGUs in 2016 would result in an estimated 0.00893 fewer IQ points lost per prenatally exposed child from self-caught freshwater fish consumption, as compared with the 2005 base case scenario.

- The present economic value of avoided IQ loss from eliminating all mercury air emissions from U.S. EGUs in 2016 is estimated at a range of $5.7 million to $8.5 million, assuming a 3% discount rate, and $0.6 million to $1.4 million, assuming a 7% discount rate.

- Reduced mercury air emissions due to implementation of the Toxics Rule in 2016 would result in an estimated 0.00209 fewer IQ points lost per prenatally exposed child from self-caught freshwater fish consumption, as compared with the 2016 base case scenario.

- The present economic value of avoided IQ loss from reduced mercury air emissions due to implementation of the Toxics Rule in 2016 is estimated at a range of $4.2 million to $6.2 million, assuming a 3% discount rate, and $0.47 million to $1 million, assuming a 7% discount rate.

4.9 Benefits Associated with Reductions in Other HAP than Mercury

Even though emissions of air toxics from all sources in the U.S. declined by approximately 42 percent since 1990, the 2005 National-Scale Air Toxics Assessment (NATA) predicts that most Americans are exposed to ambient concentrations of air toxics at levels that have the potential to cause adverse health effects (U.S. EPA, 2011d).[10] The levels of air toxics to which people are exposed vary depending on where people live and work and the kinds of activities in which they engage. In order to identify and prioritize air toxics, emission source types and locations that are of greatest potential concern, U.S. EPA conducts the NATA. [11] The most recent NATA was conducted for calendar year 2005 and was released in March 2011. NATA includes four steps:

1) Compiling a national emissions inventory of air toxics emissions from outdoor sources

2) Estimating ambient and exposure concentrations of air toxics across the United States

3) Estimating population exposures across the United States

4) Characterizing potential public health risk due to inhalation of air toxics including both cancer and noncancer effects

Based on the 2005 NATA, EPA estimates that about 5 percent of census tracts nationwide have increased cancer risks greater than 100 in a million. The average national cancer risk is about 50 in a million. Nationwide, the key pollutants that contribute most to the overall cancer risks are formaldehyde and benzene.[12,13] Secondary formation (e.g., formaldehyde forming from other emitted pollutants) was the largest contributor to cancer risks, while stationary, mobile and background sources contribute almost equal portions of the remaining cancer risk.

[10]The 2005 NATA is available on the Internet at http://www.epa.gov/ttn/atw/nata2005/.

[11]The NATA modeling framework has a number of limitations that prevent its use as the sole basis for setting regulatory standards. These limitations and uncertainties are discussed on the 2005 NATA website. Even so, this modeling framework is very useful in identifying air toxic pollutants and sources of greatest concern, setting regulatory priorities, and informing the decision making process. U.S. EPA. (2011) 2005 National-Scale Air Toxics Assessment. http://www.epa.gov/ttn/atw/nata2005/

[12]Details on EPA's approach to characterization of cancer risks and uncertainties associated with the 2005 NATA risk estimates can be found at http://www.epa.gov/ttn/atw/nata1999/riskbg.html#Z2.

[13]Details about the overall confidence of certainty ranking of the individual pieces of NATA assessments including both quantitative (e.g., model-to-monitor ratios) and qualitative (e.g., quality of data, review of emission inventories) judgments can be found at http://www.epa.gov/ttn/atw/nata/roy/page16.html.

Noncancer health effects can result from chronic,[14] subchronic,[15] or acute[16] inhalation exposures to air toxics, and include neurological, cardiovascular, liver, kidney, and respiratory effects as well as effects on the immune and reproductive systems. According to the 2005 NATA, about three-fourths of the U.S. population was exposed to an average chronic concentration of air toxics that has the potential for adverse noncancer respiratory health effects. Results from the 2005 NATA indicate that acrolein is the primary driver for noncancer respiratory risk.

Figure 4-5and Figure 46 depict the estimated census tract-level carcinogenic risk and noncancer respiratory hazard from the assessment. It is important to note that large reductions in HAP emissions may not necessarily translate into significant reductions in health risk because toxicity varies by pollutant, and exposures may or may not exceed levels of concern. For example, acetaldehyde mass emissions are more than double acrolein emissions on a national basis, according to EPA's 2005 National Emissions Inventory (NEI). However, the Integrated Risk Information System (IRIS) reference concentration (RfC) for acrolein is considerably lower than that for acetaldehyde, suggesting that acrolein could be potentially more toxic than acetaldehyde. 17 Thus, it is important to account for the toxicity and exposure, as well as the mass of the targeted emissions.

Due to methodology and data limitations, we were unable to estimate the benefits associated with the hazardous air pollutants that would be reduced as a result of these rules.. In a few previous analyses of the benefits of reductions in HAPs, EPA has quantified the benefits of potential reductions in the incidences of cancer and non-cancer risk (e.g., U.S. EPA, 1995). In those analyses, EPA relied on unit risk factors (URF) developed through risk assessment procedures.[18] These URFs are designed to be conservative, and as such, are more likely to represent the high end of the distribution of risk rather than a best or most likely estimate of risk. As the purpose of a benefit analysis is to describe the benefits most likely to occur from a

[14]Chronic exposure is defined in the glossary of the Integrated Risk Information (IRIS) database (http://www.epa.gov/iris) as repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).

[15]Defined in the IRIS database as repeated exposure by the oral, dermal, or inhalation route for more than 30 days, up to approximately 10% of the life span in humans (more than 30 days up to approximately 90 days in typically used laboratory animal species).

[16]Defined in the IRIS database as exposure by the oral, dermal, or inhalation route for 24 hours or less.

[17]Details on the derivation of IRIS values and available supporting documentation for individual chemicals (as well as chemical values comparisons) can be found at http://cfpub.epa.gov/ncea/iris/compare.cfm.

[18]The unit risk factor is a quantitative estimate of the carcinogenic potency of a pollutant, often expressed as the probability of contracting cancer from a 70-year lifetime continuous exposure to a concentration of one $\mu g/m^3$ of a pollutant.

reduction in pollution, use of high-end, conservative risk estimates would overestimate the benefits of the regulation. While we used high-end risk estimates in past analyses, advice from the EPA's Science Advisory Board (SAB) recommended that we avoid using high-end estimates

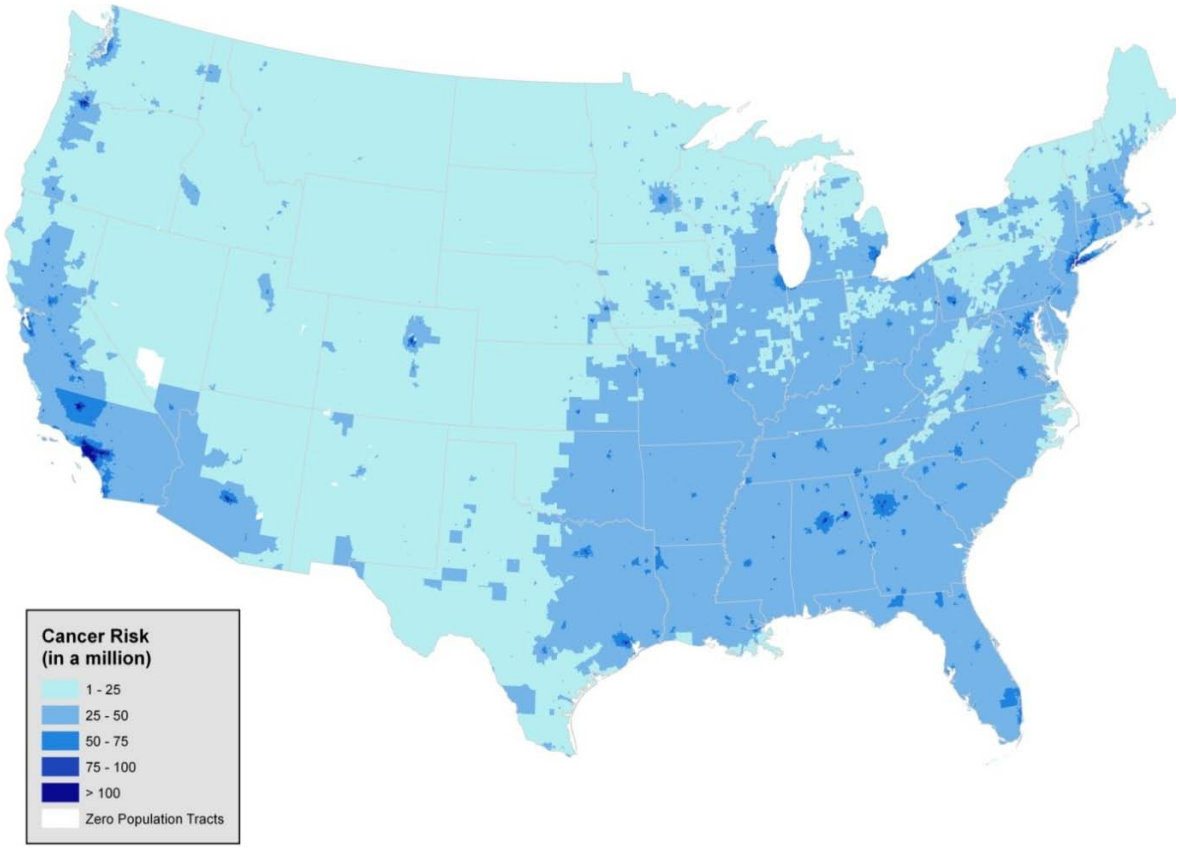

Figure 4-5. Estimated Chronic Census Tract Carcinogenic Risk from HAP Exposure from Outdoor Sources (2005 NATA)

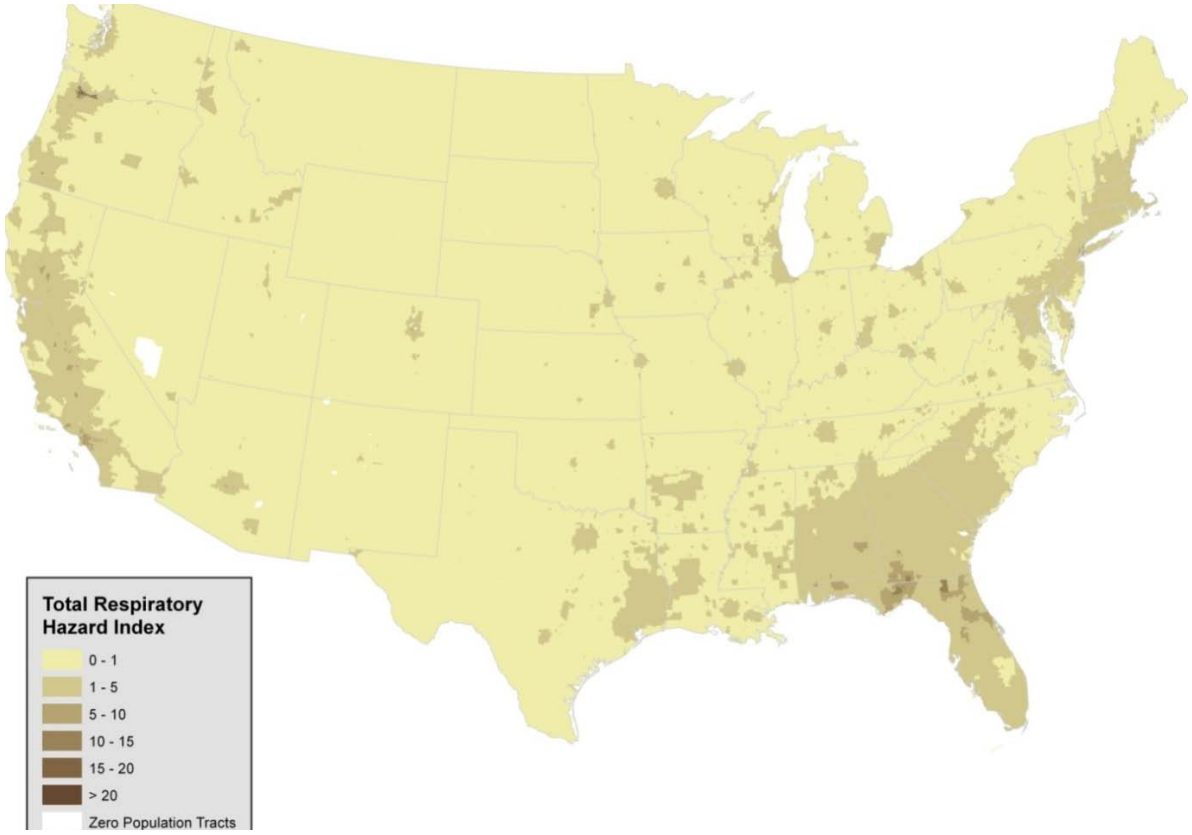

Figure 4-6. Estimated Chronic Census Tract Noncancer (Respiratory) Risk from HAP Exposure from Outdoor Sources (2005 NATA)

in benefit analyses (U.S. EPA-SAB, 2002). Since this time, EPA has continued to develop better methods for analyzing the benefits of reductions in HAPs.

As part of the second prospective analysis of the benefits and costs of the Clean Air Act (U.S. EPA, 2011a), EPA conducted a case study analysis of the health effects associated with reducing exposure to benzene in Houston from implementation of the Clean Air Act (IEc, 2009). While reviewing the draft report, EPA's Advisory Council on Clean Air Compliance Analysis concluded that "the challenges for assessing progress in health improvement as a result of reductions in emissions of hazardous air pollutants (HAPs) are daunting...due to a lack of exposure-response functions, uncertainties in emissions inventories and background levels, the difficulty of extrapolating risk estimates to low doses and the challenges of tracking health progress for diseases, such as cancer, that have long latency periods" (U.S. EPA-SAB, 2008).

In 2009, EPA convened a workshop to address the inherent complexities, limitations, and uncertainties in current methods to quantify the benefits of reducing HAPs. Recommendations from this workshop included identifying research priorities, focusing on susceptible and vulnerable populations, and improving dose-response relationships (Gwinn et al., 2011).

In summary, monetization of the benefits of reductions in cancer incidences requires several important inputs, including central estimates of cancer risks, estimates of exposure to carcinogenic HAPs, and estimates of the value of an avoided case of cancer (fatal and non-fatal). Due to methodology and data limitations, we did not attempt to monetize the health benefits of reductions in HAPs in this analysis. Instead, we provide a qualitative analysis of the health effects associated with the HAPs anticipated to be reduced by these rules and we summarize the results of the residual risk assessment for the Risk and Technology Review (RTR). EPA remains committed to improving methods for estimating HAP benefits by continuing to explore additional concepts of benefits, including changes in the distribution of risk.

Available emissions data show that several different HAPs are emitted from oil and natural gas operations, either from equipment leaks, processing, compressing, transmission and distribution, or storage tanks. Emissions of eight HAPs make up a large percentage the total HAP emissions by mass from the oil and gas sector: toluene, hexane, benzene, xylenes (mixed), ethylene glycol, methanol, ethyl benzene, and 2,2,4-trimethylpentane (U.S. EPA, 2011a). In the subsequent sections, we describe the health effects associated with the main HAPs of concern from the oil and natural gas sector: benzene, toluene, carbonyl sulfide, ethyl benzene, mixed xylenes, and n-hexane. These rules combined are anticipated to avoid or reduce 58,000 tons of HAPs per year. With the data available, it was not possible to estimate the tons of each individual HAP that would be reduced.

EPA conducted a residual risk assessment for the NESHAP rule (U.S. EPA, 2011c). The results for oil and gas production indicate that maximum lifetime individual cancer risks could be 30 in-a-million for existing sources before and after controls with a cancer incidence of 0.02 before and after controls. For existing natural gas transmission and storage, the maximum individual cancer risk decreases from 90-in-a-million before controls to 20-in-a-million after controls with a cancer incidence that decreases from 0.001 before controls to 0.0002 after controls. Benzene is the primary cancer risk driver. The results also indicate that significant noncancer impacts from existing sources are unlikely, especially after controls. EPA did not conduct a risk assessment for new sources affected by the NSPS. However, it is important to note that the magnitude of the HAP emissions avoided by new sources with the NSPS are more

than an order of magnitude higher than the HAP emissions reduced from existing sources with the NESHAP.

4.9.1 Hazards

Emissions data collected during development of this proposed rule show that HCl emissions represent the predominant HAP emitted by industrial boilers. Coal- and oil-fired EGUs emit lesser amounts of HF, chlorine, metals (As, Cd, Cr, Hg, Mn, Ni, and Pb), and organic HAP emissions. Although numerous organic HAP may be emitted from coal- and oil-fired EGUs, only a few account for essentially all the mass of organic HAP emissions. These organic HAP are formaldehyde, benzene, and acetaldehyde.

Exposure to high levels of these HAP is associated with a variety of adverse health effects. These adverse health effects include chronic health disorders (e.g., irritation of the lung, skin, and mucus membranes, effects on the central nervous system, and damage to the kidneys), and acute health disorders (e.g., lung irritation and congestion, alimentary effects such as nausea and vomiting, and effects on the kidney and central nervous system). We have classified three of the HAP as human carcinogens and five as probable human carcinogens. The following sections briefly discuss the main health effects information we have regarding the key HAPs emitted by EGUs.

4.9.1.1 Acetaldehyde

Acetaldehyde is classified in EPA's IRIS database as a probable human carcinogen, based on nasal tumors in rats, and is considered toxic by the inhalation, oral, and intravenous routes.[19] Acetaldehyde is reasonably anticipated to be a human carcinogen by the U.S. Department of Health and Human Services (DHHS) in the 11th Report on Carcinogens and is classified as possibly carcinogenic to humans (Group 2B) by the IARC.[20,21] The primary

[19]U.S. Environmental Protection Agency (U.S. EPA). 1991. Integrated Risk Information System File of Acetaldehyde. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at http://www.epa.gov/iris/subst/0290.htm.

[20]U.S. Department of Health and Human Services National Toxicology Program 11th Report on Carcinogens available at: http://ntp.niehs.nih.gov/go/16183.

[21]International Agency for Research on Cancer (IARC). 1999. Re-evaluation of some organic chemicals, hydrazine, and hydrogen peroxide. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemical to Humans, Vol 71. Lyon, France.

noncancer effects of exposure to acetaldehyde vapors include irritation of the eyes, skin, and respiratory tract.[22]

4.9.1.2 Arsenic

Arsenic, a naturally occurring element, is found throughout the environment and is considered toxic through the oral, inhalation and dermal routes. Acute (short-term) high-level inhalation exposure to As dust or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain, and gastrointestinal hemorrhage); central and peripheral nervous system disorders have occurred in workers acutely exposed to inorganic As. Chronic (long-term) inhalation exposure to inorganic As in humans is associated with irritation of the skin and mucous membranes. Chronic inhalation can also lead to conjunctivitis, irritation of the throat and respiratory tract and perforation of the nasal septum.[23] Chronic oral exposure has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, and liver or kidney damage in humans. Inorganic As exposure in humans, by the inhalation route, has been shown to be strongly associated with lung cancer, while ingestion of inorganic As in humans has been linked to a form of skin cancer and also to bladder, liver, and lung cancer. EPA has classified inorganic As as a Group A, human carcinogen.[24]

4.9.1.3 Benzene

The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia) by all routes of exposure, and concludes that exposure is associated with additional health effects, including genetic changes in both humans and animals and increased proliferation of bone marrow cells in mice.[25,26,27] EPA states in its IRIS database that data indicate a causal

[22]U.S. Environmental Protection Agency (U.S. EPA). 1991. Integrated Risk Information System File of Acetaldehyde. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at http://www.epa.gov/iris/subst/0290.htm.

[23]Agency for Toxic Substances and Disease Registry (ATSDR). Medical Management Guidelines for Arsenic. Atlanta, GA: U.S. Department of Health and Human Services. Available on the Internet at <http://www.atsdr.cdc.gov/mhmi/mmg168.html#bookmark02>

[24]U.S. Environmental Protection Agency (U.S. EPA). 1998. Integrated Risk Information System File for Arsenic. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at: http://www.epa.gov/iris/subst/0278.htm.

[25]U.S. Environmental Protection Agency (U.S. EPA). 2000. Integrated Risk Information System File for Benzene. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at: http://www.epa.gov/iris/subst/0276.htm.

[26]International Agency for Research on Cancer, IARC monographs on the evaluation of carcinogenic risk of chemicals to humans, Volume 29, Some industrial chemicals and dyestuffs, International Agency for Research on Cancer, World Health Organization, Lyon, France, p. 345–389, 1982.

[27]Irons, R.D.; Stillman, W.S.; Colagiovanni, D.B.; Henry, V.A. (1992) Synergistic action of the benzene metabolite hydroquinone on myelopoietic stimulating activity of granulocyte/macrophage colony-stimulating factor in vitro, Proc. Natl. Acad. Sci. 89:3691–3695.

relationship between benzene exposure and acute lymphocytic leukemia and suggest a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. The IARC has determined that benzene is a human carcinogen and the DHHS has characterized benzene as a known human carcinogen.[28,29]

A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with long-term exposure to benzene.[30,31]

4.9.1.4 Cadmium

Breathing air with lower levels of Cd over long periods of time (for years) results in a build-up of Cd in the kidney, and if sufficiently high, may result in kidney disease. Lung cancer has been found in some studies of workers exposed to Cd in the air and studies of rats that inhaled Cd. The U.S. DHHS has determined that Cd and Cd compounds are known human carcinogens. The IARC has determined that Cd is carcinogenic to humans. EPA has determined that Cd is a probable human carcinogen.[32]

4.9.1.5 Chlorine

The acute (short term) toxic effects of Cl_2 are primarily due to its corrosive properties. Chlorine is a strong oxidant that upon contact with water moist tissue (e.g., eyes, skin, and upper respiratory tract) can produce major tissue damage.[33] Chronic inhalation exposure to low concentrations of Cl_2 (1 to 10 parts per million, ppm) may cause eye and nasal irritation, sore throat, and coughing. Chronic exposure to Cl_2, usually in the workplace, has been reported to cause corrosion of the teeth. Inhalation of higher concentrations of Cl_2 gas (greater than 15 ppm) can rapidly lead to respiratory distress with airway constriction and accumulation of fluid in the lungs (pulmonary edema). Exposed individuals may have immediate onset of rapid breathing, blue discoloration of the skin, wheezing, rales or hemoptysis (coughing up blood or

[28]International Agency for Research on Cancer (IARC). 1987. Monographs on the evaluation of carcinogenic risk of chemicals to humans, Volume 29, Supplement 7, Some industrial chemicals and dyestuffs, World Health Organization, Lyon, France.

[29]U.S. Department of Health and Human Services National Toxicology Program 11th Report on Carcinogens available at: http://ntp.niehs.nih.gov/go/16183.

[30]Aksoy, M. (1989). Hematotoxicity and carcinogenicity of benzene. Environ. Health Perspect. 82: 193–197.

[31]Goldstein, B.D. (1988). Benzene toxicity. Occupational medicine. State of the Art Reviews. 3: 541–554.

[32]Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Public Health Statement for Cadmium. CAS# 1306-19-0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available on the Internet at <http://www.atsdr.cdc.gov/PHS/PHS.asp?id=46&tid=15>.

[33]Agency for Toxic Substances and Disease Registry (ATSDR). Medical Management Guidelines for Chlorine. Atlanta, GA: U.S. Department of Health and Human Services. http://www.atsdr.cdc.gov/mmg/mmg.asp?id=198&tid=36.

blood-stain sputum). Intoxication with high concentrations of Cl_2 may induce lung collapse. Exposure to Cl_2 can lead to reactive airways dysfunction syndrome (RADS), a chemical irritant-induced type of asthma. Dermal exposure to Cl_2 may cause irritation, burns, inflammation and blisters. EPA has not classified Cl_2 with respect to carcinogenicity.

4.9.1.6 Chromium

Chromium may be emitted in two forms, trivalent Cr (Cr^{+3}) or hexavalent Cr (Cr^{+6}). The respiratory tract is the major target organ for Cr^{+6} toxicity, for acute and chronic inhalation exposures. Shortness of breath, coughing, and wheezing have been reported from acute exposure to Cr^{+6}, while perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, and other respiratory effects have been noted from chronic exposures. Limited human studies suggest that Cr^{+6} inhalation exposure may be associated with complications during pregnancy and childbirth, but there are no supporting data from animal studies reporting reproductive effects from inhalation exposure to Cr^{+6}. Human and animal studies have clearly established the carcinogenic potential of Cr^{+6} by the inhalation route, resulting in an increased risk of lung cancer. EPA has classified Cr^{+6} as a Group A, human carcinogen. Trivalent Cr is less toxic than Cr^{+6}. The respiratory tract is also the major target organ for Cr^{+3} toxicity, similar to Cr^{+6}. EPA has not classified Cr^{+3} with respect to carcinogenicity.

4.9.1.7 Formaldehyde

Since 1987, EPA has classified formaldehyde as a probable human carcinogen based on evidence in humans and in rats, mice, hamsters, and monkeys.[34] EPA is currently reviewing recently published epidemiological data. After reviewing the currently available epidemiological evidence, the IARC (2006) characterized the human evidence for formaldehyde carcinogenicity as "sufficient," based upon the data on nasopharyngeal cancers; the epidemiologic evidence on leukemia was characterized as "strong."[35] EPA is reviewing the recent work cited above from the NCI and NIOSH, as well as the analysis by the CIIT Centers for Health Research and other studies, as part of a reassessment of the human hazard and dose-response associated with formaldehyde.

Formaldehyde exposure also causes a range of noncancer health effects, including irritation of the eyes (burning and watering of the eyes), nose and throat. Effects from repeated exposure in humans include respiratory tract irritation, chronic bronchitis and nasal epithelial

[34]U.S. EPA. 1987. Assessment of Health Risks to Garment Workers and Certain Home Residents from Exposure to Formaldehyde, Office of Pesticides and Toxic Substances, April 1987.

[35]International Agency for Research on Cancer (2006) Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxypropan-2-ol. Monographs Volume 88. World Health Organization, Lyon, France.

lesions such as metaplasia and loss of cilia. Animal studies suggest that formaldehyde may also cause airway inflammation—including eosinophil infiltration into the airways. There are several studies that suggest that formaldehyde may increase the risk of asthma—particularly in the young.[36,37]

4.9.1.8 Hydrogen Chloride

Hydrogen chloride is a corrosive gas that can cause irritation of the mucous membranes of the nose, throat, and respiratory tract. Brief exposure to 35 ppm causes throat irritation, and levels of 50 to 100 ppm are barely tolerable for 1 hour.[38] The greatest impact is on the upper respiratory tract; exposure to high concentrations can rapidly lead to swelling and spasm of the throat and suffocation. Most seriously exposed persons have immediate onset of rapid breathing, blue coloring of the skin, and narrowing of the bronchioles. Exposure to HCl can lead to RADS, a chemically- or irritant-induced type of asthma. Children may be more vulnerable to corrosive agents than adults because of the relatively smaller diameter of their airways. Children may also be more vulnerable to gas exposure because of increased minute ventilation per kg and failure to evacuate an area promptly when exposed. Hydrogen chloride has not been classified for carcinogenic effects.[39]

4.9.1.9 Hydrogen Fluoride

Acute (short-term) inhalation exposure to gaseous HF can cause severe respiratory damage in humans, including severe irritation and pulmonary edema. Chronic (long-term) oral exposure to fluoride at low levels has a beneficial effect of dental cavity prevention and may also be useful for the treatment of osteoporosis. Exposure to higher levels of fluoride may

[36]Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for Formaldehyde. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/toxprofiles/tp111.html

[37]WHO (2002) Concise International Chemical Assessment Document 40: Formaldehyde. Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organization, and the World Health Organization, and produced within the framework of the Inter-Organization Programme for the Sound Management of Chemicals. Geneva.

[38]Agency for Toxic Substances and Disease Registry (ATSDR). Medical Management Guidelines for Hydrogen Chloride. Atlanta, GA: U.S. Department of Health and Human Services. Available online at http://www.atsdr.cdc.gov/mmg/mmg.asp?id=758&tid=147#bookmark02.

[39]U.S. Environmental Protection Agency (U.S. EPA). 1995. Integrated Risk Information System File of Hydrogen Chloride. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at .http://www.epa.gov/iris/subst/0396.htm.

cause dental fluorosis. One study reported menstrual irregularities in women occupationally exposed to fluoride via inhalation. The EPA has not classified HF for carcinogenicity[40].

4.9.1.10 Lead

The main target for Pb toxicity is the nervous system, both in adults and children. Long-term exposure of adults to Pb at work has resulted in decreased performance in some tests that measure functions of the nervous system. Lead exposure may also cause weakness in fingers, wrists, or ankles. Lead exposure also causes small increases in blood pressure, particularly in middle-aged and older people. Lead exposure may also cause anemia.

Children are more sensitive to the health effects of Pb than adults. No safe blood Pb level in children has been determined. At lower levels of exposure, Pb can affect a child's mental and physical growth. Fetuses exposed to Pb in the womb may be born prematurely and have lower weights at birth. Exposure in the womb, in infancy, or in early childhood also may slow mental development and cause lower intelligence later in childhood. There is evidence that these effects may persist beyond childhood.[41]

There are insufficient data from epidemiologic studies alone to conclude that Pb causes cancer (is carcinogenic) in humans. The DHHS has determined that Pb and Pb compounds are reasonably anticipated to be human carcinogens based on limited evidence from studies in humans and sufficient evidence from animal studies, and the EPA has determined that Pb is a probable human carcinogen.

4.9.1.11 Manganese

Health effects in humans have been associated with both deficiencies and excess intakes of Mn. Chronic exposure to high levels of Mn by inhalation in humans results primarily in central nervous system effects. Visual reaction time, hand steadiness, and eye-hand coordination were affected in chronically-exposed workers. Manganism, characterized by feelings of weakness and lethargy, tremors, a masklike face, and psychological disturbances, may result from chronic exposure to higher levels. Impotence and loss of libido have been

[40]U.S. Environmental Protection Agency. Health Issue Assessment: Summary Review of Health Effects Associated with Hydrogen Fluoride and Related Compounds. EPA/600/8-89/002F. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH. 1989.

[41]Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Public Health Statement for Lead. CAS#: 7439-92-1. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available on the Internet at < http://www.atsdr.cdc.gov/ToxProfiles/phs13.html>.

noted in male workers afflicted with manganism attributed to inhalation exposures. The EPA has classified Mn in Group D, not classifiable as to carcinogenicity in humans.[42]

4.9.1.12 Nickel

Respiratory effects have been reported in humans from inhalation exposure to Ni. No information is available regarding the reproductive or developmental effects of Ni in humans, but animal studies have reported such effects. Human and animal studies have reported an increased risk of lung and nasal cancers from exposure to Ni refinery dusts and nickel subsulfide. The EPA has classified nickel subsulfide as a human carcinogen and nickel carbonyl as a probable human carcinogen.[43,44] The IARC has classified Ni compounds as carcinogenic to humans.[45]

4.9.1.13 Selenium

Acute exposure to elemental Se, hydrogen selenide, and selenium dioxide (SeO_2) by inhalation results primarily in respiratory effects, such as irritation of the mucous membranes, pulmonary edema, severe bronchitis, and bronchial pneumonia. One Se compound, selenium sulfide, is carcinogenic in animals exposed orally. EPA has classified elemental Se as a Group D, not classifiable as to human carcinogenicity, and selenium sulfide as a Group B2, probable human carcinogen.

4.10 References

Adams, Evan M., and Frederick, Peter C. Effects of methylmercury and spatial complexity on foraging behavior and foraging efficiency in juvenile white ibises (Eudocimus albus). Environmental Toxicology and Chemistry. Vol 27, No. 8, 2008.

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for Mercury. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

Amorim, M.I.M., D. Mergler, M.O. Bahia, H. Dubeau, D. Miranda, J. Lebel, R.R. Burbano, and M. Lucotte. 2000. Cytogenetic damage related to low levels of methyl mercury contamination in the Brazilian Amazon. An. Acad. Bras. Ciênc. 72(4): 497-507.

[42]U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) on Manganese. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.

[43]U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) on Nickel Subsulfide. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.

[44]U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) on Nickel Carbonyl. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.

[45]Nickel (IARC Summary & Evaluation , Volume 49, 1990), http://www.inchem.org/documents/iarc/vol49/nickel.html

Amyot, M., Lean, D.R.S., Poissant, L. and Doyon, M.-R., 2000. Distribution and transformation of elemental mercury in the St. Lawrence River and Lake Ontario. Canadian Journal of Fisheries and Aquatic Sciences, 57 (Suppl. 1): 155-163.

ATSDR (1999). *Toxicological Profile for Mercury*. Agency for Toxic Substances and Disease Registry.

Axelrad, D. A.; Bellinger, D. C.; Ryan, L. M.; Woodruff, T. J. (2007). Dose-response relationship of prenatal mercury exposure and IQ: an integrative analysis of epidemiologic data. *Environmental Health Perspectives*. 2007, 115, 609–615.

Babiarz, C.L. et al., 2001. Partitioning of total mercury and methylmercury to the colloidal phase in freshwaters. Environmental Science and Technology, 35(24): 4773-4782.

Barkay, T., Miller, S.M. and Summers, A.O., 2003. Bacterial mercury resistance from atoms to ecosystems. FEMS Microbiology Reviews, 27: 355-384.

Benoit, J.M., Gilmour, C.C. and Mason, R.P., 2001. The influence of sulfide on solid-phase mercury bioavailability for methylation by pure cultures of Desulfobulbus propionicus (1pr3). Environmental Science and Technology, 35(1): 127-132.

Benoit, J.M., Gilmour, C.C., Heyes, A., Mason, R.P. and Miller, C., 2003. Geochemical and Biological Controls over Methylmercury Production and Degradation in Aquatic Systems, Biogeochemistry of Environmentally Important Trace Metals. ACS Symposium Series 835.

Benoit, J.M., Gilmour, C.C., Mason, R.P. and Heyes, A., 1999a. Sulfide controls on mercury speciation and bioavailability to methylating bacteria in sediment pore waters. Environmental Science and Technology, 33(6): 951-957.

Benoit, J.M., Gilmour, C.C., Mason, R.P., Riedel, G.S. and Reidel, G.F., 1998. Behavior of mercury in the Patuxent River estuary. Biogeochemistry, 40: 249-265.

Benoit, J.M., Mason, R.P. and Gilmour, C.C., 1999b. Estimation of mercury-sulfide speciation in sediment pore waters using octanol-water partitioning and implications for availability to methylating bacteria. Environmental Toxicology and Chemistry, 18(10): 2138-2141.

Bergeron, CM., Bodinof, CM., Unrine, JM., Hopkins, WA. (2010a) Mercury accumulation along a contamination gradient and nondestructive indices of bioaccumulation in amphibians. Environmental Toxicology and Chemistry 29(4), 980-988.

Bergeron, CM., Bodinof, CM., Unrine, JM., Hopkins, WA. (2010b) Bioaccumulation and maternal transfer of mercury and selenium in amphibians. Environmental Toxicology and Chemistry 29(4), 989-997.

Beyers, D.W., Rice, J.A. and Clements, W.H., 1999. Evaluating biological significance of chemical exposure to fish using a bioenergetics-based stressor-response model. Canadian Journal of Fisheries and Aquatic Sciences, 56: 823-829.

Bigham, G.N. and Vandal, G.M., 1994. A drainage basin perspective of mercury transport and bioaccumulation: Onondaga Lake, New York, Twelfth International Neurotoxicology Conference, Hot Springs, Arkansas USA.

Bloom, N.S., 1992. On the chemical form of mercury in edible fish and marine invertebrate tissue. Canadian Journal of Fisheries and Aquatic Sciences, 49: 1010-1017.

Branfireun, B. and Roulet, N., 2002. Controls on the fate and transport of methylmercury in a boreal headwater catchment, northwestern Ontario, Canada. Hydrology and Earth System Sciences, 6(4): 785-794.

Brasso, Rebecka L., and Cristol, Daniel A. Effects of mercury exposure in the reproductive success of tree swallows (Tachycineta bicolor). Ecotoxicology. 17:133-141, 2008.

Budtz-Jorgensen E, Grandjean P, Jorgensen P, Weihe P, Keiding N (2004a). Association between mercury concentrations in blood and hair in methylmercury-exposed subjects at different ages. *Environmental Research,* 95(3):385-93.

Budtz-Jorgensen E, Keiding N, Grandjean P (2004b). Effects of exposure imprecision on estimation of the benchmark dose. *Risk Analysis, 2*4(6):1689-96.

Budtz-Jorgensen, E.; Grandjean, P.; Weihe, P. (2007). Separation of risks and benefits of 16 seafood intake. *Environmental Health Perspectives*. Vol. 115, 323-327.

Burger, J. (2002). Daily consumption of wild fish and game: Exposures of high end recreationalists, *International Journal of Environmental Health Research*, 12:4, p. 343-354.

Burgess, Neil M., and Meyer, Michael W. Methylmercury exposure associated with reduced productivity in common loons. Ecotoxicology. 17:83-91, 2008.

Carpi, A. and Lindberg, S.E., 1997. Sunlight-mediated emission of elemental mercury from soil amended with municipal sewage sludge. Environmental Science and Technology, 31(7): 2085-2091.

Compeau, G.C. and Bartha, R., 1987. Effect of salinity on mercury-methylating activity of sulfate reducing bacteria in estuarine sediments. Applied and Environmental Microbiology, 53: 261-265.

Connelly, N.A., B.A. Knuth, and T.L. Brown. 1996. “Sportfish Consumption Patterns of Lake Ontario Anglers and the Relationship to Health Advisories.” *North American Journal of Fisheries Management* 16:90-101.

Craig, P.J. and Bartlett, P.D., 1978. The role of hydrogen sulphide in environmental transport of mercury. Nature, 275: 635-637.

Craig, P.J. and Moreton, P.A., 1986. Total mercury, methyl mercury and sulphide levels in British estuarine sediments-III. Water Research, 20(9): 1111-1118.

Cristol D. A., Brasso R. L., Condon A. M., Fovargue R. E., Friedman S. L., Hallinger K. K., Monroe A. P., White A. E. (2008) The movement of aquatic mercury through terrestrial food webs. Science 320, 335–335.

Crump KS, Kjellstrom T, Shipp AM, Silvers A, Stewart A (1998). Influence of prenatal mercury exposure upon scholastic and psychological test performance: Benchmark analysis of a New Zealand cohort. *Risk Analysis*, 18:701-713.

Crump, Kate L., and Trudeau, Vance L. Mercury-induced reproductive impairment in fish. Environmental Toxicology and Chemistry. Vol. 28, No. 5, 2009.

Daniels, J.L., Longnecker,M.P., Rowland, A.S., et al., (2004). Fish intake during pregnancy and early cognitive development of offspring. *Epidemiology* 15, 394–402.

Davidson PW, Myers GJ, Cox C, Axtell C, Shamlaye C, Sloane-Reeves J, Cernichiari E, Needham L, Choi A, Wang Y, Berlin M, Clarkson TW (1998). Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment: outcomes at 66 months of age in the Seychelles Child Development Study. *Journal of the American Medical Association,* 280(8):701-7.

Davidson, P.W., G. Myers, C.C. Cox,C.F. Shamlaye, D.O.Marsh, M.A.Tanner, M. Berlin, J. Sloane-Reeves, E. Chernichiari, , O. Choisy, A. Choi and T.W. Clarkson. 1995. Longitudinal neurodevelopment study of Seychellois children following in utero exposure to methylemrcury form maternal fish ingestion: outcomes at 19 and 29 months. NeuroToxicology 16:677-688.

Davidson, P.W., Strain, J.J., Myers, G.J., et al., (2008). Neurodevelopmental effects of maternal nutritional status and exposure to methylmercury from eating fish during pregnancy. *NeuroToxicology* 29, 767–775.

Dellinger, JA (2004). Exposure assessment and initial intervention regarding fish consumption of tribal members in the Upper Great Lakes Region in the United States. *Environmental Research* 95 (2004) p. 325-340.

Dominici F, Samet JM, Zeger SL (2000). Combining evidence on air pollution and daily mortality from the 20 largest US cities: a hierarchical modeling strategy. *Journal of the Royal Statistical Society A,* 163:263-284.

Doyon, J.-F., Schetagne, R. and Verdon, R., 1998. Different mercury bioaccumulation rates between sympatric populations of dwarf and normal lake whitefish (Coregonus clupeaformis) in the La Grande complex watershed, James Bay, Quebec. Biogeochemistry, 40: 203-216.

Drevnick, P.E., D.E. Canfield, P.R. Gorski, A.L.C. Shinneman, D.R. Engstrom, D.C.G. Muir, G.R. Smith, P.J. Garrison, L.B. Cleckner, J.P. Hurley, R.B. Noble, R.R. Otter, and J.T. Oris. 2007. Deposition and cycling of sulfur controls mercury accumulation in Isle Royale fish. Environmental Science and Technology 41(21):7266–7272.

Driscoll, C.T. et al., 1995. The role of dissolved organic carbon in the chemistry and bioavailability of mercury in remote Adirondack lakes. Water, Air, Soil Pollution, 80: 499-508.

Dunstan, J.A., Simmer, K., Dixon, G., et al., (2006). Cognitive assessment of children at age 2.5 years after maternal fish oil supplementation in pregnancy: a randomized controlled trial. Archives of Diseases in Childhood. *Fetal Neonatal Ed*; December 21, 2006.

Ebert, E., N. Harrington, K. Boyle, J. Knight, J. and R. Keenan. 1994. "Estimating Consumption of Freshwater Fish among Maine Anglers." *North American Journal of Fisheries Management* 13:737-745.

EPA, 1997. U.S. Environmental Protection Agency, Volume I - General Factors Exposure Factors Handbook Update to Exposure Factors Handbook, EPA/600/8-89/043—May 1989, EPA/600/P-95/002Fa, August 1997.

Ericksen, J. A., Gustin, M. S., Schorran, D. E., Johnson, D. W., Lindberg, S. E., & Coleman, J. S. (2003). Accumulation of atmospheric mercury in forest foliage. Atmospheric Environment, 37(12), 1613-1622.

Evers, D.C., 2006. Status assessment and conservation plan for the common loon (Gavia immer) in North America. U.S. Fish and Wildlife Service, Hadley, MA, USA.

Evers, David C., Savoy, Lucas J., DeSorbo, Christopher R., Yates, David E., Hanson, William, Taylor, Kate M., Siegel, Lori S., Cooley, John H. Jr., Bank, Michael S., Major, Andrew, Munney, Kenneth, Mower, Barry F., Vogel, Harry S., Schoch, Nina, Pokras, Mark, Goodale, Morgan W., Fair, Jeff. Adverse effects from environmental mercury loads on breeding common loons. Ecotoxicology. 17:69-81, 2008.

Frederick, Peter, and Jayasena, Nilmini. Altered pairing behavior and reproductive success in white ibises exposed to environmentally relevant concentrations of methylmercury. Proceedings of The Royal Society B. doi: 10-1098, 2010.

Frescholtz, T. and Gustin, M.S., 2004. Soil and foliar mercury emission as a function of soil concentration. Water, Air, and Soil Pollution, 155: 223-237.

Gbundgo-Tugbawa and Driscoll, 1998. Application of the regional mercury cycling model (RMCM) to predict the fate and remediation of mercury in Onondaga Lake, New York. Water, Air, and Soil Pollution, 105: 417-426.

Gilmour, C.C. and Henry, E.A., 1991. Mercury methylation in aquatic systems affected by acid deposition. Environmental Pollution, 71: 131-169.

Gilmour, C.C. et al., 1998. Methylmercury concentrations and production rates across a trophic gradient in the northern Everglades. Biogeochemistry, 40: 327-345.

Gobas, F.A.P.C., Pasternak, J.P., Lien, K. and Duncan, R.K., 1998. Development and field validation of a multimedia exposure model for waste load allocation in aquatic ecosystems: application to 2,3,7,8-tetrachloro-p-dioxin and 2,3,7,8-tetrachlorodibenzofuran in the Fraser River watershed. Environmental Science and Technology, 32: 2442-2449.

Gobas, F.A.P.C., Z'Graggen, M.N. and Zhang, X., 1995. Time response of the Lake Ontario ecosystem to virtual elimination of PCBs. Environmental Science and Technology, 29(8): 2038-2046.

Gorissen, Leen, Snoeijs, Tinne, Van Duyse, Els, and Eens, Marcel. Heavy metal pollution affects dawn singing behavior in a small passerine bird. Oecologia. 145: 540-509, 2005.

Grandjean P, Weihe P, White R, Debes F, Araki S, Yokiyama K, Murata K, Sorensen N, Dahl R, Jorgensen P. Cognitive deficit in 7-year-old children with prenatal exposure to Methylmercury. *Neurotoxicol Teratol* . 1997. 19:(6)417-428.

Grandjean, Phillippe, Esben Budtz-Jørgensen, Roberta F. White, Poul J. Jørgensen, Pal Weihe, Frodi Debes, and Niels Keding (1999). Methylmercury Exposure Biomarkers as Indicators of Neurotoxicity in Children Aged 7 Years. *American Journal of Epidemiology*. Vol. 150 (3): 301-305.

Great Lakes Sport Fish Advisory Task Force. September 1993. *Protocol for a Uniform Great Lakes Sport Fish Consumption Advisory*.

Greenfield, B.K., Hrabik, T.R., Harvey, C.J. and Carpenter, S.R., 2001. Predicting mercury levels in yellow perch: use of water chemistry, trophic ecology, and spatial traits. Canadian Journal Fisheries and Aquatic Sciences, 58: 1419–1429.

Grieb, T.M. et al., 1990. Factors affecting mercury accumulation in fish in the upper Michigan peninsula. Environmental Toxicology and Chemistry, 9: 919-930.

Grosse, Scott D., Kurt V Krueger, Mercy Mvundura (2009). Economic Productivity by Age and Sex: 2007 Estimates for the United States. Medical Care: July 2009 - Volume 47 - Issue 7_Supplement_1 - pp S94-S103, doi: 10.1097/MLR.0b013e31819c9571.

Gustin, M. et al., 2004. Application of controlled mesocosms for understanding mercury air-soil-plant exchange. Environmental Science and Technology, 38: 6044-6050.

Hallinger, Kelly K., Cornell, Kerri L., Brasso, Rebecka L., and Cristol, Daniel A. Mercury exposure and survival in free-living tree swallows (Tachycineta bicolor). Ecotoxicology. Doi: 10.1007/s10646-010-0554-4, 2010.

Harmon, S.M., J.K. King, J.B. Gladden, and L.A. Newman. 2007. Using sulfate-amended sediment slurry batch reactors to evaluate mercury methylation. Archives of Environmental Contamination and Toxicology 52:326–333.

Harris, R., 2007. Ecosystem Responses to Mercury: Indicators of Change. CFC Press ISBN: 97808493888927.

Harris, R. and Hutchison, D., 2003. Factors Affecting the Predicted Response of Fish Mercury Concentrations to Changes in Mercury Loading. 1005521, Electric Power Research Institute, Palo Alto, CA.

Harris, R., Gherini, S. and Hudson, R., 1996. Regional Mercury Cycling Model: A Model for Mercury Cycling in Lakes, R-MCM Version 1.0 Draft User Guide and Technical Reference, Electric Power Research Institute, Wisconsin Department of Natural Resources, Lafayette, California.

Harris., R. C., John W. M. Rudd, Marc Amyot, Christopher L. Babiarz, Ken G. Beaty, Paul J. Blanchfield, R. A. Bodaly, Brian A. Branfireun, Cynthia C. Gilmour, Jennifer A. Graydon, Andrew Heyes, Holger Hintelmann, James P. Hurley, Carol A. Kelly, David P. Krabbenhoft, Steve E. Lindberg, Robert P. Mason, Michael J. Paterson, Cheryl L. Podemski, Art Robinson, Ken A. Sandilands, George R. Southworth, Vincent L. St. Louis, and Michael T. TateRudd, J. W. M., Amyot M., et al., Whole-Ecosystem study Shows Rapid Fish-Mercury Response to Changes in Mercury Deposition. Proceedings of the National Academy of Sciences Early Edition, PNAS 2007 104 (42) pp. 16586-16591; (published ahead of print September 27, 2007).

Hastie, T.J., Tibshirani, R., (1993). Varying coefficient models. *Journal of the Royal Statistical Society*: Series B 58, 379–396.

Hawley, Dana M., Hallinger, Kelly K., Cristol, Daniel A. Compromised immune competence in free-living tree swallows exposed to mercury. Ecotoxicology. 18:499-503, 2009.

Helland, I.B., Smith, L., Saarem, K., et al., (2003). Maternal supplementation with very long chain n-3 fatty acids during pregnancy and lactation augments children's IQ at 4 years of age. *Pediatrics* 111, E39–E44.

Hintelmann, H. and Evans, R.D., 1997. Application of stable isotopes in environmental tracer studies - measurement of monomethylmercury by isotope dilution ICP-MS and detection of species transformation. Fresenius Journal of Analytical Chemistry, 358: 378-385.

Hintelmann, H. et al., 2002. Reactivity and mobility of new and old mercury deposition in a boreal forest ecosystem during the first year of the METAALICUS study. Environmental Science and Technology, 36: 5034-5040.

Hintelmann, H., Keppel-Jones, K. and Evans, R.D., 2000. Constants of mercury methylation and demethylation rates in sediments and comparison of tracer and ambient mercury availability. Environmental Toxicology and Chemistry, 19(9): 2204-2211.

Hoffman, David J., Henny, Charles J., Hill, Elwood F., Grover, Robert A., Kaiser, James L., Stebbins, Katherine R. Mercury and drought along the lower Carson River, Nevada: III. Effects on blood and organ biochemistry and histopathology of snowy egrets and black-crowned night-herons on Lahontan Reservoir, 2002-2006. Journal of Toxicology and Environmental Health, Part A. 72: 20, 1223-1241, 2009.

Hrabik, T.R., and C.J. Watras. 2002. Recent declines in mercury concentration in a freshwater fishery: isolating the effects of de-acidification and decreased atmospheric mercury deposition in Little Rock Lake. Science of the Total Environment 297:229–237.

Hurley, J. et al., 1995. Influences of watershed characteristics on mercury levels in Wisconsin rivers. Environmental Science and Technology, 29(7): 1867-1875.

Hurley, J.P., Cowell, S.E., Shafer, M.M. and Hughes, P.E., 1998. Tributary loading of mercury to Lake Michigan: Importance of seasonal events and phase partitioning. Science of the Total Environment, 213: 129-137.

Hutchinson, J.W. LaBaugh, R.G. Sayre, and S.E. Schwarzbach. 2007. USGS goals for the coming decade. Science 318:200–201.

International Agency for Research on Cancer (IARC). 1994. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans and their Supplements: Beryllium, Cadmium, Mercury, and Exposures in the Glass Manufacturing Industry. Vol. 58. Jalili, H.A., and A.H. Abbasi. 1961. Poisoning by ethyl mercury toluene sulphonanilide. Br. J. Indust. Med. 18(Oct.):303-308 (as cited in NRC 2000).

Jackson, T.A., 1998. Mercury in aquatic ecosystems. In: W.J. Langston and M.J. Bebianno (Editors), Metal Metabolism in Aquatic Environments. Chapman & Hall, London, pp. 77-158.

Jakus, P., M. McGuinness, and A. Krupnick. 2002. “The Benefits and Costs of Fish Consumption Advisories for Mercury.” Discussion Paper 02-55. Washington, DC: Resources for the Future.

Judge, M.P., Harel, O., Lammi-Keefe, C.J., et al., (2007). Maternal consumption of a docosahexaenoic acid-containing functional food during pregnancy: benefit for infant performance on problem-solving but not on recognition memory tasks at age 9 months. *American Journal of Clinical Nutrition* 85, 1572–1577.

Karlsson, T. and Skyllberg, U., 2003. Bonding of ppb levels of methyl mercury to reduced sulfur groups in soil organic matter. Environmental Science and Technology, 37: 4912-4918.

Kidd, K., Hesslein, R., Fudge, R. and Hallard, K., 1995. The influence of trophic level as measured by delta-N-15 on mercury concentrations in fresh-water organisms. Water, Air, and Soil Pollution, 80(1-4): 1011-1015.

et al.Kjellstrom, T., P. Kennedy, S. Wallis, A. Stewart, L. Friberg, B. Lind, P. Witherspoon, and C. Mantell. 1989. Physical and mental development of children with prenatal exposure to mercury from fish. Stage 2: Interviews and psychological tests at age 6. National Swedish Environmental Protection Board Report No. 3642.

Knights, D. C, Sunderland, E. M., Barber, M. C., Johnston J. M., and Ambrose, R. B., Application of Ecosystem-Scale Fate and Bioaccumulation Models to Predict Fish Mercury Response Times to Changes in Atmospheric Deposition. Environmental Toxicology and Chemistry, Vol 28, No. 4., pp. 881-893.

Lalonde, J., Amyot, M., Kraepiel, A. and Morel, F., 2001. Photooxidation of Hg(0) in artificial and natural waters. Environmental Science and Technology, 35: 1367-1372.

Landis, M. and Keeler, G., 2002. Atmospheric mercury deposition to Lake Michigan during the Lake Michigan mass balance study. Environmental Science and Technology, 36(21): 4518-4524.

Landis, M.S., Lynam, M. and Stevens, R.K., 2004. The Monitoring and Modeling of Mercury Species in Support of Local Regional and Global Modeling. In: N. Pirrone and K.R. Mahaffey (Editors), Dynamics of Mercury Pollution on Regional and Global Scales. Kluwer Academic Publishers, New York, NY.

Laporte, J.-M., Andres, S. and Mason, R.P., 2002. Effect of ligands and other metals on the uptake of mercury and methylmercury across the gills and the intestine of the blue crab (Callinectes sapidus). Comparative Biochemistry and Physiology Part C, 131: 185-196.

Lawrence, A.L. and Mason, R.P., 2001. Factors controlling the bioaccumulation of mercury and methylmercury by the estuarine amphipod Leptocheirus plumulosus. Environmental Pollution, 111: 217-231.

Lawson, N.M. and Mason, R.P., 1998. Accumulation of mercury in estuarine food chains. Biogeochemistry, 40: 235-247.

Leaner, J.J. and Mason, R.P., 2002. Factors controlling the bioavailability of ingested methylmercury to channel catfish and atlantic sturgeon. Environmental Science and Technology, 36: 5124-5129.

Lin, C.J. and Pehkonen, S.O., 1999. The chemistry of atmospheric mercury: a review. Atmospheric Environment, 33: 2067-2079.

Lindberg, S.E. and Stratton, J.E., 1998. Atmospheric mercury speciation: concentrations and behavior of reactive gaseous mercury in ambient air. Environmental Science and Technology, 32(1): 49-57.

Lynch, Miranda L., Li-Shan Huang, Christopher Cox b, J.J. Strain, Gary J. Myers, Maxine P. Bonham, Conrad F. Shamlaye, Abbie Stokes-Riner, Julie M.W. Wallace, Emeir M. Duffy, Thomas W. Clarkson, Philip W. Davidson, (2010). Varying coefficient function models to explore interactions between maternal nutritional status and prenatal methylmercury toxicity in the Seychelles Child Development Nutrition Study. *Environmental Research*, doi:10.1016/j.envres.2010.09.005.

Marsh, D.O., T.W. Clarkson, C. Cox, et al. 1987. Fetal methylmercury poisoning: relationship between concentration in single strands of maternal-hair and child effects. Arch. Neurol. 44:1017-1022. (as cited in EPA 2002 IRIS documentation.)

Mason, R.P. et al., 1999. Mercury in the Chesapeake Bay. Marine Chemistry, 65: 77–96.

Mason, R.P., Morel, F.M.M. and Hemond, H.F., 1995. The role of microorganisms in elemental mercury formation in natural waters. Water, Air, and Soil Pollution, 80: 775-787.

McIntrye, J.W., and Evers, D.C.,(eds)2000. Loons: old history and new finding. Proceedings of a Symposium from the 1997 meeting, American Ornithologists' Union. North American Loon Fund, 15 August 1997, Holderness, NH, USA.

McIntyre, J.W., Barr, J.F. 1997Common Loon (Gavia immer) *in*: Pool A, Gill F (eds) The Birds of North America. Academy of Natural Sciences, Philadelphia, PA, 313

Mierle, G. and Ingram, R., 1991. The role of humic substance in the mobilization of mercury from watersheds. Water, Air, and Soil Pollution, 56: 349-357.

Mitro, Matthew G., Evers, David C., Meyer, Michael W., and Piper, Walter H. Common loon survival rates and mercury in New England and Wisconsin. Journal of Wildlife Management. 72(3): 665-673, 2008.

Morel, F., Kraepiel, A.M.L. and Amyot, M., 1998. The chemical cycle and bioaccumulation of mercury. Annual Reviews of Ecological Systems, 29: 543-566.

Morgan, J.N., M.R. Berry, and R.L. Graves. 1997. "Effects of Commonly Used Cooking Practices on Total Mercury Concentration in Fish and Their Impact on Exposure Assessments." *Journal of Exposure Analysis and Environmental Epidemiology* 7(1):119-133.

Moya, J. 2004. Overview of fish consumption rates in the United States. *Human and Ecological Risk Assessment: An International Journal* 10, no. 6: 1195–1211.

Munthe, J., Bodaly, R. A., Branfireun, B. A., Driscoll, C. T., Gilmour, C. C., Harris, R., et al. (2007). Recovery of Mercury-Contaminated Fisheries. Environmental Science & Technology, 36(1), 33-44.

Myers GJ, Davidson PW, Cox, C, Shamlaye CF, Palumbo D, Cernichiari E, Sloane-Reeves J, Wilding GE, Kost J, Huang LS, Clarkson TW (2003). Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study. *Lancet*, 361:1686-1692.

Myers, M.D., M.A. Ayers, J.S. Baron, P.R. Beauchemin, K.T. Gallagher, M.B. Goldhaber, D.R.

National Research Council (NRC). 2000. Toxicological Effects of Methylmercury. Committee on the Toxicological Effects of Methylmercury, Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council. National Academy Press, Washington, DC.

Neisser U, Boodoo G, Bouchard TJ, et al. (1996). Intelligence: Knowns and unknowns. *American Psychologist,* 51:77-101.

NRC (2000). *Toxicological Effects of Methylmercury*. National Research Council. Washington, DC: National Academies Press.

Oken E., K.P. Kleinman, W.E. Berland, S.R. Simon, J.W. Rich-Edwards, and M.W. Gillman. 2003. "Decline in Fish Consumption Among Pregnant Women After a National Mercury Advisory." *Obstetrics and Gynecology* 102(2):346-351.

Rasmussen, P.E., 1994. Current methods of estimating atmospheric mercury fluxes in remote areas. Environmental Science and Technology, 28(13): 2233-2241.

Ravichandran, M., 2004. Interactions between mercury and dissolved organic matter - a review. Chemosphere, 55: 319-331.

Rea, A. W., Lindberg, S. E., Scherbatskoy, T., & Keeler, G. J. (2002). Mercury Accumulation in Foliage over Time in Two Northern Mixed-Hardwood Forests. Water, Air, & Soil Pollution, 133(1), 49-67.

Rice GE, Hammitt JK, Evans JS. (2010). A probabilistic characterization of the health benefits of reducing methyl mercury intake in the United States. *Environmental Science Technology*. 2010 Jul 1;44(13):5216-24

Rimmer, C. C., McFarland, K. P., Evers, D. C., Miller, E. K., Aubry, Y., Busby, D., et al. (2005). Mercury Concentrations in Bicknell's Thrush and Other Insectivorous Passerines in Montane Forests of Northeastern North America. Ecotoxicology, 14(1), 223-240.

Rolfhus, K. et al., 2003. Distribution and fluxes of total and methylmercury in Lake Superior. Environmental Science and Technology, 37(5): 865-872.

Ryan, LM (2005). Effects of Prenatal Methylmercury on Childhood IQ: A Synthesis of Three Studies. Report to the U.S. Environmental Protection Agency.

Salkever, D. 1995. "Updated Estimates of Earnings Benefits from Reduced Lead Exposure of Children to Environmental Lead." *Environmental Research* 70:1-6.

Schaefer, J. et al., 2004. Role of the bacterial organomercury lyase (MerB) in controlling methylmercury accumulation in mercury-contaminated natural waters. Environmental Science and Technology, 38: 4304-4311.

Scheuhammer, Anton M., Meyer Michael W., Sandheinrich, Mark B., and Murray, Michael W. Effects of environmental methylmercury on the health of wild birds, mammals, and fish. Ambio. Vol.36, No.1, 2007.

Scholtz, M.T., Heyst, B.J.V. and Schroeder, W.H., 2003. Modeling of mercury emissions from background soils. Science of the Total Environment, 304: 185-207.

Schroeder, W.H., Munthe, J. and Lindqvist, O., 1989. Cycling of mercury between water air and soil compartments of the environment. Water, Air, and Soil Pollution, 48: 337-347.

Schwartz, Joel (1994). Societal Benefits of Reducing Lead Exposure. *Environmental Research* 66, 105-124.

Seigneur, C., Jayaraghavan, K., Lohman, K., Karamchandani, P. and Scott, C., 2004. Global Source Attribution for Mercury Deposition in the United States. Environmental Science and Technology, 38: 555-569.

Sellers, P., Kelly, C.A., Rudd, J.W.M. and MacHutchon, A.R., 1996. Photodegradation of methylmercury in lakes. Nature, 380: 694.

Sepulveda, Maria S., Frederick, Peter C., Spalding, Marilyn G., and Williams, Gary E. Jr. Mercury contamination in free-ranging great egret nestlings (Ardea albus) from southern Florida, USA. Environmental Toxicology and Chemistry. Vol. 18, No.5, 1999.

Shilling, Fraser, Aubrey White, Lucas Lippert, Mark Lubell (2010). Contaminated fish consumption in California's Central Valley Delta. *Environmental Research* 110, p. 334-344.

Siciliano, S., O'Driscoll, N. and Lean, D., 2002. Microbial reduction and oxidation of mercury in freshwater lakes. Environmental Science and Technology, 36(14): 3064-3068.

St. Louis, V. et al., 2001. Importance of the forest canopy to fluxes of methyl mercury and total mercury to boreal ecosystems. Environmental Science and Technology, 35: 3089-3098.

Stern, AH, Smith AE (2003). An assessment of the cord blood:maternal blood methylmercury ratio: Implications for risk assessment. *Environmental Health Perspectives,* 111:1465-1470.

Strain, J.J., Davidson, P.W., Bonham, M.P., et al., (2008). Associations of maternal long-chain polyunsaturated fatty acids, methylmercury, and infant development in the Seychelles Child Development Nutrition Study. *NeuroToxicology* 29, 776–782.

Sunda, W.G. and Huntsman, S.A., 1998. Processes regulating cellular metal accumulation and physiological effects: Phytoplankton as model systems. Science of the Total Environment, 219: 165-181.

Sunderland, E.M. et al., 2004. Speciation and bioavailability of mercury in well-mixed estuarine sediments. Marine Chemistry, 90: 91-105.

Swartout, J., and G. Rice. 2000. "Uncertainty Analysis of the Estimated Ingestion Rates Used to Derive the Methylmercury Reference Dose." *Drug and Chemical Toxicology* 23(1):293-306. 11-41

Tseng, C.M., Amouroux, D., Abril, G. and Donard, O.F.X., 2001. Speciation of mercury in a fluid mud profile of a highly turbid macrotidal estuary (Gironde, France). Environmental Science and Technology, 35(13): 2627-2633.

U.S. Department of the Interior (DOI), Fish and Wildlife Service and U.S. Department of Commerce, Bureau of the Census. 1992. 1991 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation. Washington, DC: U.S. Government Printing Office.

U.S. Department of the Interior (DOI), Fish and Wildlife Service and U.S. Department of Commerce, Bureau of the Census. 1997. 1996 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation. Washington, DC: U.S. Government Printing Office.

U.S. Department of the Interior (DOI), Fish and Wildlife Service and U.S. Department of Commerce, Bureau of the Census. 2002. 2001 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation. Washington, DC: U.S. Government Printing Office.

U.S. Department of the Interior (DOI), Fish and Wildlife Service and U.S. Department of Commerce, Bureau of the Census. 2007. 2006 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation. Washington, DC: U.S. Government Printing Office.

U.S. Environmental Protection Agency (EPA), 1997. Mercury Study Report to Congress. EPA-452/R-97-005, Office of Air Quality Planning and Standards United States Environmental Protection Agency, Washington.

U.S. Environmental Protection Agency (EPA). 1997. Mercury Study Report to Congress. Volume V: Health Effects of Mercury and Mercury Compounds. EPA-452/R-97-007. U.S. EPA Office of Air Quality Planning and Standards, and Office of Research and Development.

U.S. Environmental Protection Agency (EPA) (1997). Volume I - General Factors Exposure Factors Handbook Update to Exposure Factors Handbook, EPA/600/8-89/043—May 1989, EPA/600/P-95/002Fa, August 1997.

U.S. Environmental Protection Agency (U.S. EPA). 1999. 1999 National Emission Inventory Documentation and Data—Final Version 3.0; Hazardous Air Pollutants Inventory—FinalNEI Version 3; HAPS Summary Files. (12 December 2006;*www.epa.gov/ttn/chief/net/1999inventory.html*)

U.S. Environmental Protection Agency (EPA), 2001a. Mercury Maps. A Quantitative Spatial Link Between Air Deposition and Fish Tissue. Final Report. EPA/823/R-01/009, USEPA, Washington, D.C.

U.S. Environmental Protection Agency (EPA), 2001b. Methylmercury fish tissue residue criterion, United States Environmental Protection Agency, Office of Water, 4304 EPA-823-F-01-001, January 2001, www.epa.gov/waterscience/criteria/methylmercury/factsheet.html.

U.S. Environmental Protection Agency (EPA) (2002). Mercury Neurotoxicity Workshop Notes. Washington, DC. November 4, 2002. http://www.epa.gov/ttn/ecas/regdata/Benefits/mercuryworkshop.pdf.

U.S. Environmental Protection Agency (EPA 2002). Mercury Neurotoxicity Workshop Notes; available at: www.epa.gov/ttn/ecas/benefits.html

U.S. Environmental Protection Agency (EPA). 2002 (date of most recent revision of on-line materials; website accessed January 2005). Integrated Risk Information System (IRIS). Methylmercury. U.S. EPA Office of Research and Development, National Center for Environmental Assessment. Oral RfD and inhalation RfC assessments last revised 7/27/2001. Carcinogenicity assessment last revised 5/1/1995. Available online at http://www.epa.gov/iris/subst/0073.htm

U.S. Environmental Protection Agency (U.S. EPA). 2005. *Regulatory Impact Analysis of the Final Clean Air Mercury Rule.* Office of Air Quality Planning and Standards, Research Triangle Park, NC., March; EPA report no. EPA-452/R-05-003. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/RIAs/mercury_ria_final.pdf

U.S. Environmental Protection Agency (EPA) (2005). Regulatory Impact Analysis of the Clean Air Mercury Rule, Research Triangle Park, NC. June 2008. EPA-452/R-05-003.

U.S EPA (Environmental Protection Agency). 2008. Integrated Science Assessment (ISA) for Oxides of Nitrogen and Sulfur—Ecological Criteria (Final Report). EPA/600/R-08/082F. U.S. Environmental Protection Agency, National Center for Environmental Assessment-RTP Division, Office of Research and Development, Research Triangle Park, N.C. Available at http://cfpub.epa.gov/ncea/cfm/recorddisplay.cfm?deid+201485.

U.S. Environmental Protection Agency (EPA) (2008). Proposed Lead NAAQS Regulatory Impact Analysis Office of Air Quality Planning and Standards, Research Triangle Park, NC. June 2008.

U.S. Environmental Protection Agency (EPA). 2008. Integrated Science Assessment (ISA) for Oxides of Nitrogen and Sulfur–Ecological Criteria (Final Report). EPA/600/R- 08/082F. U.S. Environmental Protection Agency, National Center for Environmental Assessment–RTP Division, Office of Research and Development, Research Triangle Park, NC. Available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=201485.

United States Department of Health and Human Services (DHHS), Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), Division of Vital Statistics, Natality public-use data 2003-2006, on CDC WONDER Online Database, March 2009. Accessed at http://wonder.cdc.gov/natality-current.html on Nov 3, 2010 4:44:23 PM

USDA Forest Service (1994). “National Survey on Recreation and the Environment: 1994-95.” Washington DC: USDA Forest Service.

Wada, H. and Cristol, D.A. and McNabb, F.M.A. and Hopkins, W.A. (2009) Suppressed adrenocortical responses and thyroid hormone levels in birds near a mercury-contaminated river. Environmental Science & Technology 43(15), 6031-6038.

Wada., H., Yates, DE., Evers, DC., Taylor, RJ., Hopkins, WA. (2010) Tissue mercury concentrations and adrenocortical responses of female big brown bats (Eptesicus fuscus) near a contaminated river. Ecotoxicology. 19(7), 1277-1284.

Watras, C.J. and Bloom, N.S., 1992. Mercury and methylmercury in individual zooplankton: implications for bioaccumulation. Limnol. Oceanogr., 37: 1313-1318.

Watras, C.J. et al., 1998. Bioaccumulation of mercury in pelagic freshwater food webs. Science of the Total Environment, 219(2-3): 183-208.

Wechsler D (1991). *WISC-III Manual*. San Antonio: The Psychological Corporation.

West, P.C., J.M. Fly, R. Marans, F. Larkin, and D. Rosenblatt. May 1993. *1991-92 Michigan Sport Anglers Fish Consumption study*. Prepared by the University of Michigan, School of Natural Resources for the Michigan Department of Natural Resources, Ann Arbor, MI. Technical Report No. 6.

West, P.C., M.J. Fly, R. Marans, and F. Larkin. 1989. *Michigan Sport Anglers Fish Consumption Survey. A report to the Michigan Toxic Substance Control Commission*. Michigan Department of Management and Budget Contract No. 87-20141.

Woods & Poole Economics, Inc. 2008. Population by Single Year of Age CD. CD-ROM. Woods & Poole Economics, Inc.

Wren, C.D. and MacCrimmon, H.R., 1986. Comparative bioaccumulation of mercury in two adjacent freshwater ecosystems. Water Research, 6: 763-769.

Yates, David E., Mayack, David T., Munney, Kenneth, Evers David C., Major, Andrew, Kaur, Taranjit, and Taylor, Robert J. Mercury levels in mink (Mustela vison) and river otter (Lonra canadensis) from northeastern North America. Ecotoxicology. 14, 263-274, 2005.

Zhang, H. and Lindberg, S.E., 2001. Sunlight and Iron(III)-Induced Photochemical Production of Dissolved Gaseous Mercury in Freshwater. Environmental Science and Technology, 35: 928-935.

APPENDIX 4A
ANALYSIS OF TRIP TRAVEL DISTANCE FOR RECREATIONAL FRESHWATER ANGLERS

As described in Section 3.7.7, the method used to estimate exposures to mercury in freshwater fish requires information about how far individuals typically travel for freshwater fishing. This appendix describes the data and methods used to analyze travel distance patterns by freshwater anglers, and it reports the results that were used to estimate exposures.

4A.1 Data

To conduct an analysis of trip travel distance for freshwater anglers, we used data from the NSRE 1994. As described previously, this 16,000-person survey elicited information on water-based recreation activities—specifically boating, fishing, swimming, and wildlife viewing—during the previous year. Respondents were asked about *their most recent trip* taken in each of the four categories. Of particular interest to this analysis is data concerning fishing trip characteristics for all respondents who fished in freshwater bodies during the previous year. Of the 3,220 respondents who had reported fishing, 2,482 visited either a lake, pond, river, or stream on their most recent trip.

The fishing module elicited location information about most recent fishing trip taken during the preceding 12 months. This trip was recorded as either a single- or multiday trip to a specific water body ("site") identified by the respondent. Subsequently, a series of questions were asked to gather location data on the specific site visited, including the site name, the state in which the site was located, and the name of the city or town nearest the site. To identify potential determinants of travel distance for a freshwater fishing trip, we analyzed the 2,384 available responses to the following survey question: "What was the one way travel distance, in miles from your home, to your destination on *site*?" Table C-1 presents summary statistics for travel distance, which are reported separately for single-day, multiday, and aggregated trips. As would be expected, median travel distance varied according to trip type, from 20 miles for a single-day trip to almost 140 miles for a multiday trip. Across both trip types, the average travel distance was slightly less than 100 miles.

4A.2 Analysis of Travel Distance Data

The influence of multiple demographic characteristics on travel distance was tested using multivariate regression analysis. Table C-2 reports descriptive statistics for the anglers included in this analysis. As indicated by the table, over 90 percent of the sample is white; males comprise a higher percentage of the sample (62 percent) than females. More than half

Table 4A-1. Reported Trip Travel Distance for Freshwater Anglers (Miles)

	N	Mina	P5	P25	P50	Mean	P75	P95	Max
All trip types	2,384	0	2	10	20	91.9	45	125	3,000
Single-day trips only	1,791	0	2	10	20	41	45	125	1,100
Multiday trips only	586	3	18	70	138	248.2	300	850	3,000

[a] Seven respondents reported traveling 0 miles for their most recent trip; all were described as single-day trips.

Note: Ninety-eight respondents who visited freshwater bodies on their most recent fishing trip did not report the travel distance.

Table 4A-2. Demographic Characteristics of Freshwater Anglers[a]

	N	Frequency	
Gender	2,267	62%	Male
Race	2,250	91%	White
		4%	Black
		2%	Hispanic
		2%	Other
Education	2,262	11%	Less than high school degree
		34%	High school degree/equivalent
		55%	Some college or more
Work status	2,263	75%	Employed
Geography	2,237	23%	Urban
		37%	Suburban
		41%	Rural
Region	2,205	13%	Northeast
		33%	South
		31%	Midwest
		23%	West

[a] In total, 2,384 respondents reported information on trip travel distance to a freshwater destination.

Note: Values may not add to 100 percent due to rounding.

the sample had completed at least some college and three-fourths of the sample reported being employed. The survey asked respondents to classify their place of residence as either rural, suburban, or urban. Approximately 40 percent described their area as rural, 37 percent as suburban, and 23 percent as urban. Respondents were assigned to a U.S. Census geographic region by matching their zip code to a corresponding state. The states were then aggregated to the appropriate Census region (http://www.census.gov/geo/www/us_regdiv.pdf). The majority of respondents resided in the South and Midwest, followed by the West and Northeast.

Table C-3 presents additional characteristics on the demographic distribution of the sample. The average age of respondents was 38 years, while household size averaged approximately three members, with less than one person under the age of six. Respondents' average weekly leisure time was 28 hours. However, this varied significantly across the sample, from zero to 168 hours. In the survey, family income is reported as a categorical variable, with respondents selecting the income range that reflected family income in the previous year. The midpoint of this range was taken to produce a continuous income variable. Subsequently, this value was converted to (2000$) using the consumer price index. Median (mean) income was estimated to be $57,325 ($66,496) annually.

Table 4A-3. Demographic Characteristics of Freshwater Anglers

	N	Mean	SD	Min	Max
Age	2,245	38.4	14.5	16	92
Household size	2,255	3.1	1.5	1	10
Persons $\leq$6 yrs	2,270	0.3	0.7	1	5
Persons $\geq$16 yrs	2,254	2.2	0.9	0	7
Weekly leisure time (hrs)	2,025	27.7	23.9	0	168
Family income (2,000$)	1,851	66,496	57,324	8,938	208,547

Multivariate regression analysis was used to identify determinants of travel distance to freshwater fishing sites. The dependent variable in this analysis was the miles traveled to the most recent freshwater fishing site. The explanatory variables included several demographic and geographic characteristics of the respondents.

Separate regressions were conducted for the full sample (1), single-day trips only (2), and multiday trips only (3). The results are reported in Table C-4. Family income was estimated

Table 4A-4. OLS Regression Results for Determinants of Reported Trip Travel Distance (Miles)

	(1) Full Sample (both single- and multiday trips)		(2) Single-Day Trips Only		(3) Multiday Trips Only	
Variable Description	**Coefficient**	**t-stat**	**Coefficient**	**t-stat**	**Coefficient**	**t-stat**
CONSTANT	0.6966	1.54	1.7954	3.89**	2.2493	3.26**
AGE	0.0044	1.83*	0.0011	0.44	0.001	0.28
GENDER	0.0572	0.83	0.0173	0.25	0.1446	1.39
EDUC	0.1729	2.48**	0.1552	2.21**	0.128	1.22
MINORITY	−0.0437	−0.36	0.0228	0.19	−0.1391	−0.76
FAMILY INCOME (log)	0.187	4.41**	0.0827	1.92*	0.1759	2.78**
URBAN	0.3491	3.95**	0.2799	3.12**	0.2121	1.62*
SUBURBAN	0.3422	4.48**	0.193	2.50**	0.4298	3.67**
NEAST	−0.0387	−0.36	−0.2549	−2.42**	0.1525	0.89
MIDWEST	0.3856	4.65**	0.1	1.21	0.4923	3.63**
WEST	0.6103	6.73**	0.3374	3.59**	0.3239	2.32**
	R^2 = 0.077		R^2 = 0.041		R^2 = 0.112	
	N = 1,798		N = 1,360		N = 434	

** = significant at 5 percent level.

* = significant at 10 percent level.

to have a positive and highly significant effect in all three models. Dummy variables for urban and suburban location were also found to have positive and highly significant effects in all models. These results suggest that wealthier anglers and those living in or near metropolitan areas tend to travel further to fishing sites, relative to less-wealthy anglers and those living in rural areas. In models (1) and (2) dummy variables for the Midwest and West regions also had positive and highly significant effects on trip travel distance, relative to the South region. The Northeast region did not have a statistically significant effect on distance traveled. Education was estimated to be positively and significantly related to distance traveled in the first and second models. (Note that the respondent's level of education, recorded in the survey as a categorical variable, was recoded as a continuous variable for the regression analysis.) Neither age, race, nor gender had significant effects (at a 5 percent level) on travel distance in any of the models.

4A.3 Summary Results Applied in the Population Centroid Approach

Given the high significance of geographic area and family income across the regressions, nonparametric results (frequency distributions) were generated for four mutually exclusive subgroups of respondents and five travel distance categories. The results are reported in Table C-5. Respondents were categorized into the four following groups:

- G1: family income ≥$50,000 (in 2000 dollars) and urban or suburban resident
  - (N = 452 for single-day trips)
  - (N = 649 for single- and multiday trips)
- G2: family income ≤$50,000 and urban or suburban resident
  - (N = 329 for single-day trips)
  - (N = 417 for single- and multiday trips)
- G3: family income ≥$50,000 and rural resident
  - (N = 295 for single-day trips
  - (N = 376 for single- and multiday trips)
- G4: family income ≤$50,000 and rural resident
  - (N = 309 for single-day trips)
  - (N = 386 for single- and multiday trips)

These categories were selected because they match categories that can be easily identified in Census data and because they split the sample into roughly similar group sizes. Travel distance was categorized into ranges reported in the first column of Table C-5. The results are consistent with those generated from the regression analysis. Among respondents on single-day trips, the number that traveled longer distances (greater than 100 miles) increased from the low-income rural cohort (5 percent) to the higher-income urban/suburban cohort (11 percent). The same pattern holds for those taking either a single- or multiday trip. The number traveling longer distances more than doubled, from 11 percent among low-income rural respondents to 27 percent among high-income urban/suburban respondents. These results indicate higher-income urban/suburban anglers travel greater distances to freshwater destinations than lower-income urban/suburban anglers and rural anglers.

As described in Section 3.7, the trip frequency estimates reported in Table C-5 for the full sample were used in the population centroid approach to weight exposures to mercury in

fish according to distance from the Census tract centroid, income levels in the tract, and whether the tract is predominantly rural or urban/suburban.

Table 4A-5. Travel Distance Frequencies by Demographic Group (Percentage in Each Distance Category)

Travel Distance (mi)	(G1) High-Income and Urban/Suburban Resident	(G2) Low-Income and Urban/Suburban Resident	(G3) High-Income and Rural Resident	(G4) Low-Income and Rural Resident
Single-day trips only (N = 1,385)				
N	(N = 452)	(N = 329)	(N = 295)	(N = 309)
Distance ≤10 mi	23%	32%	31%	34%
>10 mi to 20 mi	18%	23%	22%	24%
>20 mi to 50 mi	31%	20%	28%	26%
>50 mi to 100 mi	17%	19%	14%	11%
Distance >100 mi	11%	6%	5%	5%
Full sample (both single- and multiday trips) (N = 1,828)				
N	(N = 649)	(N = 417)	(N = 376)	(N = 386)
Distance <10 mi	16%	26%	24%	29%
>10 mi to 20 mi	13%	18%	18%	21%
>20 mi to 50 mi	24%	18%	25%	25%
>50 mi to 100 mi	19%	19%	16%	14%
Distance >100 mi	27%	18%	17%	11%

CHAPTER 5
HEALTH AND WELFARE CO-BENEFITS

Synopsis

Implementation of HAP emissions controls required by this rule is expected to have ancillary co-benefits, including lower overall ambient concentrations of SO_2, NO_2, $PM_{2.5}$ and ozone across the U.S. Pollutants such as SO_2, NO_x, and direct $PM_{2.5}$ contribute to ambient $PM_{2.5}$ levels in the atmosphere, and NOx contributes to ambient ozone concentrations. Furthermore, this rule is expected to reduce CO_2 emissions affecting climate change. These health and welfare co-benefits comprise a significant share of the total monetized benefits from this rule. This chapter provides estimates for this subset of the expected annual health and climate co-benefits of this rule in 2016.

Due to limits in available air quality modeling, the quantified co-benefits of this rule consist of only $PM_{2.5}$-related health co-benefits from reductions in SO_2 (a precursor to $PM_{2.5}$ formation) and direct $PM_{2.5}$ and climate co-benefits from reductions in CO_2. These co-benefits are estimated by applying a benefit-per–ton (BPT) approach described below to estimated reductions in SO_2 and direct $PM_{2.5}$ emissions reported in Chapter 3. The monetized co-benefits assessment omits several important categories of benefits, including health and ecological co-benefits from reducing exposure to ozone, ecosystem co-benefits for reducing nitrogen and sulfate deposition, and the direct health co-benefits from reducing exposure to ozone, SO_2 and NO_2. We describe these co-benefits qualitatively in Section 5.5.

We estimate the monetized health and climate co-benefits of MATS to be $37 billion to $90 billion at a 3% discount rate and $33 billion to $81 billion at a 7% discount rate in 2016, depending on the epidemiological function used to estimate reductions in premature mortality. All estimates are in 2007$.

5.1 Overview

The analysis in this chapter aims to characterize the co-benefits of the Mercury and Air Toxics Standards by answering two key questions:

1. What are the health effects of changes in ambient particulate matter ($PM_{2.5}$) resulting from reductions in directly-emitted $PM_{2.5}$ and SO_2?

2. What is the economic value of these effects?

Additionally, this chapter describes health effects that are not quantified for this rule, unquantified welfare effects, and visibility co-benefits.

In implementing these rules, emission controls may lead to reductions in ambient $PM_{2.5}$ below the National Ambient Air Quality Standards (NAAQS) for PM in some areas and assist other areas with attaining the PM NAAQS. Because the PM NAAQS RIAs also calculate PM benefits, there are important differences worth noting in the design and analytical objectives of each RIA. The NAAQS RIAs illustrate the potential costs and benefits of attaining a new air quality standard nationwide based on an array of emission control strategies for different sources. In short, NAAQS RIAs hypothesize, but do not predict, the control strategies that States may choose to enact when implementing a NAAQS. The setting of a NAAQS does not directly result in costs or benefits, and as such, the NAAQS RIAs are merely illustrative and are not intended to be added to the costs and benefits of other regulations that result in specific costs of control and emission reductions. However, some costs and benefits estimated in this RIA account for the same air quality improvements as estimated in the illustrative $PM_{2.5}$ NAAQS RIA.

By contrast, the emission reductions for this rule are from a specific class of well-characterized sources. In general, EPA is more confident in the magnitude and location of the emission reductions for these rules. It is important to note that emission reductions anticipated from these rules do not result in emission increases elsewhere (other than potential energy disbenefits). Emission reductions achieved under these and other promulgated rules will ultimately be reflected in the baseline of future NAAQS analyses, which would reduce the incremental costs and benefits associated with attaining the NAAQS. EPA remains forward looking towards the next iteration of the 5-year review cycle for the NAAQS, and as a result does not issue updated RIAs for existing NAAQS that retroactively update the baseline for NAAQS implementation. For more information on the relationship between the NAAQS and rules such as analyzed here, please see Section 1.2.4 of the SO_2 NAAQS RIA (U.S. EPA, 2010a).

To estimate a subset of the co-benefits from reducing $PM_{2.5}$ exposure, EPA used an approach that is consistent with the approach utilized to estimate the co-benefits of the proposed MATS (U.S. EPA 2011a) and the Cross-State Air Pollution Rule (U.S. EPA 2011b). In this analysis we consider an array of health impacts attributable to changes in $PM_{2.5}$ air quality. The 2009 $PM_{2.5}$ Integrated Science Assessment (U.S. EPA, 2009a) identified the human health effects associated with these ambient pollutants, which include premature mortality and a variety of morbidity effects associated with acute and chronic exposures.

Table 5-1 summarizes the total monetized co-benefits of the rule in 2016. This table reflects the economic value of the change in $PM_{2.5}$-related human health impacts and the monetized value of CO_2 reductions occurring as a result of the Mercury and Air Toxics Standards.

Table 5-1. Estimated Monetized Co-benefits of the Mercury and Air Toxics Standards in 2016 (billions of 2007$)[a]

Benefits Estimate	Eastern U.S. [b]	Western U.S.	Total
Pope et al. (2002) $PM_{2.5}$ mortality estimate			
Using a 3% discount rate	$35+B ($2.8 – $110)	$1.1+B ($0.03 – $3.4)	$37+B ($3.2 – $110)
Using a 7% discount rate	$32+B ($2.5 – $98)	$1.0+B ($0.03 – $3.1)	$33+B ($2.9 – $100)
Laden et al. (2006) $PM_{2.5}$ mortality estimate			
Using a 3% discount rate	$87+B ($7.5 – $250)	$2.7+B ($0.1 – $7.9)	$90+B ($8.0 – $260)
Using a 7% discount rate	$78+B ($6.8 – $230)	$2.4+B ($0.1 – $7.2)	$81+B ($7.3 – $240)

[a] For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. Data limitations prevented us from quantifying these endpoints, and as such, these benefits are inherently more uncertain than those benefits that we were able to quantify. A detailed listing of unquantified health and welfare effects is provided in Tables 5-2 and 5-3. Estimates here are subject to uncertainties discussed further in the body of the document. Estimates are rounded to two significant figures. Value of total co-benefits includes CO_2-related co benefits discounted at 3%.

[b] Includes Texas and those states to the north and east.

Tables 5-2 and 5-3 summarize the human health and environmental co-benefits categories contained within the total monetized benefits estimate, and those categories that were unquantified due to limited data or time. It is important to emphasize that the list of unquantified benefit categories is not exhaustive, nor is quantification of each effect complete. In order to identify the most meaningful human health and environmental co-benefits, we excluded effects not identified as having at least a causal, likely causal, or suggestive relationship with the affected pollutants in the most recent comprehensive scientific assessment, such as an Integrated Science Assessment. This does not imply that additional relationships between these and other human health and environmental co-benefits and the affected pollutants do not exist. Due to this decision criterion, some effects that were identified in previous lists of unquantified benefits in other RIAs have been dropped (e.g., UVb exposure). In addition, some quantified effects represent only a partial accounting of likely impacts due to limitations in the currently available data (e.g., climate effects from CO_2, etc).

Table 5-2. Human Health Effects of Pollutants Affected by the Mercury and Air Toxics Standards

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information
Improved Human Health				
Reduced incidence of premature mortality from exposure to $PM_{2.5}$	Adult premature mortality based on cohort study estimates and expert elicitation estimates (age >25 or age >30)	✓	✓	Section 5.4
	Infant mortality (age <1)	✓	✓	Section 5.4
Reduced incidence of morbidity from exposure to $PM_{2.5}$	Non-fatal heart attacks (age > 18)	✓	✓	Section 5.4
	Hospital admissions—respiratory (all ages)	✓	✓	Section 5.4
	Hospital admissions—cardiovascular (age >18)	✓	✓	Section 5.4
	Emergency room visits for asthma (<18)	✓	✓	Section 5.4
	Acute bronchitis (age 8-12)	✓	✓	Section 5.4
	Lower respiratory symptoms (age 7–14)	✓	✓	Section 5.4
	Upper respiratory symptoms (asthmatics age 9–11)	✓	✓	Section 5.4
	Asthma exacerbation (asthmatics age 6–18)	✓	✓	Section 5.4
	Lost work days (age 18–65)	✓	✓	Section 5.4
	Minor restricted-activity days (age 18–65)	✓	✓	Section 5.4
	Chronic Bronchitis (age >26)	✓	✓	Section 5.4
	Other cardiovascular effects (e.g., other ages)	—	—	PM ISA[b]
	Other respiratory effects (e.g., pulmonary function, non-asthma ER visits, non-bronchitis chronic diseases, other ages and populations)	—	—	PM ISA[b]
	Reproductive and developmental effects (e.g., low birth weight, pre-term births, etc)	—	—	PM ISA[b,c]
	Cancer, mutagenicity, and genotoxicity effects	—	—	PM ISA[b,c]
Reduced incidence of mortality from exposure to ozone	Premature mortality based on short-term study estimates (all ages)	—	—	Ozone CD, Draft Ozone ISA[a]
	Premature mortality based on long-term study estimates (age 30–99)	—	—	Ozone CD, Draft Ozone ISA[a]
Reduced incidence of morbidity from exposure to ozone	Hospital admissions—respiratory causes (age > 65)	—	—	Ozone CD, Draft Ozone ISA[a]
	Hospital admissions—respiratory causes (age <2)	—	—	Ozone CD, Draft Ozone ISA[a]
	Emergency room visits for asthma (all ages)	—	—	Ozone CD, Draft Ozone ISA[a]
	Minor restricted-activity days (age 18–65)	—	—	Ozone CD, Draft Ozone ISA[a]

(continued)

Table 5-2. Human Health Effects of Pollutants Affected by the Mercury and Air Toxics Standards (continued)

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information
	School absence days (age 5–17)	—	—	Ozone CD, Draft Ozone ISA[a]
	Decreased outdoor worker productivity (age 18–65)	—	—	Ozone CD, Draft Ozone ISA[a]
	Other respiratory effects (e.g., premature aging of lungs)	—	—	Ozone CD, Draft Ozone ISA[b]
	Cardiovascular and nervous system effects	—	—	Ozone CD, Draft Ozone ISA[c]
	Reproductive and developmental effects	—	—	Ozone CD, Draft Ozone ISA[c]
Reduced incidence of morbidity from exposure to NO_2	Asthma hospital admissions (all ages)	—	—	NO_2 ISA[a]
	Chronic lung disease hospital admissions (age > 65)	—	—	NO_2 ISA[a]
	Respiratory emergency department visits (all ages)	—	—	NO_2 ISA[a]
	Asthma exacerbation (asthmatics age 4–18)	—	—	NO_2 ISA[a]
	Acute respiratory symptoms (age 7–14)	—	—	NO_2 ISA[a]
	Premature mortality	—	—	NO_2 ISA[b,c]
	Other respiratory effects (e.g., airway hyperresponsiveness and inflammation, lung function, other ages and populations)	—	—	NO_2 ISA[b,c]
Reduced incidence of morbidity from exposure to SO_2	Respiratory hospital admissions (age > 65)	—	—	SO_2 ISA[a]
	Asthma emergency room visits (all ages)	—	—	SO_2 ISA[a]
	Asthma exacerbation (asthmatics age 4–12)	—	—	SO_2 ISA[a]
	Acute respiratory symptoms (age 7–14)	—	—	SO_2 ISA[a]
	Premature mortality	—	—	SO_2 ISA[b,c]
	Other respiratory effects (e.g., airway hyperresponsiveness and inflammation, lung function, other ages and populations)	—	—	SO_2 ISA[b,c]
Reduced incidence of morbidity from exposure to methylmercury (through reduced mercury deposition as well as the role of sulfate in methylation)	Neurologic effects - IQ loss	✓	✓	IRIS; NRC, 2000[a]
	Other neurologic effects (e.g., developmental delays, memory, behavior)	—	—	IRIS; NRC, 2000[b]
	Cardiovascular effects	—	—	IRIS; NRC, 2000[b,c]
	Genotoxic, immunologic, and other toxic effects	—	—	IRIS; NRC, 2000[b,c]

[a] We assess these co-benefits qualitatively due to time and resource limitations for this analysis.
[b] We assess these co-benefits qualitatively because we do not have sufficient confidence in available data or methods.
[c] We assess these co-benefits qualitatively because current evidence is only suggestive of causality or there are other significant concerns over the strength of the association.

Table 5-3. Environmental Effects of Pollutants Affected by the Mercury and Air Toxics Standards

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information
Improved Environment				
Reduced visibility impairment	Visibility in Class I areas in SE, SW, and CA regions	—	—	PM ISA[a]
	Visibility in Class I areas in other regions	—	—	PM ISA[a]
	Visibility in residential areas	—	—	PM ISA[a]
Reduced climate effects	Global climate impacts from CO_2	—	✓	Section 5.6
	Climate impacts from ozone and PM	—	—	Section 5.6
	Other climate impacts (e.g., other GHGs, other impacts)	—	—	IPCC[b]
Reduced effects on materials	Household soiling	—	—	PM ISA[b]
	Materials damage (e.g., corrosion, increased wear)	—	—	PM ISA[b]
Reduced effects from PM deposition (metals and organics)	Effects on Individual organisms and ecosystems	—	—	PM ISA[b]
Reduced vegetation and ecosystem effects from exposure to ozone	Visible foliar injury on vegetation	—	—	Ozone CD, Draft Ozone ISA[b]
	Reduced vegetation growth and reproduction	—	—	Ozone CD, Draft Ozone ISA[a]
	Yield and quality of commercial forest products and crops	—	—	Ozone CD, Draft Ozone ISA[a,c]
	Damage to urban ornamental plants	—	—	Ozone CD, Draft Ozone ISA[b]
	Carbon sequestration in terrestrial ecosystems	—	—	Ozone CD, Draft Ozone ISA[b]
	Recreational demand associated with forest aesthetics	—	—	Ozone CD, Draft Ozone ISA[b]
	Other non-use effects			Ozone CD, Draft Ozone ISA[b]
	Ecosystem functions (e.g., water cycling, biogeochemical cycles, net primary productivity, leaf-gas exchange, community composition)	—	—	Ozone CD, Draft Ozone ISA[b]

(continued)

Table 5-3. Environmental Effects of Pollutants Affected by the Mercury and Air Toxics Standards (continued)

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	More Information
Reduced effects from acid deposition	Recreational fishing	—	—	NOx SOx ISA[a]
	Tree mortality and decline	—	—	NOx SOx ISA[b]
	Commercial fishing and forestry effects	—	—	NOx SOx ISA[b]
	Recreational demand in terrestrial and aquatic ecosystems	—	—	NOx SOx ISA[b]
	Other non-use effects			NOx SOx ISA[b]
	Ecosystem functions (e.g., biogeochemical cycles)	—	—	NOx SOx ISA[b]
Reduced effects from nutrient enrichment	Species composition and biodiversity in terrestrial and estuarine ecosystems	—	—	NOx SOx ISA[b]
	Coastal eutrophication	—	—	NOx SOx ISA[b]
	Recreational demand in terrestrial and estuarine ecosystems	—	—	NOx SOx ISA[b]
	Other non-use effects			NOx SOx ISA[b]
	Ecosystem functions (e.g., biogeochemical cycles, fire regulation)	—	—	NOx SOx ISA[b]
Reduced vegetation effects from ambient exposure to SO_2 and NO_x	Injury to vegetation from SO_2 exposure	—	—	NOx SOx ISA[b]
	Injury to vegetation from NO_x exposure	—	—	NOx SOx ISA[b]
Reduced ecosystem effects from exposure to methylmercury (through reduced mercury deposition as well as the role of sulfate in methylation)	Effects on fish, birds, and mammals (e.g., reproductive effects)	—	—	Mercury Study RTC[b,c]
	Commercial, subsistence and recreational fishing	—	—	Mercury Study RTC[b]

[a] We assess these co-benefits qualitatively due to time and resource limitations for this analysis.

[b] We assess these co-benefits qualitatively because we do not have sufficient confidence in available data or methods.

[c] We assess these co-benefits qualitatively because current evidence is only suggestive of causality or there are other significant concerns over the strength of the association.

The co-benefits analysis in this chapter relies on an array of data inputs—including air quality modeling, health impact functions and valuation functions among others—which are themselves subject to uncertainty and may also contribute to the overall uncertainty in this analysis. As a means of characterizing this uncertainty we employ two primary techniques. First,

we use Monte Carlo methods for characterizing random sampling error associated with the concentration response functions from epidemiological studies and economic valuation functions. Second, because this characterization of random statistical error may omit important sources of uncertainty we also employ the results of an expert elicitation on the relationship between premature mortality and ambient $PM_{2.5}$ concentration (Roman et al., 2008). This provides additional insight into the likelihood of different outcomes and about the state of knowledge regarding the co-benefits estimates. Both approaches have different strengths and weaknesses, which are fully described in Chapter 5 of the PM NAAQS RIA (U.S. EPA, 2006a). While the contributions from additional data inputs to uncertainty in the results are not quantified here, this analysis employs best practices in every aspect of its development.

Given that co-benefits of reductions in premature mortality are a dominant share of the overall monetized co-benefits, more focus on uncertainty in mortality-related co-benefits gives us greater confidence in our uncertainty characterization surrounding total $PM_{2.5}$-related co-benefits. Additional sensitivity analyses have been performed for the 2006 PM NAAQS RIA, and were not specifically included here as the results would be similar and would not change the conclusions of the analyses to support this rule. In particular, these analyses characterized the sensitivity of the monetized co-benefits to the specification of alternate cessation lags and income growth adjustment factors. As shown in these RIAs, the estimated co-benefits increased or decreased in proportion to the specification of alternate income growth adjustments and cessation lags. Therefore, readers can infer the sensitivity of the results in this RIA to these parameters by referring to the sensitivity analyses in the PM NAAQS RIA (2006d) and Ozone NAAQS RIA (2008a). For example, based on the results from previous analyses, the use of an alternate lag structure would change the $PM_{2.5}$-related mortality co-benefits discounted at 3% discounted by between 10.4% and –27%; when discounted at 7%, these co-benefits change by between 31% and -49%. When applying higher and lower income growth adjustments, the monetary value of $PM_{2.5}$ -related premature changes between 30% and -10%; the value of chronic endpoints change between 5% and -2% and the value of acute endpoints change between 6% and -7%.

Additionally, in this RIA we binned the estimated population exposed to projected future baseline $PM_{2.5}$ air quality levels for comparison against the “Lowest Measured Level” (LML) of $PM_{2.5}$ air quality in the mortality studies. The purpose of this analysis is to show whether the estimated premature deaths associated with reduced $PM_{2.5}$ exposure occur at or above the range of ambient $PM_{2.5}$ observations studied in Pope et al. (2002) and Laden et al. (2006), which are the two epidemiological studies that EPA uses to estimate $PM_{2.5}$-related

premature mortality co-benefits. We found that a significant proportion of the avoided PM-related premature deaths we estimated in this analysis occurred among populations exposed at or above the LML of each study in the baseline, increasing our confidence in our estimate of the magnitude of the PM-related premature deaths avoided. Approximately 11% of the avoided premature deaths occur at or above an annual mean $PM_{2.5}$ level of 10 $\mu g/m^3$ (the LML of the Laden et al. 2006 study), and about 73% occur at or above an annual mean $PM_{2.5}$ level of 7.5 $\mu g/m^3$ (the LML of the Pope et al. 2002 study). As we model avoided premature deaths among populations exposed to levels of $PM_{2.5}$ that are successively lower than the LML of each study our confidence in the results diminishes.

5.2 Benefits Analysis Methods

We follow a "damage-function" approach in calculating health co-benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints (specific effects that can be associated with changes in air quality) and estimates values of those changes assuming independence between the values of individual endpoints. Total benefits are calculated simply as the sum of the values for all non-overlapping health and welfare endpoints. The "damage-function" approach is the standard method for assessing costs and benefits of environmental quality programs and has been used in several recent published analyses (Levy et al., 2009; Hubbell et al., 2009; Tagaris et al., 2009).

To assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. In some cases, the changes in environmental quality can be directly valued, as is the case for changes in visibility. In other cases, such as for changes in ozone and PM, a health and welfare impact analysis must first be conducted to convert air quality changes into effects that can be assigned dollar values.

We note at the outset that EPA rarely has the time or resources to perform extensive new research to measure directly either the health outcomes or their values for regulatory analyses. Thus, similar to Kunzli et al. (2000) and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is a means of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Adjustments are made for the level of environmental quality change, the socio-demographic and economic characteristics of the affected population, and other factors to improve the accuracy and robustness of benefits estimates.

5.2.1 Health Impact Assessment

Health Impact Assessment (HIA) quantifies changes in the incidence of adverse health impacts resulting from changes in human exposure to specific pollutants, such as $PM_{2.5}$. HIAs are a well-established approach for estimating the retrospective or prospective change in adverse health impacts expected to result from population-level changes in exposure to pollutants (Levy et al. 2009). PC-based tools such as the environmental Benefits Mapping and Analysis Program (BenMAP) can systematize health impact analyses by applying a database of key input parameters, including health impact functions and population projections. Analysts have applied the HIA approach to estimate human health impacts resulting from hypothetical changes in pollutant levels (Hubbell et al. 2005; Davidson et al. 2007, Tagaris et al. 2009). EPA and others have relied upon this method to predict future changes in health impacts expected to result from the implementation of regulations affecting air quality (e.g. U.S. EPA, 2008a). For this assessment, the HIAs are limited to those health effects that are directly linked to ambient $PM_{2.5}$ concentrations. There may be other indirect health impacts associated with implementing emissions controls, such as occupational health impacts for coal miners.

The HIA approach used in this analysis involves three basic steps: (1) utilizing CAMx-generated projections of $PM_{2.5}$ and ozone air quality and estimating the change in the spatial distribution of the ambient air quality; (2) determining the subsequent change in population-level exposure; (3) calculating health impacts by applying concentration-response relationships drawn from the epidemiological literature (Hubbell et al. 2009) to this change in population exposure.

A typical health impact function might look as follows:

$$\Delta y = y_o \cdot \left(e^{\beta \cdot \Delta x} - 1\right) \cdot Pop$$

where y_0 is the baseline incidence rate for the health endpoint being quantified (for example, a health impact function quantifying changes in mortality would use the baseline, or background, mortality rate for the given population of interest); *Pop* is the population affected by the change in air quality; Δx is the change in air quality; and β is the effect coefficient drawn from the epidemiological study. Tools such as BenMAP can systematize the HIA calculation process, allowing users to draw upon a library of existing air quality monitoring data, population data and health impact functions.

Figure 5-1 provides a simplified overview of this approach.

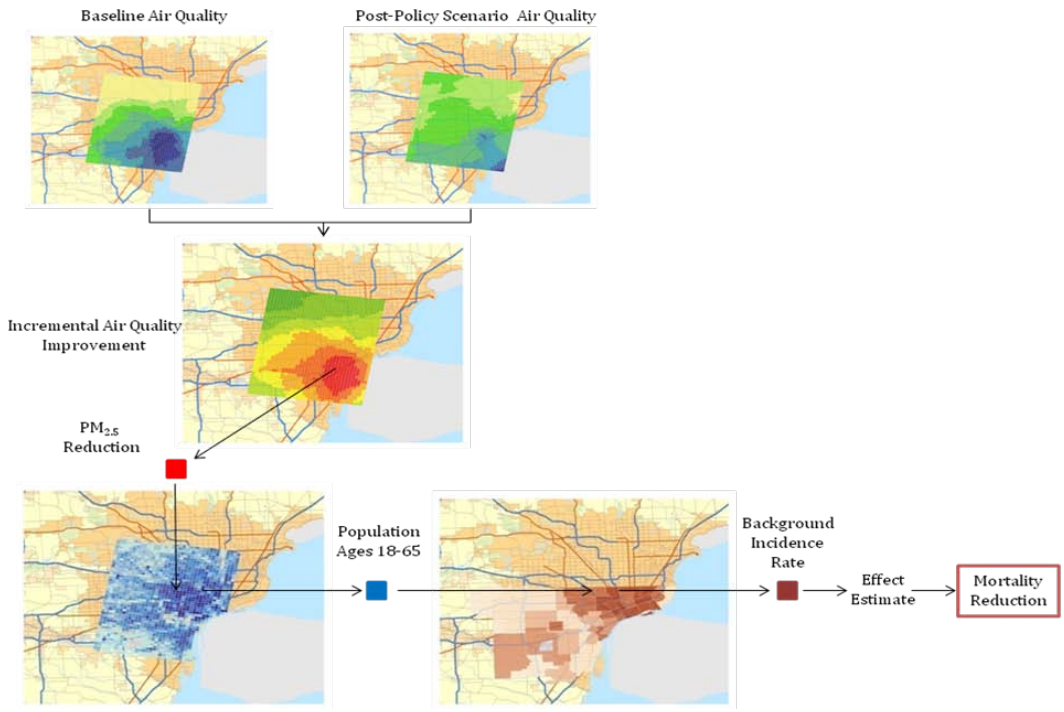

Figure 5-1. Illustration of BenMAP Approach

5.2.2 Economic Valuation of Health Impacts

After quantifying the change in adverse health impacts, the final step is to estimate the economic value of these avoided impacts. The appropriate economic value for a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects by a small amount for a large population. The appropriate economic measure is therefore *ex ante* Willingness to Pay (WTP) for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature mortality amounts to $1 million ($100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These cost of illness (COI) estimates generally (although not in every case) understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect.

We use the BenMAP model version 4 (Abt Associates, 2010) to estimate the health impacts and monetized health co-benefits for the Mercury and Air Toxics Standards. Figure 5-2 shows the data inputs and outputs for the BenMAP model.

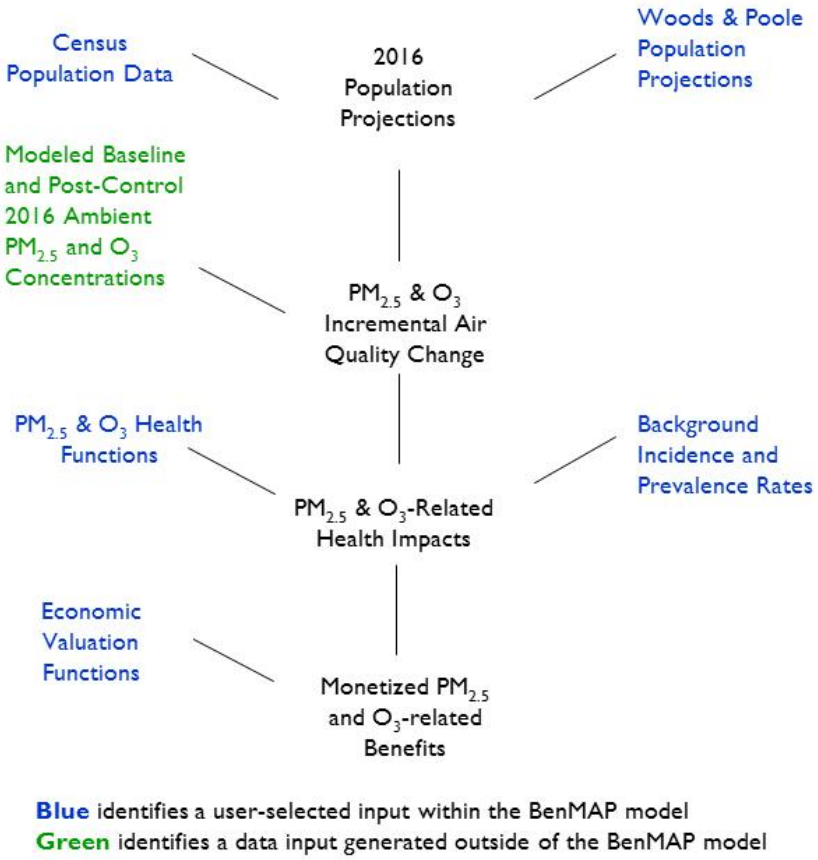

Figure 5-2. Data Inputs and Outputs for the BenMAP Model

5.2.3 Adjusting the Results of the $PM_{2.5}$ co-benefits Analysis to Account for the Emission Reductions in the Final Mercury and Air Toxics Standards

As described in Chapter 3 of this RIA, EPA finalized the rule requirements after the completion of the air quality modeling for this rule. These changes to the rule affected both the overall level and distribution of $PM_{2.5}$ precursor emissions across the U.S., which in turn affect the level of $PM_{2.5}$ co-benefits. We determined that the geographic distribution of emissions reductions resulting from the final rule requirements were sufficiently similar to the modeled interim emissions reductions that we could adjust our co-benefits estimates to reflect these emission changes by applying benefit per-ton estimates generated using the modeled air quality changes.

Benefit per-ton (BPT) estimates quantify the health impacts and monetized human health co-benefits of an incremental change in air pollution precursor emissions. In circumstances where we are unable to perform air quality modeling because of resource or time constraints, this approach can provide a reasonable estimate of the co-benefits of emission reductions. EPA has used the BPT technique in previous RIAs, including the recent Ozone NAAQS RIA (U.S. EPA, 2008a), the NO_2 NAAQS RIA (U.S. EPA, 2010b), the proposed Mercury and Air Toxics Standards RIA (U.S. EPA 2011a), and the Cross-State Air Pollution Rule (U.S. EPA, 2011b).

For this co-benefits analysis we created per-ton estimates of $PM_{2.5}$-related incidence- and monetized co-benefits based on the co-benefits of the air quality modeled scenario. Our approach here is methodologically consistent with the technique reported in Fann, Fulcher & Hubbell (2009), but adjusted for this analysis to better match the spatial distribution of air quality changes expected under the Mercury and Air Toxics Standards. To derive the BPT estimates for this analysis, we:

1. *Quantified the $PM_{2.5}$-related human and monetized health co-benefits of SO_2 and direct $PM_{2.5}$ changes for Eastern and Western states*. We first estimated the health impacts and monetized co-benefits of reductions in directly emitted $PM_{2.5}$ and particulate sulfate.[1] MATS is expected to reduce both SO_2 and NO_x emissions. In general SO_2 is a precursor to particulate sulfate and NO_x is a precursor to particulate nitrate. However, there are also several interactions between the $PM_{2.5}$ precursors which cannot be easily quantified. For example, under conditions in which SO_2 levels are reduced by a substantial margin, "nitrate replacement" may occur. This occurs

[1] Consistent with advice from the Health Effects Subcommittee of the Science Advisory Board (U.S. EPA-SAB, 2010), we assume that each PM species is equally toxic. We quantify the change in incidence for each PM component by applying risk coefficients based on undifferentiated $PM_{2.5}$ mass.

when particulate ammonium sulfate concentrations are reduced, thereby freeing up excess gaseous ammonia. The excess ammonia is then available to react with gaseous nitric acid to form particulate nitrate when meteorological conditions are conducive (cold temperatures and high humidity). The impact of nitrate replacement is also affected by concurrent NO_x reductions. NO_x reductions can lead to decreases in nitrate, which competes with the process of nitrate replacement. NO_x reductions can also lead to reductions in photochemical by-products which can reduce both particulate sulfate and secondary organic carbon PM concentrations.

We found that reductions in NO_x and SO_x resulting from MATS led to significant decreases in particulate sulfate and small increases in particulate nitrate in some locations, indicating that nitrate replacement limited the nitrate decreases from NO_x reductions in some locations. Reductions in directly emitted crustal and carbonaceous $PM_{2.5}$ (elemental carbon and organic carbon) were fairly modest. Carbonaceous $PM_{2.5}$ decreased slightly in the eastern US but did not significantly change in the western US. We elected not to generate a NO_x BPT for three reasons: (a) reductions in NO_x emissions for this rule were relatively small; (b) previous EPA modeling indicates that $PM_{2.5}$ formation is less sensitive to NO_x emission reductions on a per-$\mu g/m^3$ basis (Fann, Fulcher and Hubbell, 2009); and (c) particulate nitrate formation is governed by complex non-linear chemistry that is difficult to characterize using BPT estimates that are derived from a single air quality modeling run which includes both NO_x and SO_2 reductions. Additional modeling runs with SO_2 and NO_x emissions changes modeled separately can provide information that can be used to estimate NOx benefits, and these runs have been conducted for other sectors, but have not been conducted for this rule. For the modeled scenario, sulfate reductions contributed 95% of the health co-benefits of all $PM_{2.5}$ components, with an additional 5% from direct $PM_{2.5}$ reductions (see Appendix 5C). Health co-benefits of sulfate reductions were two orders of magnitude larger than the health disbenefits of nitrate increases. Thus, the SO_2 emission reductions are the main driver for the health co-benefits of this rule.

2. *Divided the health impacts and monetized co-benefits by the emission reduction for the air quality modeling in the corresponding geographic area.* For the reasons described above, we quantified BPT estimates for SO_2 and directly emitted $PM_{2.5}$ (separately for carbonaceous and crustal). For SO_2, we generated an array of eastern and western BPT estimates by dividing the particulate sulfate-related co-benefits in the eastern and western U.S. by the total SO_2-related emission reductions in these two areas. As the chemistry of nitrate formation is complex and non-linear, nitrate impacts were excluded from the BPT analysis. Nitrates can be reduced when NO_x emissions are reduced or increased when SO_2 emissions are reduced. The increased nitrate health impacts in the modeled interim scenario were two orders of magnitude smaller than the sulfate health benefits. Thus, we estimate that including nitrate health impacts on the calculation for SO_2 BPT would reduce the SO_2 BPT by 1-2%, with a similar magnitude impact on the total health benefits of the rule.

Carbonaceous and crustal $PM_{2.5}$ BPT estimates were similarly generated using the co-benefits and emissions of those species.

The resulting BPT estimates (listed in Table 5C-3) were then multiplied by the projected SO_2 (1.33 million tons), carbonaceous $PM_{2.5}$ (6,100 tons), and crustal $PM_{2.5}$ (39,000 tons) emission reductions for the final policy to produce an estimate of the $PM_{2.5}$-related health impacts and monetized co-benefits. Due to time limitations, direct $PM_{2.5}$ benefits are based on direct $PM_{2.5}$ emission reductions from an earlier policy scenario. However, since direct $PM_{2.5}$ benefits contribute only approximately 5% to the total $PM_{2.5}$ health co-benefits of this rule, and differences between direct $PM_{2.5}$ emission reductions between the earlier and final policy scenarios are expected to be modest, use of earlier $PM_{2.5}$ emission changes is unlikely to materially affect the results. Additional details on the BPT methodology and derivation are given in Appendix 5C.

An implicit assumption in our approach is that the size and distribution of SO_2 emissions, and the relative levels of NO_x and SO_2 emissions, are fairly similar in the modeled and revised policy cases. In general, the modeled and revised policy cases achieve roughly similar levels of SO_2 reductions (1.42 versus 1.33 million tons, respectively) with a similar distribution among states. However, for some states (notably Alabama, Colorado, Louisiana, Michigan, Missouri, North Dakota, Oklahoma, and Texas), SO_2 emission reductions were lower for the final case versus the interim case. By far, the greatest difference in SO_2 emission reductions was in Michigan where the final case emission reduction was 70% lower than for the interim case. In a few states (notably Arkansas, Ohio, and South Carolina), SO_2 emission reductions were slightly larger for the final case versus the interim case. Since differences between the interim and final cases are not concentrated in any particular region of the country and the overall distribution of emission reductions is similar, we conclude that it is reasonable to apply BPT values derived from the interim case to the final case. While NO_x emissions reductions decreased by 70% between the interim and final cases (141,000 vs. 46,000 tons), the impact of NO_x on $PM_{2.5}$ concentrations and mortality is very minor relative to the impact of SO_2 emission reductions. Therefore, differences in the magnitude and distribution of NO_x emission reductions are likely to have only a minor effect on results.

We did not develop ozone BPT estimates for this rule for two reasons. First, the overall level of ozone-related co-benefits in the modeled case is relatively small compared to those associated with $PM_{2.5}$ reductions, due in part to the modest NO_x emission reductions. Second, the complex non-linear chemistry of ozone formation introduces uncertainty to the

development and application of BPT estimates. Taken together, these factors argued against developing ozone BPT estimates for this RIA.

As there is no analogous approach for estimating visibility co-benefits using the BPT approach, visibility co-benefits are calculated for the modeled interim policy scenario only and are not included in estimate of co-benefits for the final rule. However, since the magnitude of SO_2 emission reductions did not significantly change in the visibility study areas between the interim and final emissions scenarios, we expect the visibility benefit for the final policy scenario would be similar to that calculated for the interim policy scenario ($1.1 billion in total for the U.S., using 2007$; see Appendix 5C).

5.3 Uncertainty Characterization

As for any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty affecting estimated results, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain and, depending on its role in the co-benefits analysis, may have a disproportionately large impact on estimates of total monetized co-benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to large impacts on total monetized co-benefits.

The National Research Council (NRC) (2002, 2008) highlighted the need for EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates and to present these estimates to decision makers in ways that foster an appropriate appreciation of their inherent uncertainty. In general, the NRC concluded that EPA's methodology for calculating the benefits of reducing air pollution is reasonable and informative in spite of inherent uncertainties. Since the publication of these reports, EPA continues to improve the characterization of uncertainties for both health incidence and benefits estimates. We use both Monte Carlo analysis and expert-derived concentration-response functions to assess uncertainty quantitatively, as well as to provide a qualitative assessment for those aspects that we are unable to address quantitatively.

First, we used Monte Carlo methods to characterize both sampling error and variability across the economic valuation functions, including random sampling error associated with the

concentration response functions from epidemiological studies and random effects modeling. Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables, such as incidence of premature mortality. Specifically, we used Monte Carlo methods to generate confidence intervals around the estimated health impact and dollar benefits. The reported standard errors in the epidemiological studies determined the distributions for individual effect estimates.

Second, because characterization of random statistical error omits important sources of uncertainty (e.g., in the functional form of the model—e.g., whether or not a threshold may exist), we also incorporate the results of an expert elicitation on the relationship between premature mortality and ambient $PM_{2.5}$ concentration (Roman et al., 2008). Use of the expert elicitation and incorporation of the standard errors approaches provide insights into the likelihood of different outcomes and about the state of knowledge regarding the benefits estimates. However, there remain significant unquantified uncertainties present in upstream inputs including emission and air quality. Both uncertainty characterization approaches have different strengths and weaknesses, as detailed in Chapter 5 of the PM NAAQS RIA (U.S. EPA, 2006a).

In benefit analyses of air pollution regulations conducted to date, the estimated impact of reductions in premature mortality has accounted for 85% to 95% of total monetized benefits. Therefore, it is particularly important to attempt to characterize the uncertainties associated with reductions in premature mortality. The health impact functions used to estimate avoided premature deaths associated with reductions in ozone have associated standard errors that represent the statistical errors around the effect estimates in the underlying epidemiological studies. In our results, we report credible intervals based on these standard errors, reflecting the uncertainty in the estimated change in incidence of avoided premature deaths. We also provide multiple estimates, to reflect model uncertainty between alternative study designs.

For premature mortality associated with exposure to PM, we follow the same approach used in the RIA for 2006 PM NAAQS (U.S. EPA, 2006a), presenting two empirical estimates of premature deaths avoided, and a set of twelve estimates based on results of the expert elicitation study. Even these multiple characterizations, including confidence intervals, omit the contribution to overall uncertainty of uncertainty in air quality changes, baseline incidence rates, populations exposed and transferability of the effect estimate to diverse locations. Furthermore, the approach presented here does not include methods for addressing correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty in additional model elements. As a

result, the reported confidence intervals and range of estimates give an incomplete picture about the overall uncertainty in the estimates. This information should be interpreted within the context of the larger uncertainty surrounding the entire analysis.

EPA estimates PM-related mortality without assuming a health effect threshold at low concentrations, based on the current body of scientific literature (U.S. EPA-SAB, 2009a, U.S. EPA-SAB, 2009b). However, as we model mortality impacts among populations exposed to levels of $PM_{2.5}$ that are successively lower than the lowest measured level (LML) in each epidemiology study our confidence in the results diminishes. In addition to the uncertainty analyses described above, we therefore include an assessment of the mortality benefits accruing to populations exposed to baseline $PM_{2.5}$ concentrations above the LML in the two main epidemiology studies used to quantify benefits (see Section 5.7). Based on the modeled interim baseline which is approximately equivalent to the final baseline (see Appendix 5A), 11% and 73% of the estimated avoided mortality impacts occur at or above an annual mean $PM_{2.5}$ level of 10 $\mu g/m^3$ (the LML of the Laden et al. 2006 study) and 7.5 $\mu g/m^3$ (the LML of the Pope et al. 2002 study), respectively.

Key sources of uncertainty in the $PM_{2.5}$ health impact assessment include:

- gaps in scientific data and inquiry;
- variability in estimated relationships, such as epidemiological effect estimates, introduced through differences in study design and statistical modeling;
- errors in measurement and projection for variables such as population growth rates;
- errors due to misspecification of model structures, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions;
- biases due to omissions or other research limitations; and
- additional uncertainties from benefits transfer method using BPT estimates.

In Table 5-4 we summarize some of the key uncertainties in the benefits analysis.

Table 5-4. Primary Sources of Uncertainty in the Benefits Analysis

1. Uncertainties Associated with Impact Functions

- The value of the ozone or PM effect estimate in each impact function.
- Application of a single impact function to pollutant changes and populations in all locations.
- Similarity of future-year impact functions to current impact functions.
- Correct functional form of each impact function.
- Extrapolation of effect estimates beyond the range of ozone or PM concentrations observed in the source epidemiological study.
- Application of impact functions only to those subpopulations matching the original study population.

2. Uncertainties Associated with CAMx-Modeled Ozone and PM Concentrations

- Responsiveness of the models to changes in precursor emissions from the control policy.
- Projections of future levels of precursor emissions, especially ammonia and crustal materials.
- Lack of ozone and $PM_{2.5}$ monitors in all rural areas requires extrapolation of observed ozone data from urban to rural areas.

3. Uncertainties Associated with PM Mortality Risk

- Limited scientific literature supporting a direct biological mechanism for observed epidemiological evidence.
- Direct causal agents within the complex mixture of PM have not been identified.
- The extent to which adverse health effects are associated with low-level exposures that occur many times in the year versus peak exposures.
- The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study.
- Reliability of the $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.

4. Uncertainties Associated with Possible Lagged Effects

- The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels that would occur in a single year is uncertain as well as the portion that might occur in subsequent years.

5. Uncertainties Associated with Baseline Incidence Rates

- Some baseline incidence rates are not location specific (e.g., those taken from studies) and therefore may not accurately represent the actual location-specific rates.
- Current baseline incidence rates may not approximate well baseline incidence rates in 2016.
- Projected population and demographics may not represent well future-year population and demographics.

6. Uncertainties Associated with Economic Valuation

- Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them.
- Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates because of differences in income or other factors.

7. Uncertainties Associated with Aggregation of Monetized Benefits

- Health and welfare benefits estimates are limited to the available impact functions. Thus, unquantified or unmonetized benefits are not included.

$PM_{2.5}$ mortality benefits represent a substantial proportion of total monetized co-benefits (over 90%), and these estimates have following key assumptions and uncertainties.

1. The $PM_{2.5}$-related co-benefits were derived through a benefit per-ton approach, which does not fully reflect local variability in population density, meteorology, exposure, baseline health incidence rates, or other local factors that might lead to

an over-estimate or under-estimate of the actual co-benefits of controlling PM precursors. In addition, differences in the distribution of emissions reductions across states between the modeled scenario and the final rule scenario add uncertainty to the final benefits estimates.

2. We assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because $PM_{2.5}$ produced via transported precursors emitted from EGUs may differ significantly from direct $PM_{2.5}$ released from diesel engines and other industrial sources, but the scientific evidence is not yet sufficient to allow differential effects estimates by particle type.

3. We assume that the health impact function for fine particles is linear within the range of ambient concentrations under consideration. Thus, the estimates include health co-benefits from reducing fine particles in areas with varied concentrations of $PM_{2.5}$, including both regions that are in attainment with fine particle standard and those that do not meet the standard down to the lowest modeled concentrations.

5.4 Benefits Analysis Data Inputs

In Figure 5-2, we summarized the key data inputs to the health impact and economic valuation estimate. Below we summarize the data sources for each of these inputs, including demographic projections, effect coefficients, incidence rates and economic valuation. Our approach here is generally consistent with the Regulatory Impact Analysis for the Cross-State Air Pollution Rule (U.S. EPA, 2011b).

5.4.1 Demographic Data

Quantified and monetized human health impacts depend on the demographic characteristics of the population, including age, location, and income. We use projections based on economic forecasting models developed by Woods and Poole, Inc. (Woods and Poole, 2008). The Woods and Poole (WP) database contains county-level projections of population by age, sex, and race out to 2030. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman et al., 2000). According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four-stage process:

1. First, national-level variables such as income, employment, and populations are forecasted.

2. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an "export-base" approach, which relies on linking industrial-sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-based approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector.

3. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a cohort-component method based on fertility and mortality in each area.

4. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2016 based on historical rates of mortality, fertility, and migration.

5.4.2 Effect Coefficients

The first step in selecting effect coefficients is to identify the health endpoints to be quantified. We base our selection of health endpoints on consistency with EPA's Integrated Science Assessments (which replace the Criteria Document), with input and advice from the EPA Science Advisory Board - Health Effects Subcommittee (SAB-HES), a scientific review panel specifically established to provide advice on the use of the scientific literature in developing benefits analyses for air pollution regulations (http://www.epa.gov/sab/). In general, we follow a weight of evidence approach, based on the biological plausibility of effects, availability of concentration-response functions from well conducted peer-reviewed epidemiological studies, cohesiveness of results across studies, and a focus on endpoints reflecting public health impacts (like hospital admissions) rather than physiological responses (such as changes in clinical measures like Forced Expiratory Volume (FEV1)).

There are several types of data that can support the determination of types and magnitude of health effects associated with air pollution exposures. These sources of data include toxicological studies (including animal and cellular studies), human clinical trials, and observational epidemiology studies. All of these data sources provide important contributions to the weight of evidence surrounding a particular health impact. However, only epidemiology studies provide direct concentration-response relationships which can be used to evaluate population-level impacts of reductions in ambient pollution levels in a health impact assessment.

For the data-derived estimates, we relied on the published scientific literature to ascertain the relationship between PM and adverse human health effects. We evaluated epidemiological studies using the selection criteria summarized in Table 5-5. These criteria include consideration of whether the study was peer-reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility. In general, the use of results from more than a single study can provide a more robust estimate of the relationship between a pollutant and a given health effect. However, there are often differences between studies examining the same endpoint, making it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants or different age groups. For this reason, we consider very carefully the set of studies available examining each endpoint and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either because of a lack of multiple studies, consistency problems, or clear superiority in the quality or comprehensiveness of one study over others, a single published study is selected as the basis of the effect estimate.

Table 5-5. Criteria Used When Selecting C-R Functions

Consideration	Comments
Peer-Reviewed Research	Peer-reviewed research is preferred to research that has not undergone the peer-review process.
Study Type	Among studies that consider chronic exposure (e.g., over a year or longer), prospective cohort studies are preferred over ecological studies because they control for important individual-level confounding variables that cannot be controlled for in ecological studies.
Study Period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and lifestyle over time. However, when there are only a few studies available, studies from all years will be included.

(continued)

Table 5-5. Criteria Used when Selecting C-R Functions (continued)

Consideration	Comments
Population Attributes	The most technically appropriate measures of benefits would be based on impact functions that cover the entire sensitive population but allow for heterogeneity across age or other relevant demographic factors. In the absence of effect estimates specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select effect estimates that cover the broadest population to match with the desired outcome of the analysis, which is total national-level health impacts. When available, multi-city studies are preferred to single city studies because they provide a more generalizable representation of the C-R function.
Study Size	Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population or through repeated observations on a smaller population (e.g., through a symptom diary recorded for a panel of asthmatic children).
Study Location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior, and lifestyle.
Pollutants Included in Model	When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it is important to use properly specified impact functions that include both pollutants. Using single-pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated.
Measure of PM	For this analysis, impact functions based on $PM_{2.5}$ are preferred to PM_{10} because of the focus on reducing emissions of $PM_{2.5}$ precursors, and because air quality modeling was conducted for this size fraction of PM. Where $PM_{2.5}$ functions are not available, PM_{10} functions are used as surrogates, recognizing that there will be potential downward (upward) biases if the fine fraction of PM_{10} is more (less) toxic than the coarse fraction.
Economically Valuable Health Effects	Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis.
Non-overlapping Endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double-counting of benefits.

When several effect estimates for a pollutant and a given health endpoint have been selected, they are quantitatively combined or pooled to derive a more robust estimate of the relationship. The BenMAP Technical Appendices provides details of the procedures used to combine multiple impact functions (Abt Associates, 2010). In general, we used fixed or random effects models to pool estimates from different studies of the same endpoint. Fixed effects pooling simply weights each study's estimate by the inverse variance, giving more weight to studies with greater statistical power (lower variance). Random effects pooling accounts for both within-study variance and between-study variability, due, for example, to differences in population susceptibility. We used the fixed effects model as our null hypothesis and then

determined whether the data suggest that we should reject this null hypothesis, in which case we would use the random effects model. Pooled impact functions are used to estimate hospital admissions and asthma exacerbations. For more details on methods used to pool incidence estimates, see the BenMAP Manual Appendices (Abt Associates, 2010), which are available with the BenMAP software at http://www.epa.gov/benmap.html.

Effect estimates selected for a given health endpoint were applied consistently across all locations nationwide. This applies to both impact functions defined by a single effect estimate and those defined by a pooling of multiple effect estimates. Although the effect estimate may, in fact, vary from one location to another (e.g., because of differences in population susceptibilities or differences in the composition of PM), location-specific effect estimates are generally not available.

The specific studies from which effect estimates for the primary analysis are drawn are included in Table 5-6. In all cases where effect estimates are drawn directly from epidemiological studies, standard errors are used as a partial representation of the uncertainty in the size of the effect estimate. Below we provide the basis for selecting these studies.

5.4.2.1 $PM_{2.5}$ Premature Mortality Effect Coefficients

Both long- and short-term exposures to ambient levels of $PM_{2.5}$ air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most significant health endpoint quantified in this analysis.

Table 5-6. Health Endpoints and Epidemiological Studies Used to Quantify Health Impacts[a]

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature mortality—cohort study, all-cause	$PM_{2.5}$ (annual avg)	Pope et al. (2002) Laden et al. (2006)	>29 years >25 years
Premature mortality, total exposures	$PM_{2.5}$ (annual avg)	Expert Elicitation (Roman et al., 2008)	>24 years
Premature mortality—all-cause	$PM_{2.5}$ (annual avg)	Woodruff et al. (2006)	Infant (<1 year)

(continued)

Table 5-6. Health Endpoints and Epidemiological Studies Used to Quantify Health Impacts[a] (continued)

Endpoint	Pollutant	Study	Study Population
Chronic Illness			
Chronic bronchitis	$PM_{2.5}$ (annual avg)	Abbey et al. (1995)	>26 years
Non-fatal heart attacks	$PM_{2.5}$ (24-hour avg)	Peters et al. (2001)	Adults (>18 years)
Hospital Admissions			
Respiratory	$PM_{2.5}$ (24-hour avg)	*Pooled estimate:* Moolgavkar (2003)—ICD 490–496 (COPD) Ito (2003)—ICD 490–496 (COPD)	>64 years
	$PM_{2.5}$ (24-hour avg)	Moolgavkar (2000)—ICD 490–496 (COPD)	20–64 years
	$PM_{2.5}$ (24-hour avg)	Ito (2003)—ICD 480–486 (pneumonia)	>64 years
	$PM_{2.5}$ (24-hour avg)	Sheppard (2003)—ICD 493 (asthma)	<65 years
Cardiovascular	$PM_{2.5}$ (24-hour avg)	*Pooled estimate:* Moolgavkar (2003)—ICD 390–429 (all cardiovascular) Ito (2003)—ICD 410–414, 427–428 (ischemic heart disease, dysrhythmia, heart failure)	>64 years
	$PM_{2.5}$ (24-hour avg)	Moolgavkar (2000)—ICD 390–429 (all cardiovascular)	20–64 years
Asthma-related ER visits	$PM_{2.5}$ (24-hour avg)	Norris et al. (1999)	0–18 years
Other Health Endpoints			
Acute bronchitis	$PM_{2.5}$ (annual avg)	Dockery et al. (1996)	8–12 years
Upper respiratory symptoms	PM_{10} (24-hour avg)	Pope et al. (1991)	Asthmatics, 9–11 years
Lower respiratory symptoms	$PM_{2.5}$ (24-hour avg)	Schwartz and Neas (2000)	7–14 years
Asthma exacerbations	$PM_{2.5}$ (24-hour avg)	Pooled estimate: Ostro et al. (2001) (cough, wheeze and shortness of breath) Vedal et al. (1998) (cough)	6–18 years[b]

(continued)

Table 5-6. Health Endpoints and Epidemiological Studies Used to Quantify Health Impacts[a] (continued)

Endpoint	Pollutant	Study	Study Population
Work loss days	$PM_{2.5}$ (24-hour avg)	Ostro (1987)	18–65 years
Minor Restricted Activity Days (MRADs)	$PM_{2.5}$ (24-hour avg)	Ostro and Rothschild (1989)	18–65 years

[a] Studies or air quality metrics highlighted in blue represent updates incorporated since the 2005 CAIR RIA

[b] The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the Science Advisory Board Health Effects Subcommittee (SAB-HES), we extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group. See: U.S. Science Advisory Board. 2004. Advisory Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis –Benefits and Costs of the Clean Air Act, 1990—2020. EPA-SAB-COUNCIL-ADV-04-004. See also National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.

Although a number of uncertainties remain to be addressed by continued research (NRC, 2002), a substantial body of published scientific literature documents the correlation between elevated $PM_{2.5}$ concentrations and increased mortality rates (U.S. EPA, 2009a). Time-series methods have been used to relate short-term (often day-to-day) changes in $PM_{2.5}$ concentrations and changes in daily mortality rates up to several days after a period of elevated $PM_{2.5}$ concentrations. Cohort methods have been used to examine the potential relationship between community-level PM exposures over multiple years (i.e., long-term exposures) and community-level annual mortality rates. Researchers have found statistically significant associations between $PM_{2.5}$ and premature mortality using both types of studies. In general, the risk estimates based on the cohort studies are larger than those derived from time-series studies. Cohort analyses are thought to better capture the full public health impact of exposure to air pollution over time, because they account for the effects of long-term exposures and possibly some component of short-term exposures (Kunzli et al., 2001; NRC, 2002). This section discusses some of the issues surrounding the estimation of $PM_{2.5}$-related premature mortality. To demonstrate the sensitivity of the benefits estimates to the specific sources of information regarding the impact of $PM_{2.5}$ exposures on the risk of premature death, we are providing estimates in our results tables based on studies derived from the epidemiological literature and from the EPA sponsored expert elicitation. The epidemiological studies from which these estimates are drawn are described below. The expert elicitation project and the derivation of effect estimates from the expert elicitation results are described in the 2006 $PM_{2.5}$ NAAQS RIA and Roman et al. (2008). In the interest of brevity we do not repeat those details here.

However, Figure 5-13 summarizes the estimated $PM_{2.5}$-related premature mortalities avoided using risk estimates drawn from the expert elicitation.

Over a dozen epidemiological studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977). Most of the published studies found positive (but not always statistically significant) associations with available PM indices such as total suspended particles (TSP). However, exploration of alternative model specifications sometimes raised questions about causal relationships (e.g., Lipfert et al., 1989). These early "ecological cross-sectional" studies (Lave and Seskin, 1977; Ozkaynak and Thurston, 1987) were criticized for a number of methodological limitations, particularly for inadequate control at the individual level for variables that are potentially important in causing mortality, such as wealth, smoking, and diet.

Over the last 17 years, several studies using "prospective cohort" designs have been published that appear to be consistent with the earlier body of literature. These new "prospective cohort" studies reflect a significant improvement over the earlier work because they include individual level information with respect to health status and residence. The most extensive analyses have been based on data from two prospective cohort groups, often referred to as the Harvard "Six-Cities Study" (Dockery et al., 1993; Laden et al., 2006) and the "American Cancer Society or ACS study" (Pope et al., 1995; Pope et al., 2002; Pope et al., 2004, Krewski et al. 2009); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the United States. A third major data set comes from the California-based 7th Day Adventist Study (e.g., Abbey et al., 1999), which reported associations between long-term PM exposure and mortality in men. Results from this cohort, however, have been inconsistent, and the air quality results are not geographically representative of most of the United States, and the lifestyle of the population is not reflective of much of the U.S. population. Analysis is also available for a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000; Lipfert et al., 2003, 2006). The characteristics of this group differ from the cohorts in the Six-Cities, ACS, and 7th Day Adventist studies with respect to income, race, health status, and smoking status. Unlike previous long-term analyses, this study found some associations between mortality and ozone but found inconsistent results for PM indicators. Because of the selective nature of the population in the veteran's cohort, we have chosen not to include any effect estimates from the Lipfert et al. (2000) study in our co-benefits assessment.

Given their consistent results and broad geographic coverage, and importance in informing the NAAQS development process, the Six-Cities and ACS data have been particularly important in benefits analyses. The credibility of these two studies is further enhanced by the fact that the initial published studies (Pope et al., 1995 and Dockery et al., 1993) were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by the Health Effect Institute (HEI) (Krewski et al., 2000). The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded the conclusions of the original investigators. While the HEI reexamination lends credibility to the original studies, it also highlights sensitivities concerning the relative impact of various pollutants, such as SO_2, the potential role of education in mediating the association between pollution and mortality, and the influence of spatial correlation modeling. Further confirmation and extension of the findings of the 1993 Six City Study and the 1995 ACS study were recently completed using more recent air quality and a longer follow-up period for the ACS cohort was published over the past several years (Pope et al., 2002, 2004; Laden et al., 2006, Krewski et al. 2009). The follow up to the Harvard Six City Study both confirmed the effect size from the first analysis and provided additional confirmation that reductions in $PM_{2.5}$ are likely to result in reductions in the risk of premature death. This additional evidence stems from the observed reductions in $PM_{2.5}$ in each city during the extended follow-up period. Laden et al. (2006) found that mortality rates consistently went down at a rate proportionate to the observed reductions in $PM_{2.5}$.

A number of additional analyses have been conducted on the ACS cohort data (Jerrett et al., 2009; Pope et al., 2009). These studies have continued to find a strong significant relationship between $PM_{2.5}$ and mortality outcomes and life expectancy. Specifically, much of the recent research has suggested a stronger relationship between cardiovascular mortality and lung cancer mortality with $PM_{2.5}$, and a less significant relationship between respiratory-related mortality and $PM_{2.5}$. The extended analyses of the ACS cohort data (Krewski et al. 2009) provides additional refinements to the analysis of PM-related mortality by (a) extend the follow-up period by 2 years to the year 2000, for a total of 18 years; (b) incorporate ecological., or neighborhood-level co-variates so as to better estimate personal exposure; (c) perform an extensive spatial analysis using land use regression modeling. These additional refinements may make this analysis well-suited for the assessment of PM-related mortality for EPA benefits analyses.

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, EPA consulted with the SAB-HES. That panel

recommended using long-term prospective cohort studies in estimating mortality risk reduction (U.S. EPA-SAB, 1999). This recommendation has been confirmed by a report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure" (NRC, 2002, p. 108). More specifically, the SAB recommended emphasis on the ACS study because it includes a much larger sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six Cities Study) than other studies of its kind. Because of the refinements in the extended follow-up analysis, the SAB-HES recommended using the Pope et al. (2002) study as the basis for the primary mortality estimate for adults and suggests that alternate estimates of mortality generated using other cohort and time-series studies could be included as part of the sensitivity analysis (U.S. EPA-SAB, 2004a). The PM NAAQS Risk and Exposure Assessment (U.S. EPA, 2010c) utilized risk coefficients drawn from the Krewski et al. (2009) study. In a December of 2009 consultation with the SAB-HES, the Agency proposed utilizing the Krewski et al. (2009) extended analysis of the ACS cohort data. The panel is scheduled to issue an advisory in early 2010.

As noted above, since 2004 SAB review, an extended follow-up of the Harvard Six cities study has been published (Laden et al., 2006) and in recent RIAs (see for example the Cross-State Air Pollution Rule RIA, U.S. EPA 2011b), we have included this estimate of mortality impacts based on application of the C-R function derived from this study. We use this specific estimate to represent the Six Cities study because it both reflects among the most up-to-date science and was cited by many of the experts in their elicitation responses. It is clear from the expert elicitation that the results published in Laden et al. (2006) are potentially influential, and in fact the expert elicitation results encompass within their range the estimates from both the Pope et al. (2002) and Laden et al. (2006) studies (see Figure 5-3). These are logical choices for anchor points in our presentation because, while both studies are well designed and peer reviewed, there are strengths and weaknesses inherent in each, which we believe argues for using both studies to generate benefits estimates.

5.4.2.2 Chronic Bronchitis (CB)

CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al. (1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Because $PM_{2.5}$ reductions are expected from MATS, this analysis uses only the Abbey et

al. (1995) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB.

5.4.2.3 Non-fatal Myocardial Infarctions (Heart Attacks)

Non-fatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between $PM_{2.5}$ and non-fatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including those for non-fatal heart attacks, and PM. Given the lasting impact of a heart attack on long-term health costs and earnings, we provide a separate estimate for non-fatal heart attacks. The estimate used in the MATS analysis is based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997; Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

5.4.2.4 Hospital and Emergency Room Admissions

Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. In addition, some studies have examined the relationship between air pollution and emergency room visits. Since most emergency room visits do not result in an admission to the hospital (the majority of people going to the emergency room are treated and return home), we treat hospital admissions and emergency room visits separately, taking account of the fraction of emergency room visits that are admitted to the hospital.

The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking ozone or PM

with other types of hospital admissions. The only type of emergency room visits that have been consistently linked to ozone and PM in the United States are asthma-related visits.

To estimate avoided incidences of cardiovascular hospital admissions associated with $PM_{2.5}$, we used studies by Moolgavkar (2003) and Ito (2003). Additional published studies show a statistically significant relationship between PM_{10} and cardiovascular hospital admissions. However, given that the control options we are analyzing are expected to reduce primarily $PM_{2.5}$, we focus on the two studies that examine $PM_{2.5}$. Both of these studies provide an effect estimate for populations over 65, allowing us to pool the impact functions for this age group. Only Moolgavkar (2000) provided a separate effect estimate for populations 20 to 64.[2] Total cardiovascular hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. To avoid double-counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

To estimate total avoided incidences of respiratory hospital admissions, we used impact functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, additional published studies show a statistically significant relationship between PM_{10} and respiratory hospital admissions. We used only those focusing on $PM_{2.5}$. Both Moolgavkar (2000) and Ito (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000) provides a separate effect estimate for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Ito (2003) estimated pneumonia and only for the population 65 and older. In addition, Sheppard (2003) provided an effect estimate for asthma hospital admissions for populations under age 65. Total avoided incidences of PM-related respiratory-related hospital admissions are the sum of COPD, pneumonia, and asthma admissions.

[2] Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we chose to use the existing study. Given the very small (<5 percent) difference in the effect estimates for people 65 and older with cardiovascular hospital admissions between the original and reanalyzed results, we do not expect this choice to introduce much bias.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the effect estimate from a study of children 18 and under by Norris et al. (1999). As noted earlier, there is another study by Schwartz examining a broader age group (less than 65), but the Schwartz study focused on PM_{10} rather than $PM_{2.5}$. We selected the Norris et al. (1999) effect estimate because it better matched the pollutant of interest. Because children tend to have higher rates of hospitalization for asthma relative to adults under 65, we will likely capture the majority of the impact of $PM_{2.5}$ on asthma emergency room visits in populations under 65, although there may still be significant impacts in the adult population under 65.

To estimate avoided incidences of respiratory hospital admissions associated with ozone, we used a number of studies examining hospital admissions for a range of respiratory illnesses, including pneumonia and COPD. Two age groups, adults over 65 and children under 2, were examined. For adults over 65, Schwartz (1995) provides effect estimates for two different cities relating ozone and hospital admissions for all respiratory causes (defined as ICD codes 460–519). Impact functions based on these studies were pooled first before being pooled with other studies. Two studies (Moolgavkar et al., 1997; Schwartz, 1994a) examine ozone and pneumonia hospital admissions in Minneapolis. One additional study (Schwartz, 1994b) examines ozone and pneumonia hospital admissions in Detroit. The impact functions for Minneapolis were pooled together first, and the resulting impact function was then pooled with the impact function for Detroit. This avoids assigning too much weight to the information coming from one city. For COPD hospital admissions, two studies are available: Moolgavkar et al. (1997), conducted in Minneapolis, and Schwartz (1994b), conducted in Detroit. These two studies were pooled together. To estimate total respiratory hospital admissions for adults over 65, COPD admissions were added to pneumonia admissions, and the result was pooled with the Schwartz (1995) estimate of total respiratory admissions. Burnett et al. (2001) is the only study providing an effect estimate for respiratory hospital admissions in children under 2.

We used two studies as the source of the concentration-response functions we used to estimate the effects of ozone exposure on asthma-related emergency room (ER) visits: Peel et al. (2005) and Wilson et al. (2005). We estimated the change in ER visits using the effect estimate(s) from each study and then pooled the results using the random effects pooling technique (see Abt, 2005). The Peel et al. (2005) study estimated asthma-related ER visits for all ages in Atlanta, using air quality data from 1993 to 2000. Using Poisson generalized estimating equations, the authors found a marginal association between the maximum daily 8-hour average ozone level and ER visits for asthma over a 3-day moving average (lags of 0, 1, and 2 days) in a single pollutant model. Wilson et al. (2005) examined the relationship between ER

visits for respiratory illnesses and asthma and air pollution for all people residing in Portland, Maine from 1998–2000 and Manchester, New Hampshire from 1996–2000. For all models used in the analysis, the authors restricted the ozone data incorporated into the model to the months ozone levels are usually measured, the spring-summer months (April through September). Using the generalized additive model, Wilson et al. (2005) found a significant association between the maximum daily 8-hour average ozone level and ER visits for asthma in Portland, but found no significant association for Manchester. Similar to the approach used to generate effect estimates for hospital admissions, we used random effects pooling to combine the results across the individual study estimates for ER visits for asthma. The Peel et al. (2005) and Wilson et al. (2005) Manchester estimates were not significant at the 95 percent level, and thus, the confidence interval for the pooled incidence estimate based on these studies includes negative values. This is an artifact of the statistical power of the studies, and the negative values in the tails of the estimated effect distributions do not represent improvements in health as ozone concentrations are increased. Instead, these should be viewed as a measure of uncertainty due to limitations in the statistical power of the study. We included both hospital admissions and ER visits as separate endpoints associated with ozone exposure because our estimates of hospital admission costs do not include the costs of ER visits and most asthma ER visits do not result in a hospital admission.

5.4.2.5 Acute Health Events and School/Work Loss Days

In addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of ozone and PM. The sources for the effect estimates used to quantify these effects are described below.

Around 4 percent of U.S. children between the ages of 5 and 17 experience episodes of acute bronchitis annually (American Lung Association, 2002c). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia,[3] with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. Incidence of episodes of acute bronchitis in children between the ages of 5 and 17 were estimated using an effect estimate developed from Dockery et al. (1996).

[3] See http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm, accessed January 2002.

Incidences of lower respiratory symptoms (e.g., wheezing, deep cough) in children aged 7 to 14 were estimated using an effect estimate from Schwartz and Neas (2000).

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged 9 to 11 are estimated using an effect estimate developed from Pope et al. (1991).

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Days of work lost due to $PM_{2.5}$ were estimated using an effect estimate developed from Ostro (1987). Children may also be absent from school because of respiratory or other diseases caused by exposure to air pollution. Most studies examining school absence rates have found little or no association with $PM_{2.5}$, but several studies have found a significant association between ozone levels and school absence rates. We used two recent studies, Gilliland et al. (2001) and Chen et al. (2000), to estimate changes in absences (school loss days) due to changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined absence on a given day. We converted the Gilliland estimate to days of absence by multiplying the absence periods by the average duration of an absence. We estimated an average duration of school absence of 1.6 days by dividing the average daily school absence rate from Chen et al. (2000) and Ransom and Pope (1992) by the episodic absence rate from Gilliland et al. (2001). This provides estimates from Chen et al. (2000) and Gilliland et al. (2001), which can be pooled to provide an overall estimate.

Minor Restricted Activity Days (MRAD) occur when individuals reduce most usual daily activities and replace them with less strenuous activities or rest, yet not to the point of missing work or school. For example, a mechanic who would usually be doing physical work most of the day will instead spend the day at a desk doing paper and phone work because of difficulty breathing or chest pain. The effect of $PM_{2.5}$ and ozone on MRAD was estimated using an effect estimate derived from Ostro and Rothschild (1989).

For this analysis, we have followed the SAB-HES recommendations regarding asthma exacerbations in developing the primary estimate. To prevent double-counting, we focused the estimation on asthma exacerbations occurring in children and excluded adults from the

calculation.[4] Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, we would likely double-count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001; Vedal et al., 1998) that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between $PM_{2.5}$, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM_{10} and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly

[4] Estimating asthma exacerbations associated with air pollution exposures is difficult, due to concerns about double counting of benefits. Concerns over double counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double counting. In one specific case (upper respiratory symptoms in children), the only study available is limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double counting, especially if they evaluate similar endpoints. The SAB-HES, in commenting on the analytical blueprint for 812, acknowledged these challenges in evaluating asthmatic symptoms and appropriately adding them into the primary analysis (SAB-HES, 2004). However, despite these challenges, the SAB-HES recommends the addition of asthma-related symptoms (i.e., asthma exacerbations) to the primary analysis, provided that the studies use the panel study approach and that they have comparable design and baseline frequencies in both asthma prevalence and exacerbation rates. Note also, that the SAB-HES, while supporting the incorporation of asthma exacerbation estimates, does not believe that the association between ambient air pollution, including ozone and PM, and the new onset of asthma is sufficiently strong to support inclusion of this asthma-related endpoint in the primary estimate.

defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

5.4.3 Baseline Incidence Estimates

Epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the *relative risk* of a health effect, rather than estimating the absolute number of avoided cases. For example, a typical result might be that a 10 ppb decrease in daily ozone levels might, in turn, decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. A baseline incidence rate is the estimate of the number of cases of the health effect per year in the assessment location, as it corresponds to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number. For example, if the baseline incidence rate is the number of cases per year per million people, that number must be multiplied by the millions of people in the total population.

Table 5-7 summarizes the sources of baseline incidence rates and provides average incidence rates for the endpoints included in the analysis. For both baseline incidence and prevalence data, we used age-specific rates where available. We applied concentration-response functions to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits. Rates for mortality, hospitalizations, asthma ER visits, and non-fatal myocardial infarction (heart attacks) have been updated since the MATS Proposal RIA, consistent with the Cross-State Air Pollution Rule RIA (U.S. EPA 2011b).

Table 5-7. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population

Endpoint	Parameter	Rates	
		Value	Source
Mortality	Daily or annual mortality rate projected to 2015	Age-, cause-, and county-specific rate	CDC Wonder (2004–2006) U.S. Census bureau
Hospitalizations	Daily hospitalization rate	Age-, region-, state-, county- and cause-specific rate	2007 HCUP data files[a]
Asthma ER Visits	Daily asthma ER visit rate	Age-, region-, state-, county- and cause-specific rate	2007 HCUP data files[a]
Chronic Bronchitis	Annual prevalence rate per person		1999 NHIS (American Lung Association, 2002b, Table 4)
	• Aged 18–44	0.0367	
	• Aged 45–64	0.0505	
	• Aged 65 and older	0.0587	
	Annual incidence rate per person	0.00378	Abbey et al. (1995, Table 3)

(continued)

Table 5-7. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population (continued)

Endpoint	Parameter	Rates	
		Value	Source
Non-fatal Myocardial Infarction (heart attacks)	Daily non-fatal myocardial infarction incidence rate per person, 18+	Age-, region-, state-, and county- specific rate	2007 HCUP data files[a]; adjusted by 0.93 for probability of surviving after 28 days (Rosamond et al., 1999)
Asthma Exacerbations	Incidence among asthmatic African-American children		Ostro et al. (2001)
	• daily wheeze • daily cough • daily dyspnea	0.076 0.067 0.037	
Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002c, Table 11)
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children[b]	0.0012	Schwartz et al. (1994, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children	0.3419	Pope et al. (1991, Table 2)
Work Loss Days	Daily WLD incidence rate per person (18–65)		1996 HIS (Adams, Hendershot, and Marano, 1999, Table 41); U.S. Bureau of the Census (2000)
	• Aged 18–24 • Aged 25–44 • Aged 45–64	0.00540 0.00678 0.00492	
School Loss Days	Rate per person per year, assuming 180 school days per year	9.9	National Center for Education Statistics (1996) and 1996 HIS (Adams et al., 1999, Table 47);
Minor Restricted-Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)

[a] Healthcare Cost and Utilization Program (HCUP) database contains individual level, state and regional-level hospital and emergency department discharges for a variety of ICD codes.

[b] Lower respiratory symptoms are defined as two or more of the following: cough, chest pain, phlegm, and wheeze.

The baseline incidence rates for hospital and emergency department visits that we applied in this analysis are an improvement over the rates we used in the proposal analysis in two ways. First, these data are newer, and so are a more recent representation of the rates at which populations of different ages, and in different locations, visit the hospital and emergency

department for illnesses that may be air pollution related. Second, these newer data are also more spatially refined. For many locations within the U.S., these data are resolved at the county- or state-level, providing a better characterization of the geographic distribution of hospital and emergency department visits. Newer and more spatially resolved incidence rates are likely to yield a more reliable estimate of air pollution-related hospitalizations and emergency department visits. Consistent with the proposal RIA, we continue to use county-level mortality rates. We have projected mortality rates such that future mortality rates are consistent with our projections of population growth (Abt Associates, 2010).

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 5-8 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years. These rates have all been updated since the MATS proposal RIA, consistent with the Cross-State Air Pollution Rule (U.S. EPA 2011b).

Table 5-8. Asthma Prevalence Rates Used for this Analysis[a]

	Asthma Prevalence Rates	
Population Group	**Value**	**Source**
All Ages	0.0780	American Lung Association (2010, Table 7)
< 18	0.0941	
5–17	0.1070	
18–44	0.0719	
45–64	0.0745	
65+	0.0716	
African American, 5 to 17	0.1776	American Lung Association (2010, Table 9)
African American, <18	0.1553	American Lung Association[b]

[a] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000/.

[b] Calculated by ALA for U.S. EPA, based on NHIS data (CDC, 2009)

5.4.4 Economic Valuation Estimates

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects for a large population. Therefore, the appropriate economic measure is WTP for changes in risk of a health effect rather than WTP for a health effect that would occur

with certainty (Freeman, 1993). Epidemiological studies generally provide estimates of the relative risks of a particular health effect that is avoided because of a reduction in air pollution. We converted those to units of avoided statistical incidence for ease of presentation. We calculated the value of avoided statistical incidences by dividing individual WTP for a risk reduction by the related observed change in risk.[5]

WTP estimates generally are not available for some health effects, such as hospital admissions. In these cases, we used the cost of treating or mitigating the effect as a primary estimate. These cost-of-illness (COI) estimates generally understate the true value of reducing the risk of a health effect, because they reflect the direct expenditures related to treatment, but not the value of avoided pain and suffering (Harrington and Portney, 1987; Berger, 1987). We provide unit values for health endpoints (along with information on the distribution of the unit value) in Tables 5-10 through 5-12. All values are in constant year 2006 dollars, adjusted for growth in real income out to 2016 using projections provided by Standard and Poor's. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real income increases. Many of the valuation studies used in this analysis were conducted in the late 1980s and early 1990s. Because real income has grown since the studies were conducted, people's willingness to pay for reductions in the risk of premature death and disease likely has grown as well. We did not adjust cost of illness-based values because they are based on current costs. Similarly, we did not adjust the value of school absences, because that value is based on current wage rates. For these two reasons, these cost of illness estimates may underestimate the economic value of avoided health impacts in 2016. The discussion below provides additional details on ozone and $PM_{2.5}$-related related endpoints.

5.4.4.1 Mortality Valuation

Following the advice of the EEAC of the SAB, EPA currently uses the VSL approach in calculating the primary estimate of mortality co-benefits, because we believe this calculation

[5] To comply with Circular A-4, EPA provides monetized benefits using discount rates of 3% and 7% (OMB, 2003). These benefits are estimated for a specific analysis year (i.e., 2016), and most of the PM benefits occur within that year with two exceptions: acute myocardial infarctions (AMIs) and premature mortality. For AMIs, we assume 5 years of follow-up medical costs and lost wages. For premature mortality, we assume that there is a "cessation" lag between PM exposures and the total realization of changes in health effects. Although the structure of the lag is uncertain, EPA follows the advice of the SAB-HES to assume a segmented lag structure characterized by 30% of mortality reductions in the first year, 50% over years 2 to 5, and 20% over the years 6 to 20 after the reduction in $PM_{2.5}$ (U.S. EPA-SAB, 2004c). Changes in the lag assumptions do not change the total number of estimated deaths but rather the timing of those deaths. Therefore, discounting only affects the AMI costs after the analysis year and the valuation of premature mortalities that occur after the analysis year. As such, the monetized benefits using a 7% discount rate are only approximately 10% less than the monetized benefits using a 3% discount rate.

provides the most reasonable single estimate of an individual's willingness to trade off money for reductions in mortality risk (U.S. EPA-SAB, 2000). The VSL approach is a summary measure for the value of small changes in mortality risk experienced by a large number of people. For a period of time (2004-2008), the Office of Air and Radiation (OAR) valued mortality risk reductions using a value of statistical life (VSL) estimate derived from a limited analysis of some of the available studies. OAR arrived at a VSL using a range of $1 million to $10 million (2000$) consistent with two meta-analyses of the wage-risk literature. The $1 million value represented the lower end of the interquartile range from the Mrozek and Taylor (2002) meta-analysis of 33 studies. The $10 million value represented the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis of 43 studies. The mean estimate of $5.5 million (2000$) was also consistent with the mean VSL of $5.4 million estimated in the Kochi et al. (2006) meta-analysis. However, the Agency neither changed its official guidance on the use of VSL in rule-makings nor subjected the interim estimate to a scientific peer-review process through the Science Advisory Board (SAB) or other peer-review group.

During this time, the Agency continued work to update its guidance on valuing mortality risk reductions, including commissioning a report from meta-analytic experts to evaluate methodological questions raised by EPA and the SAB on combining estimates from the various data sources. In addition, the Agency consulted several times with the Science Advisory Board Environmental Economics Advisory Committee (SAB-EEAC) on the issue. With input from the meta-analytic experts, the SAB-EEAC advised the Agency to update its guidance using specific, appropriate meta-analytic techniques to combine estimates from unique data sources and different studies, including those using different methodologies (i.e., wage-risk and stated preference) (U.S. EPA-SAB, 2007).

Until updated guidance is available, the Agency determined that a single, peer-reviewed estimate applied consistently best reflects the SAB-EEAC advice it has received. Therefore, the Agency has decided to apply the VSL that was vetted and endorsed by the SAB in the Guidelines for Preparing Economic Analyses (U.S. EPA, 2000)[6] while the Agency continues its efforts to update its guidance on this issue. This approach calculates a mean value across VSL estimates derived from 26 labor market and contingent valuation studies published between 1974 and

[6] In EPA's recently revised *Economic Guidelines* (U.S. EPA, 2010d), EPA retained the VSL endorsed by the SAB with the understanding that further updates to the mortality risk valuation guidance would be forthcoming in the near future. Therefore, this report does not represent final agency policy.

1991. The mean VSL across these studies is $6.3 million (2000$).[7] The Agency is committed to using scientifically sound, appropriately reviewed evidence in valuing mortality risk reductions and has made significant progress in responding to the SAB-EEAC's specific recommendations. The Agency anticipates presenting results from this effort to the SAB-EEAC in Spring 2010 and that draft guidance will be available shortly thereafter.

As indicated in the previous section on quantification of premature mortality benefits, we assumed for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the 20 years following exposure. To take this into account in the valuation of reductions in premature mortality, we applied an annual 3% discount rate to the value of premature mortality occurring in future years.[8]

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economics and public policy analysis community. EPA strives to use the best economic science in its analyses. Given the mixed theoretical finding and empirical evidence regarding adjustments to VSL for risk and population characteristics, we use a single VSL for all reductions in mortality risk.

Although there are several differences between the labor market studies EPA uses to derive a VSL estimate and the PM air pollution context addressed here, those differences in the affected populations and the nature of the risks imply both upward and downward adjustments. Table 5-9 lists some of these differences and the expected effect on the VSL estimate for air pollution-related mortality. In the absence of a comprehensive and balanced set of adjustment factors, EPA believes it is reasonable to continue to use the $6.3 million value while acknowledging the significant limitations and uncertainties in the available literature.

---

[7] In this analysis, we adjust the VSL to account for a different currency year (2007$) and to account for income growth to 2016. After applying these adjustments to the $6.3 million value, the VSL is $8.9M.

[8] The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. EPA adopted a 3% discount rate for its base estimate in this case to reflect reliance on a "social rate of time preference" discounting concept. We have also calculated benefits and costs using a 7% rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. In this case, the benefit and cost estimates were not significantly affected by the choice of discount rate. Further discussion of this topic appears in EPA's *Guidelines for Preparing Economic Analyses* (EPA, 2010).

Table 5-9. Expected Impact on Estimated Benefits of Premature Mortality Reductions of Differences Between Factors Used in Developing Applied VSL and Theoretically Appropriate VSL

Attribute	Expected Direction of Bias
Age	Uncertain, perhaps overestimate
Life Expectancy/Health Status	Uncertain, perhaps overestimate
Attitudes Toward Risk	Underestimate
Income	Uncertain
Voluntary vs. Involuntary	Uncertain, perhaps underestimate
Catastrophic vs. Protracted Death	Uncertain, perhaps underestimate

The SAB-EEAC has reviewed many potential VSL adjustments and the state of the economics literature. The SAB-EEAC advised EPA to "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk" (U.S. EPA, 2000). In developing our primary estimate of the co-benefits of premature mortality reductions, we have followed this advice and discounted over the lag period between exposure and premature mortality.

Uncertainties Specific to Premature Mortality Valuation. The economic co-benefits associated with reductions in the risk of premature mortality are the largest category of monetized co-benefits of the MATS. In addition, in prior analyses, EPA has identified valuation of mortality-related benefits as the largest contributor to the range of uncertainty in monetized benefits (U.S. EPA, 1999).[9] Because of the uncertainty in estimates of the value of reducing premature mortality risk, it is important to adequately characterize and understand the various types of economic approaches available for valuing reductions in mortality risk. Such an assessment also requires an understanding of how alternative valuation approaches reflect that some individuals may be more susceptible to air pollution-induced mortality or reflect differences in the nature of the risk presented by air pollution relative to the risks studied in the relevant economics literature.

[9] This conclusion was based on an assessment of uncertainty based on statistical error in epidemiological effect estimates and economic valuation estimates. Additional sources of model error such as those examined in the PM mortality expert elicitation may result in different conclusions about the relative contribution of sources of uncertainty.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's WTP to improve one's own chances of survival plus WTP to improve other individuals' survival rates. The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves depend on such characteristics as age, health state, and the current age to which the individual is likely to survive.

Although a survival curve approach provides a theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value reductions in premature mortality risk using the VSL approach.

Other uncertainties specific to premature mortality valuation include the following:

- *Across-study variation:* There is considerable uncertainty as to whether the available literature on VSL provides adequate estimates of the VSL for risk reductions from air pollution reduction. Although there is considerable variation in the analytical designs and data used in the existing literature, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a hedonic wage approach. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the average WTP to reduce the risk. The appropriateness of a distribution of WTP based on the current VSL literature for valuing the mortality-related benefits of reductions in air pollution concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on the extent to which the risks

being valued are similar and the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations.

- *Level of risk reduction:* The transferability of estimates of the VSL from the wage-risk studies to the context of the PM NAAQS analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. For example, suppose a study provides a result that the average WTP for a reduction in mortality risk of 1/100,000 is $50, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $50 for a reduction of 1/100,000 implies a WTP of $500 for a risk reduction of 1/10,000 (which is 10 times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the VSL does not depend on the particular amount of risk reduction being valued. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al., 1998).

- *Voluntariness of risks evaluated:* Although job-related mortality risks may differ in several ways from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily, or generally assumed to be, whereas air pollution-related risks are incurred involuntarily. Some evidence suggests that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may understate WTP to reduce involuntarily incurred air pollution-related mortality risks.

- *Sudden versus protracted death:* A final important difference related to the nature of the risk may be that some workplace mortality risks tend to involve sudden, catastrophic events, whereas air pollution-related risks tend to involve longer periods of disease and suffering prior to death. Some evidence suggests that WTP to avoid a risk of a protracted death involving prolonged suffering and loss of dignity and personal control is greater than the WTP to avoid a risk (of identical magnitude) of sudden death. To the extent that the mortality risks addressed in this assessment are associated with longer periods of illness or greater pain and suffering than are the risks addressed in the valuation literature, the WTP measurements employed in the present analysis would reflect a downward bias.

- *Self-selection and skill in avoiding risk:* Recent research (Shogren and Stamland, 2002) suggests that VSL estimates based on hedonic wage studies may overstate the average value of a risk reduction. This is based on the fact that the risk-wage trade-off revealed in hedonic studies reflects the preferences of the marginal worker (i.e., that worker who demands the highest compensation for his risk reduction). This worker must have either a higher workplace risk than the average worker, a lower risk tolerance than the average worker, or both. However, the risk estimate used in hedonic studies is generally based on average risk, so the VSL may be upwardly biased because the wage differential and risk measures do not match.

- *Baseline risk and age:* Recent research (Smith, Pattanayak, and Van Houtven, 2006) finds that because individuals reevaluate their baseline risk of death as they age, the marginal value of risk reductions does not decline with age as predicted by some lifetime consumption models. This research supports findings in recent stated preference studies that suggest only small reductions in the value of mortality risk reductions with increasing age.

5.4.4.2 Chronic Bronchitis Valuation

The best available estimate of WTP to avoid a case of CB comes from Viscusi, Magat, and Huber (1991). The Viscusi, Magat, and Huber study, however, describes a severe case of CB to the survey respondents. We therefore employ an estimate of WTP to avoid a pollution-related case of CB, based on adjusting the Viscusi, Magat, and Huber (1991) estimate of the WTP to avoid a severe case. This is done to account for the likelihood that an average case of pollution-related CB is not as severe. The adjustment is made by applying the elasticity of WTP with respect to severity reported in the Krupnick and Cropper (1992) study. Details of this adjustment procedure are provided in the *Benefits Technical Support Document (TSD)* for the Nonroad Diesel rulemaking (Abt Associates, 2003).

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of CB in this analysis. The distribution incorporates uncertainty from three sources: the WTP to avoid a case of severe CB, as described by Viscusi, Magat, and Huber; the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi, Magat, and Huber); and the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollution-related case of CB by statistical uncertainty analysis techniques. The expected value (i.e., mean) of this

distribution, which is about $340,000 (2006$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

5.4.4.3 Non-fatal Myocardial Infarctions Valuation

We were not able to identify a suitable WTP value for reductions in the risk of non-fatal heart attacks. Instead, we use a COI unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3% discount rate, we estimated a present discounted value in lost earnings (in 2006$) over 5 years due to a myocardial infarction of $8,774 for someone between the ages of 25 and 44, $12,932 for someone between the ages of 45 and 54, and $74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2006$) using a 7% discount rate are $7,855, $11,578, and $66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. As such, we do not include lost earnings in the cost estimates for these age groups.

We found three possible sources in the literature of estimates of the direct medical costs of myocardial infarction:

- Wittels et al. (1990) estimated expected total medical costs of myocardial infarction over 5 years to be $51,211 (in 1986$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Wittels et al. was used to value coronary heart disease in the 812 Retrospective Analysis of the Clean Air Act. Using the CPI-U for medical care, the Wittels estimate is $144,111 in year 2006$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The authors note that the average length of hospitalization for acute myocardial infarction has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay for myocardial infarction may have decreased from 1983 to the present. The average length of stay for ICD code 410 (myocardial infarction) in the year-2000 Agency for Healthcare Research and Quality (AHRQ) HCUP database is 5.5 days.

However, this may include patients who died in the hospital (not included among our non-fatal myocardial infarction cases), whose length of stay was therefore substantially shorter than it would be if they had not died.

- Eisenstein et al. (2001) estimated 10-year costs of $44,663 in 1997$, or $64,003 in 2006$ for myocardial infarction patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.

- Russell et al. (1998) estimated first-year direct medical costs of treating non-fatal myocardial infarction of $15,540 (in 1995$) and $1,051 annually thereafter. Converting to year 2006$, that would be $30,102 for a 5-year period (without discounting) or $38,113 for a 10-year period.

In summary, the three different studies provided significantly different values (see Table 5-10).

Table 5-10. Alternative Direct Medical Cost of Illness Estimates for Non-fatal Heart Attacks

Study	Direct Medical Costs (2006$)	Over an x-Year Period, for x =
Wittels et al. (1990)	144,111[a]	5
Russell et al. (1998)	30,102[b]	5
Eisenstein et al. (2001)	64,003[b]	10
Russell et al. (1998)	$38,113[b]	10

[a] Wittels et al. (1990) did not appear to discount costs incurred in future years.

[b] Using a 3% discount rate. Discounted values as reported in the study.

As noted above, the estimates from these three studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period (i.e., estimates from Wittels et al. (1990) and Russell et al. (1998). We used a simple average of the two 5-year estimates, or $65,902, and added it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 5-11.

Table 5-11. Estimated Costs Over a 5-Year Period (in 2006$) of a Non-fatal Myocardial Infarction

	Cost, $		
Age Group	**Opportunity**	**Medical[a]**	**Total**
0–24	0	84,955	84,955
25–44	10,757[b]	84,955	95,713
45–54	15,855[b]	84,955	100,811
55–65	91,647[b]	84,955	176,602
> 65	0	84,955	84,955

[a] An average of the 5-year costs estimated by Wittels et al. (1990) and Russell et al. (1998).

[b] From Cropper and Krupnick (1990), using a 3% discount rate.

5.4.5 Hospital Admissions Valuation

In the absence of estimates of societal WTP to avoid hospital visits/admissions for specific illnesses, estimates of total cost of illness (total medical costs plus the value of lost productivity) typically are used as conservative, or lower bound, estimates. These estimates are biased downward, because they do not include the willingness-to-pay value of avoiding pain and suffering.

The International Classification of Diseases (ICD-9, WHO 1977) code-specific COI estimates used in this analysis consist of estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). We based all estimates of hospital charges and length of stays on statistics provided by the Agency for Healthcare Research and Quality (AHRQ 2000). We estimated the opportunity cost of a day spent in the hospital as the value of the lost daily wage, regardless of whether the hospitalized individual is in the workforce. To estimate the lost daily wage, we divided the 1990 median weekly wage by five and inflated the result to year 2006$ using the CPI-U "all items." The resulting estimate is $127.93. The total cost-of-illness estimate for an ICD code-specific hospital stay lasting *n* days, then, was the mean hospital charge plus $127.93 multiplied by *n.*

5.4.5.1 Asthma-Related Emergency Room Visits Valuation

To value asthma emergency room visits, we used a simple average of two estimates from the health economics literature. The first estimate comes from Smith et al. (1997), who reported approximately 1.2 million asthma-related emergency room visits in 1987, at a total cost of $186.5 million (1987$). The average cost per visit that year was $155; in 2006$, that cost

was $400.88 (using the CPI-U for medical care to adjust to 2006$). The second estimate comes from Stanford et al. (1999), who reported the cost of an average asthma-related emergency room visit at $335.14, based on 1996–1997 data. A simple average of the two estimates yields a (rounded) unit value of $368.

5.4.5.2 Minor Restricted Activity Days Valuation

No studies are reported to have estimated WTP to avoid a minor restricted activity day. However, one of EPA's contractors, IEc (1994) has derived an estimate of willingness to pay to avoid a minor *respiratory* restricted activity day, using estimates from Tolley et al. (1986) of WTP for avoiding a combination of coughing, throat congestion and sinusitis. The IEc estimate of WTP to avoid a minor respiratory restricted activity day is $38.37 (1990$), or about $62.04 (2006$).

Table 5-12. Unit Values for Economic Valuation of Health Endpoints (2006$)[a]

Health Endpoint	Central Estimate of Value Per Statistical Incidence, Income Level		Derivation of Distributions of Estimates
	2000	2016	
Premature Mortality (Value of a Statistical Life)	$6,300,000	$8,600,000	EPA currently recommends a central VSL of $6.3m (2000$) based on a Weibull distribution fitted to 26 published VSL estimates (5 contingent valuation and 21 labor market studies). The underlying studies, the distribution parameters, and other useful information are available in Appendix 5B of EPA's current Guidelines for Preparing Economic Analyses (U.S. EPA, 2000).
Chronic Bronchitis (CB)	$340,000	$470,000	The WTP to avoid a case of pollution-related CB is calculated as where x is the severity of an average CB case, WTP_{13} is the WTP for a severe case of CB, and $ is the parameter relating WTP to severity, based on the regression results reported in Krupnick and Cropper (1992). The distribution of WTP for an average severity-level case of CB was generated by Monte Carlo methods, drawing from each of three distributions: (1) WTP to avoid a severe case of CB is assigned a 1/9 probability of being each of the first nine deciles of the distribution of WTP responses in Viscusi et al. (1991); (2) the severity of a pollution-related case of CB (relative to the case described in the Viscusi study) is assumed to have a triangular distribution, with the most likely value at severity level 6.5 and endpoints at 1.0 and 12.0; and (3) the constant in the elasticity of WTP with respect to severity is normally distributed with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper [1992]). This process and the rationale for choosing it is described in detail in the Costs and Benefits of the Clean Air Act, 1990 to 2010 (U.S. EPA, 1999).

(continued)

Table 5-12. Unit Values for Economic Valuation of Health Endpoints (2006$) (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence, Income Level 2000	2016	Derivation of Distributions of Estimates
Non-fatal Myocardial Infarction (heart attack)			No distributional information available. Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year period following a non-fatal MI. Lost earnings estimates are based on Cropper and Krupnick (1990). Direct medical costs are based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990). Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings: age of onset: at 3% at 7% 25–44 $8,774 $7,855 45–54 $12,932 11,578 55–65 $74,746 66,920 Direct medical expenses: An average of: 1. Wittels et al. (1990) ($102,658—no discounting) 2. Russell et al. (1998), 5-year period ($22,331 at 3% discount rate; $21,113 at 7% discount rate)
3% discount rate			
Age 0–24	$79,685	$79,685	
Age 25–44	$88,975	$88,975	
Age 45–54	$93,897	$93,897	
Age 55–65	$167,532	$167,532	
Age 66 and over	$79,685	$79,685	
7% discount rate			
Age 0–24	$77,769	$77,769	
Age 25–44	$87,126	$87,126	
Age 45–54	$91,559	$91,559	
Age 55–65	$157,477	$157,477	
Age 66 and over	$77,769	$77,769	
Hospital Admissions			
Chronic Obstructive Pulmonary Disease (COPD)	$16,606	$16,606	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
Asthma Admissions	$8,900	$8,900	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).

(continued)

Table 5-12. Unit Values for Economic Valuation of Health Endpoints (2006$) (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence, Income Level		Derivation of Distributions of Estimates
	2000	2016	
All Cardiovascular	$24,668	$24,668	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
All respiratory (ages 65+)	$24,622	$24,622	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
All respiratory (ages 0–2)	$10,385	$10,385	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Emergency Room Visits for Asthma	$384	$384	No distributional information available. Simple average of two unit COI values: (1) $311.55, from Smith et al. (1997) and (2) $260.67, from Stanford et al. (1999).

(continued)

Table 5-12. Unit Values for Economic Valuation of Health Endpoints (2006$) (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence, Income Level 2000	2016	Derivation of Distributions of Estimates
Respiratory Ailments Not Requiring Hospitalization			
Upper Respiratory Symptoms (URS)	$30	$30	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, we assumed a uniform distribution between $9.2 and $43.1.
Lower Respiratory Symptoms (LRS)	$16	$19	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS. In the absence of information surrounding the frequency with which each of the 11 types of LRS occurs within the LRS symptom complex, we assumed a uniform distribution between $6.9 and $24.46.
Asthma Exacerbations	$43	$53	Asthma exacerbations are valued at $45 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between $15.6 and $70.8.

(continued)

Table 5-12. Unit Values for Economic Valuation of Health Endpoints (2006$) (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence, Income Level 2000	2016	Derivation of Distributions of Estimates
Acute Bronchitis	$360	$440	Assumes a 6-day episode, with the distribution of the daily value specified as uniform with the low and high values based on those recommended for related respiratory symptoms in Neumann et al. (1994). The low daily estimate of $10 is the sum of the mid-range values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high daily estimate was taken to be twice the value of a minor respiratory restricted-activity day, or $110.
Work Loss Days (WLDs)	Variable (U.S. median = $130)	Variable (U.S. median = $130)	No distribution available. Point estimate is based on county-specific median annual wages divided by 52 and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
Minor Restricted Activity Days (MRADs)	$51	$62	Median WTP estimate to avoid one MRAD from Tolley et al. (1986). Distribution is assumed to be triangular with a minimum of $22 and a maximum of $83, with a most likely value of $52. Range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom—for eye irritation—is $16.00) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme.

[a]Values reported in this table are in 2006$, but we used 2007$ for this analysis. Inflating to 2007$ would increase the values approximately 2.8% for WTP estimates up to 4.4% for COI estimates.

Although Ostro and Rothschild (1989) statistically linked ozone and minor restricted activity days, it is likely that most MRADs associated with ozone exposure are, in fact, minor *respiratory* restricted activity days. For the purpose of valuing this health endpoint, we used the estimate of mean WTP to avoid a minor respiratory restricted activity day.

5.4.5.3 Growth in WTP Reflecting National Income Growth Over Time

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes

increase. There is substantial empirical evidence that the income elasticity[10] of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases, the WTP for environmental improvements also increases. Although many analyses assume that the income elasticity of WTP is unit elastic (i.e., a 10% higher real income level implies a 10% higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefits estimates in two different ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the Science Advisory Board (SAB) advised EPA to adjust WTP for increases in real income over time but not to adjust WTP to account for cross-sectional income differences "because of the sensitivity of making such distinctions, and because of insufficient evidence available at present" (U.S. EPA-SAB, 2000). A recent advisory by another committee associated with the SAB, the Advisory Council on Clean Air Compliance Analysis, has provided conflicting advice. While agreeing with "the general principle that the willingness to pay to reduce mortality risks is likely to increase with growth in real income (U.S. EPA-SAB, 2004b, p. 52)" and that "The same increase should be assumed for the WTP for serious non-fatal health effects (U.S. EPA-SAB, 2004b, p. 52)," they note that "given the limitations and uncertainties in the available empirical evidence, the Council does not support the use of the proposed adjustments for aggregate income growth as part of the primary analysis" (U.S. EPA-SAB, 2004b, p. 53). Until these conflicting advisories have been reconciled, EPA will continue to adjust valuation estimates to reflect income growth using the methods described below, while providing sensitivity analyses for alternative income growth adjustment factors.

Based on a review of the available income elasticity literature, we adjusted the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be

[10] Income elasticity is a common economic measure equal to the percentage change in WTP for a 1% change in income.

found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2016 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. Note that because of the variety of empirical sources used in deriving the income elasticities, there may appear to be inconsistencies in the magnitudes of the income elasticities relative to the severity of the effects (*a priori* one might expect that more severe outcomes would show less income elasticity of WTP). We have not imposed any additional restrictions on the empirical estimates of income elasticity. One explanation for the seeming inconsistency is the difference in timing of conditions. WTP for minor illnesses is often expressed as a short term payment to avoid a single episode. WTP for major illnesses and mortality risk reductions are based on longer term measures of payment (such as wages or annual income). Economic theory suggests that relationships become more elastic as the length of time grows, reflecting the ability to adjust spending over a longer time period. Based on this theory, it would be expected that WTP for reducing long term risks would be more elastic than WTP for reducing short term risks. We also expect that the WTP for improved visibility in Class I areas would increase with growth in real income. The relative magnitude of the income elasticity of WTP for visibility compared with those for health effects suggests that visibility is not as much of a necessity as health, thus, WTP is more elastic with respect to income. The elasticity values used to adjust estimates of benefits in 2016 are presented in Table 5-13.

Table 5-13. Elasticity Values Used to Account for Projected Real Income Growth[a]

Benefit Category	Central Elasticity Estimate
Minor Health Effect	0.14
Severe and Chronic Health Effects	0.45
Premature Mortality	0.40
Visibility	0.90

[a] Derivation of estimates can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

In addition to elasticity estimates, projections of real gross domestic product (GDP) and populations from 1990 to 2020 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, we used national population

estimates for the years 1990 to 1999 based on U.S. Census Bureau estimates (Hollman, Mulder, and Kallan, 2000). These population estimates are based on application of a cohort-component model applied to 1990 U.S. Census data projections (U.S. Bureau of Census, 2000). For the years between 2000 and 2016, we applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. We used projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.[11] We used projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's (2000) for the years 2010 to 2016.[12]

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, we calculated WTP adjustment factors for each of the elasticity estimates listed in Table 5-14. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) are adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Note that, for premature mortality, we applied the income adjustment factor to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that because of a lack of data on the dependence of COI and income, and a lack of data on projected growth in average wages, no adjustments are made to benefits based on the COI approach or to work loss days and worker productivity. This assumption leads us to underpredict benefits in future years because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

[11] U.S. Bureau of Economic Analysis, Table 2A (1992$) (available at http://www.bea.doc.gov/bea/dn/0897nip2/tab2a.htm.) and U.S. Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

[12] In previous analyses, we used the Standard and Poor's projections of GDP directly. This led to an apparent discontinuity in the adjustment factors between 2010 and 2011. We refined the method by applying the relative growth rates for GDP derived from the Standard and Poor's projections to the 2010 projected GDP based on the Bureau of Economic Analysis projections.

Table 5-14. Adjustment Factors Used to Account for Projected Real Income Growth[a]

Benefit Category	2016
Minor Health Effect	1.06
Severe and Chronic Health Effects	1.19
Premature Mortality	1.16
Visibility	1.41

[a] Based on elasticity values reported in Table 5-13, U.S. Census population projections, and projections of real GDP per capita.

5.5 Unquantified Health and Welfare Benefits

This analysis is limited by the available data and resources. As such, we are not able to quantify several welfare benefit categories, as shown in Tables 5-3 and 5-4. This section provides an overview of what is meant by ecosystem services as well as a description of visibility benefits, which are typically assessed and monetized in relevant RIAs but that were not quantified in this benefits analysis. The RIA for the final Cross-State Air Pollution Rule (U.S. EPA, 2011b) provides more information on additional major health and welfare benefit categories associated with reducing NO_2 and SO_2 emissions including: health and ecosystem benefits of reducing nitrogen and sulfur emissions and deposition; vegetation benefits from reducing ozone; mercury benefits associated with reducing mercury emissions; and the role of sulfate deposition in mercury methylation. While we are unable to quantify these benefits, previous relevant EPA assessments show that these benefits could be substantial (U.S. EPA, 2008a; U.S. EPA, 2009a; U.S. EPA, 2007; U.S. EPA, 1999, U.S. EPA, 2011b). The omission of these endpoints from the monetized results should not imply that the impacts are small or unimportant.

5.5.1 Visibility Valuation

Reductions in NO_2 and SO_2 emissions along with the secondary formation of $PM_{2.5}$ would improve the level of visibility throughout the United States because these suspended particles and gases degrade visibility by scattering and absorbing light (U.S. EPA, 2009a). Visibility has direct significance to people's enjoyment of daily activities and their overall sense of wellbeing (U.S. EPA, 2009a). Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Great Smokey Mountains National Park. This section discusses the measurement of the economic benefits of improved visibility. As there is no analogous approach for estimating visibility benefits using the BPT approach, visibility benefits are calculated for the modeled interim policy scenario only and are not included in the co-benefits estimate of the final policy. However,

since the magnitude of SO_2 emission reductions did not significantly change in the visibility study areas between the interim and final emissions scenarios, we expect the visibility benefit for the final policy scenario would be similar to that calculated for the interim policy scenario ($1.1 billion in total for the U.S., using 2007$; see Appendix 5C).

Visual air quality (VAQ) is commonly measured as either light extinction, which is defined as the loss of light per unit of distance in terms of inverse megameters (Mm^{-1}) or the deciview (dv) metric (Pitchford and Malm, 1993), which is a logarithmic function of extinction. Extinction and deciviews are physical measures of the amount of visibility impairment (e.g., the amount of "haze"), with both extinction and deciview increasing as the amount of haze increases. Pitchford and Malm characterize a change of one deciview as "a small but perceptible scenic change under many circumstances." Light extinction is the optical characteristic of the atmosphere that occurs when light is either scattered or absorbed, which converts the light to heat. Particulate matter and gases can both scatter and absorb light. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon, and soil (Sisler, 1996). The extent to which any amount of light extinction affects a person's ability to view a scene depends on both scene and light characteristics. For example, the appearance of a nearby object (i.e. a building) is generally less sensitive to a change in light extinction than the appearance of a similar object at a greater distance. See Figure 5-3 for an illustration of the important factors affecting visibility.

In conjunction with the U.S. National Park Service, the U.S. Forest Service, other Federal land managers, and State organizations in the U.S., the U.S. EPA has supported visibility monitoring in national parks and wilderness areas since 1988. The monitoring network known as IMPROVE (Interagency Monitoring of Protected Visual Environments) now includes 150 sites that represent almost all of the Class I areas across the country (see Figure 5-4) (U.S. EPA, 2009a).

Annual average visibility conditions (reflecting light extinction due to both anthropogenic and non-anthropogenic sources) vary regionally across the U.S. (U.S. EPA, 2009a). The rural East generally has higher levels of impairment than remote sites in the West, with the exception of urban-influenced sites such as San Gorgonio Wilderness (CA) and Point Reyes National Seashore (CA), which have annual average levels comparable to certain sites in the Northeast (U.S. EPA, 2004). Higher visibility impairment levels in the East are due to generally higher concentrations of fine particles, particularly sulfates, and higher average relative humidity levels. While visibility trends have improved in most Class I areas, the recent data show that these areas continue to suffer from visibility impairment. In eastern parks,

average visual range has decreased from 90 miles to 15-25 miles, and in the West, visual range has decreased from 140 miles to 35-90 miles (U.S. EPA, 2004; U.S. EPA, 1999).

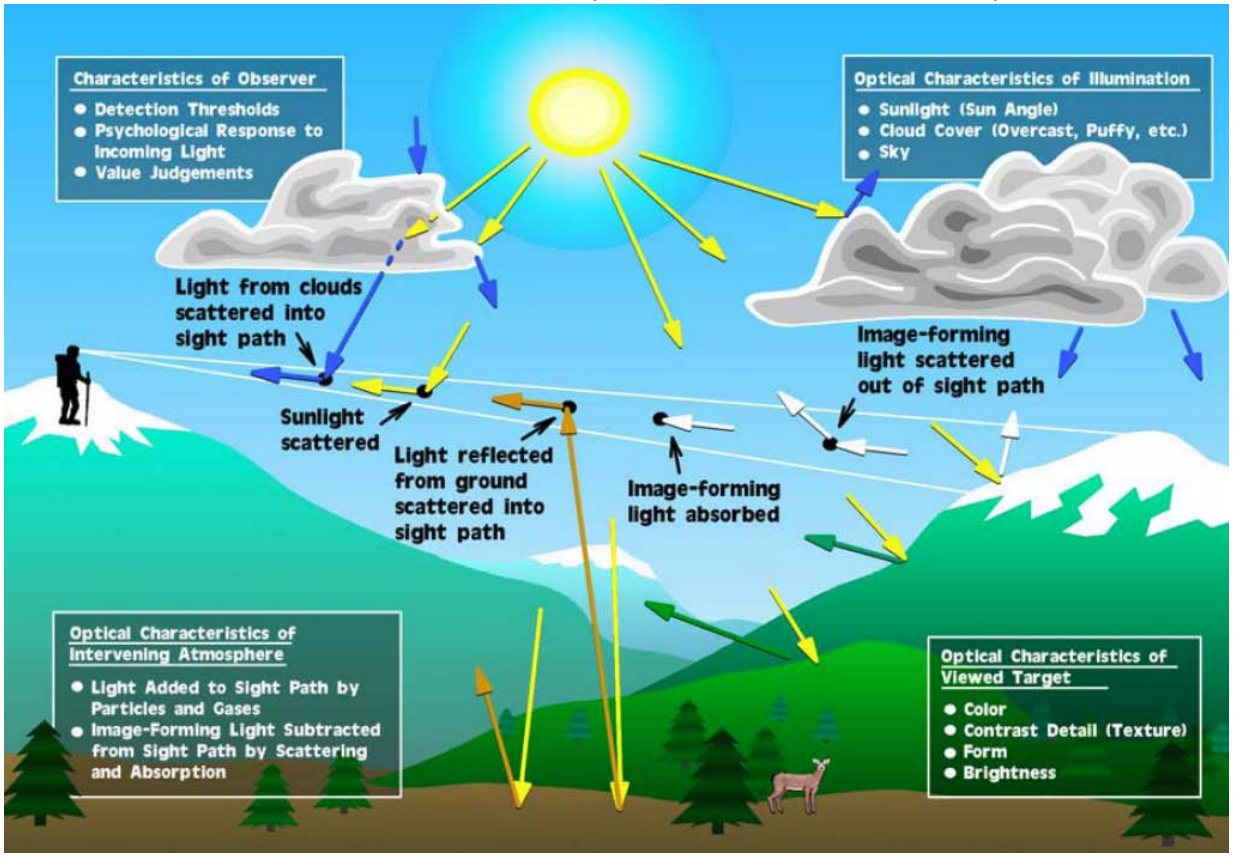

Figure 5-3. Important Factors Involved in Seeing a Scenic Vista (Malm, 1999)

Figure 5-4. Mandatory Class I Areas in the U.S.

EPA distinguishes benefits from two categories of visibility changes: residential visibility and recreational visibility. In both cases economic benefits are believed to consist of use values and nonuse values. Use values include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and birdwatching. Nonuse values are based on people's beliefs that the environment ought to exist free of human-induced haze. Nonuse values may be more important for recreational areas, particularly national parks and monuments.

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas. In previous assessments, EPA used a study on residential visibility valuation conducted in 1990 (McClelland et al., 1993). Subsequently, EPA designated the McClelland et al. study as significantly less reliable for regulatory benefit-cost analysis consistent with SAB advice (U.S. EPA-SAB, 1999). Although a wide range of published, peer-review literature supports a non-zero value for residential visibility (Brookshire et al., 1982; Rae, 1983; Tolley et al., 1986; Chestnut and Rowe, 1990c; McClelland et al., 1993; Loehman et al., 1994), the residential visibility benefits have not been calculated in this analysis.

For recreational visibility, only one existing study provides defensible monetary estimates of the value of visibility changes in a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). Although there are a number of other studies in the literature, they were conducted in the early 1980s and did not use methods that are considered defensible by current standards. The Chestnut and Rowe study uses the CV method. There has been a great deal of controversy and significant development of both theoretical and empirical knowledge about how to conduct CV surveys in the past decade. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas.[13] This study serves as an essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates.

For the purposes of the analysis of the visibility benefits of the modeled interim policy (Appendix 5C), recreational visibility improvements are defined as those that occur specifically in federal Class I areas.[14] A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from residential visibility, while all households in the United States are assumed to derive some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.[15] The Chestnut and Rowe study measured the demand for visibility in Class I areas managed by the National Park Service (NPS) in three broad regions of the country: California, the Southwest, and the Southeast. Respondents in five states were asked about their WTP to protect national parks or NPS-managed wilderness areas within a particular region. The survey used photographs reflecting different visibility levels in the specified recreational areas. The visibility levels in these photographs were later converted to deciviews for the current analysis. The survey data collected were used to estimate a WTP equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

[13] In SAB advisory letter indicates that "many members of the Council believe that the Chestnut and Rowe study is the best available" (EPA-SAB-COUNCIL-ADV-00-002, 1999, p. 13). However, the committee did not formally approve use of these estimates because of concerns about the peer-reviewed status of the study. EPA believes the study has received adequate review and has been cited in numerous peer-reviewed publications (Chestnut and Dennis, 1997).

[14] The Clean Air Act designates 156 national parks and wilderness areas as Class I areas for visibility protection.

[15] For details of the visibility estimates discussed in this chapter, please refer to the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the United States. We can infer the value of visibility changes in the other Class I areas by transferring values of visibility changes at Class I areas in the study regions. A complete description of the benefits transfer method used to infer values for visibility changes in Class I areas outside the study regions is provided in the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

The Chestnut and Rowe study (Chestnut and Rowe, 1990a; 1990b), although representing the best available estimates, has a number of limitations. These include the following:

- The age of the study (late 1980s) will increase the uncertainty about the correspondence of the estimated values to those that might be provided by current or future populations.

- The survey focused only on populations in five states, so the application of the estimated values to populations outside those states requires that preferences of populations in the five surveyed states be similar to those of non-surveyed states.

- There is an inherent difficulty in separating values expressed for visibility improvements from an overall value for improved air quality. The Chestnut and Rowe study attempted to control for this by informing respondents that "other households are being asked about visibility, human health, and vegetation protections in urban areas and at national parks in other regions." However, most of the respondents did not feel that they were able to segregate visibility at national parks entirely from residential visibility and health effects.

- It is not clear exactly what visibility improvements the respondents to the Chestnut and Rowe survey were valuing. The WTP question asked about changes in average visibility, but the survey respondents were shown photographs of only summertime conditions, when visibility is generally at its worst. It is possible that the respondents believed those visibility conditions held year-round, in which case they would have been valuing much larger overall improvements in visibility than what otherwise would be the case. For the purpose of the benefits analysis for this rule, EPA assumed that respondents provided values for changes in annual average visibility. Because most policies will result in a shift in the distribution of visibility (usually

affecting the worst days more than the best days), the annual average may not be the most relevant metric for policy analysis.

- The survey did not include reminders of possible substitutes (e.g., visibility at other parks) or budget constraints. These reminders are considered to be best practice for stated preference surveys.

- The Chestnut and Rowe survey focused on visibility improvements in and around national parks and wilderness areas. The survey also focused on visibility improvements of national parks in the southwest United States. Given that national parks and wilderness areas exhibit unique characteristics, it is not clear whether the WTP estimate obtained from Chestnut and Rowe can be transferred to other national parks and wilderness areas, without introducing additional uncertainty.

In general, the survey design and implementation reflect the period in which the survey was conducted. Since that time, many improvements to the stated preference methodology have been developed. As future survey efforts are completed, EPA will incorporate values for visibility improvements reflecting the improved survey designs.

The estimated relationship from the Chestnut and Rowe study is only directly applicable to the populations represented by survey respondents. EPA used benefits transfer methodology to extrapolate these results to the population affected by the reductions in precursor emissions associated with this rule. A general WTP equation for improved visibility (measured in deciviews) was developed as a function of the baseline level of visibility, the magnitude of the visibility improvement, and household income. The behavioral parameters of this equation were taken from analysis of the Chestnut and Rowe data. These parameters were used to calibrate WTP for the visibility changes resulting from this rule. The method for developing calibrated WTP functions is based on the approach developed by Smith et al. (2002). Available evidence indicates that households are willing to pay more for a given visibility improvement as their income increases (Chestnut, 1997). The benefits estimates here incorporate Chestnut's estimate that a 1% increase in income is associated with a 0.9% increase in WTP for a given change in visibility. A more detailed explanation of the visibility benefits methodology is provided in Appendix I of the PM NAAQS RIA (U.S. EPA, 2006a).

One major source of uncertainty for the visibility benefits estimate is the benefits transfer process used. Judgments used to choose the functional form and key parameters of the estimating equation for WTP for the affected population could have significant effects on

the size of the estimates. Assumptions about how individuals respond to changes in visibility that are either very small or outside the range covered in the Chestnut and Rowe study could also affect the results.

In addition, our estimate of visibility benefits of the modeled interim policy in Appendix 5C is incomplete. For example, we anticipate improvement in visibility in residential areas for which we are currently unable to monetize benefits, such as the Northeastern and Central regions of the U.S. The value of visibility benefits in areas where we were unable to monetize benefits could also be substantial. EPA requests public comment on the approach taken here to quantify the monetary value of changes in visibility in Class I areas.

5.5.2 Ecosystem Services

Ecosystem services can be generally defined as the benefits that individuals and organizations obtain from ecosystems. EPA has defined ecological goods and services as the "outputs of ecological functions or processes that directly or indirectly contribute to social welfare or have the potential to do so in the future. Some outputs may be bought and sold, but most are not marketed" (U.S. EPA, 2006b). Figure 5-5 provides the Millennium Ecosystem Assessment's schematic demonstrating the connections between the categories of ecosystem services and human well-being. The interrelatedness of these categories means that any one ecosystem may provide multiple services. Changes in these services can affect human well-being by affecting security, health, social relationships, and access to basic material goods (MEA, 2005).

In the Millennium Ecosystem Assessment (MEA, 2005), ecosystem services are classified into four main categories:

1. Provisioning: Products obtained from ecosystems, such as the production of food and water

2. Regulating: Benefits obtained from the regulation of ecosystem processes, such as the control of climate and disease

3. Cultural: Nonmaterial benefits that people obtain from ecosystems through spiritual enrichment, cognitive development, reflection, recreation, and aesthetic experiences

4. Supporting: Services necessary for the production of all other ecosystem services, such as nutrient cycles and crop pollination

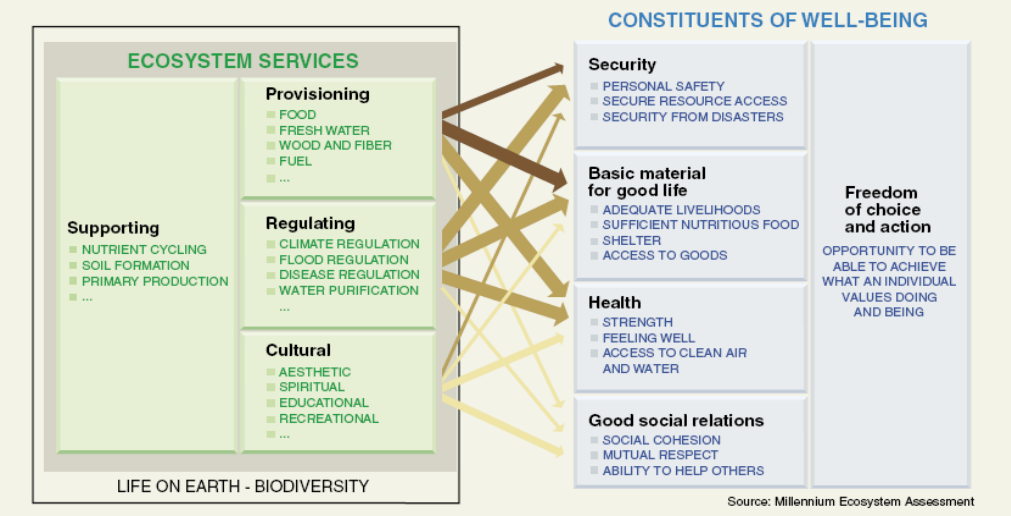

Figure 5-5. Linkages Between Categories of Ecosystem Services and Components of Human Well-Being from Millennium Ecosystem Assessment (MEA, 2005)

The monetization of ecosystem services generally involves estimating the value of ecological goods and services based on what people are willing to pay (WTP) to increase ecological services or by what people are willing to accept (WTA) in compensation for reductions in them (U.S. EPA, 2006b). There are three primary approaches for estimating the monetary value of ecosystem services: market-based approaches, revealed preference methods, and stated preference methods (U.S. EPA, 2006b). Because economic valuation of ecosystem services can be difficult, nonmonetary valuation using biophysical measurements and concepts also can be used. An example of a nonmonetary valuation method is the use of relative-value indicators (e.g., a flow chart indicating uses of a water body, such as boatable, fishable, swimmable, etc.). It is necessary to recognize that in the analysis of the environmental responses associated with any particular policy or environmental management action, only a subset of the ecosystem services likely to be affected are readily identified. Of those ecosystem services that are identified, only a subset of the changes can be quantified. Within those services whose changes can be quantified, only a few will likely be monetized, and many will remain nonmonetized. The stepwise concept leading up to the valuation of ecosystems services is graphically depicted in Figure 5-6.

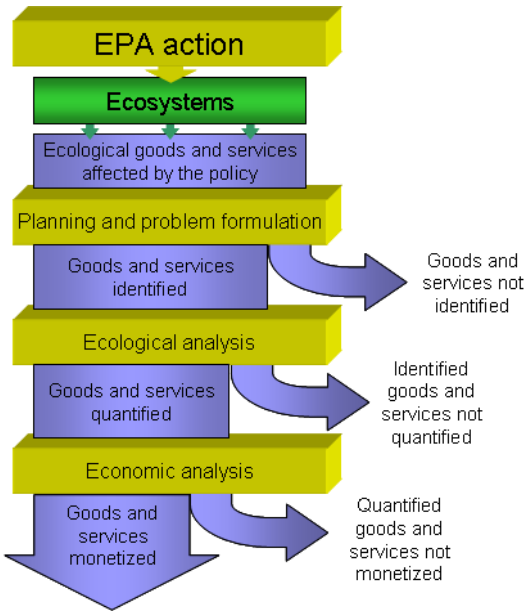

Figure 5-6. Schematic of the Benefits Assessment Process (U.S. EPA, 2006b)

5.5.3 Ecosystem Benefits of Reduced Nitrogen and Sulfur Deposition

5.5.3.1 Science of Deposition

Nitrogen and sulfur emissions occur over large regions of North America. Once these pollutants are lofted to the middle and upper troposphere, they typically have a much longer lifetime and, with the generally stronger winds at these altitudes, can be transported long distances from their source regions. The length scale of this transport is highly variable owing to differing chemical and meteorological conditions encountered along the transport path (U.S. EPA, 2008b). Sulfur is primarily emitted as SO_2, and nitrogen can be emitted as NO, NO_2, or NH_3. Secondary particles are formed from NO_x and SO_X gaseous emissions and associated chemical reactions in the atmosphere. Deposition can occur in either a wet (i.e., rain, snow, sleet, hail, clouds, or fog) or dry form (i.e., gases or particles). Together these emissions are deposited onto terrestrial and aquatic ecosystems across the U.S., contributing to the problems of acidification, nutrient enrichment, and methylmercury production as represented in Figure 5-7. Although there is some evidence that nitrogen deposition may have positive effects on agricultural and forest output through passive fertilization, it is likely that the overall value is very small relative to other health and welfare effects.

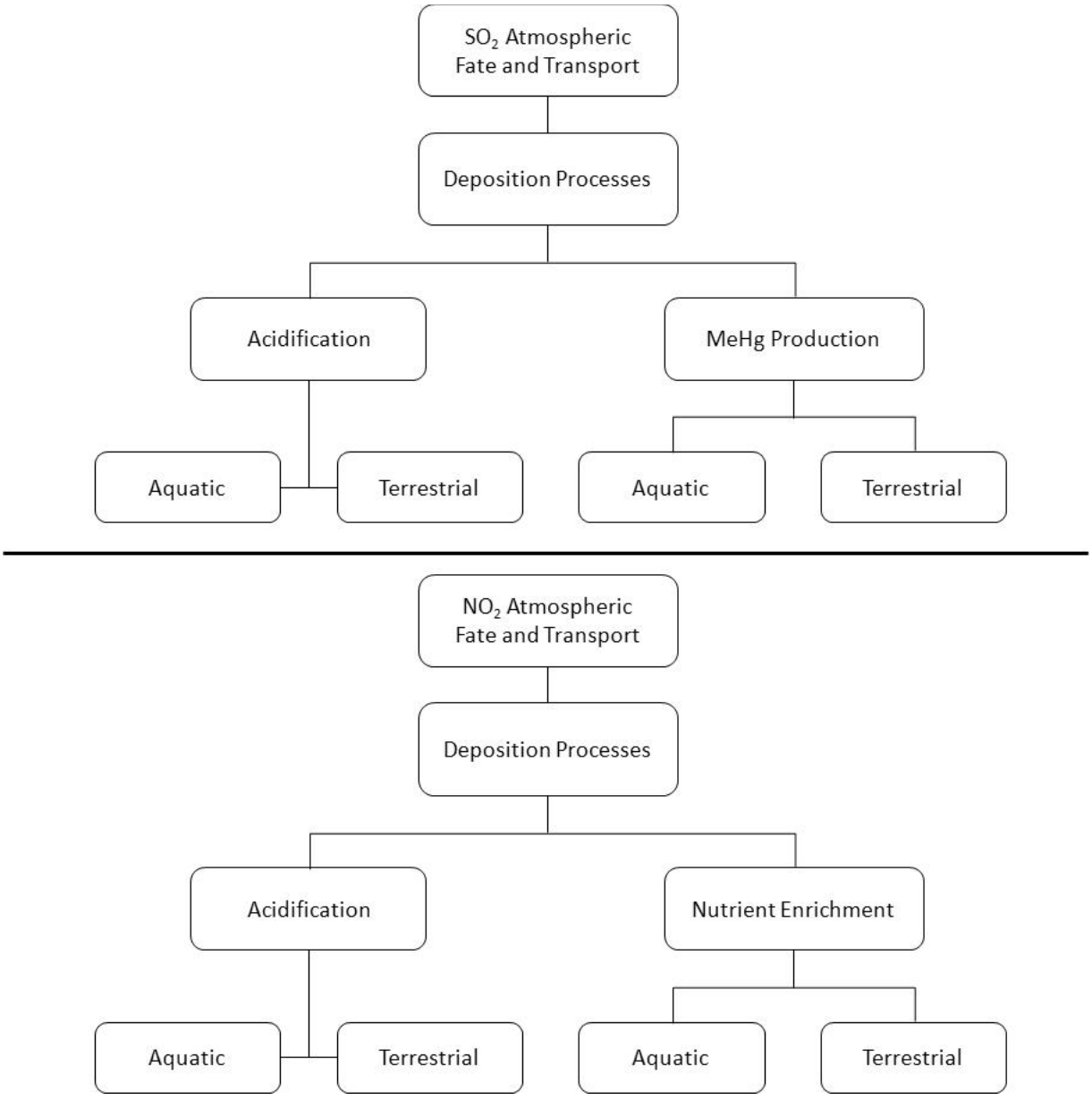

Figure 5-7. Schematics of Ecological Effects of Nitrogen and Sulfur Deposition

The lifetimes of particles vary with particle size. Accumulation-mode particles such as sulfates are kept in suspension by normal air motions and have a lower deposition velocity than coarse-mode particles; they can be transported thousands of kilometers and remain in the atmosphere for a number of days. They are removed from the atmosphere primarily by cloud processes. Particulates affect acid deposition by serving as cloud condensation nuclei and contribute directly to the acidification of rain. In addition, the gas-phase species that lead to the dry deposition of acidity are also precursors of particles. Therefore, reductions in NO_2 and SO_2

emissions will decrease both acid deposition and PM concentrations, but not necessarily in a linear fashion. (U.S. EPA, 2008b). Sulfuric acid is also deposited on surfaces by dry deposition and can contribute to environmental effects (U.S. EPA, 2008b).

5.5.3.2 Ecological Effects of Acidification

Deposition of nitrogen and sulfur can cause acidification, which alters biogeochemistry and affects animal and plant life in terrestrial and aquatic ecosystems across the U.S. Soil acidification is a natural process, but is often accelerated by acidifying deposition, which can decrease concentrations of exchangeable base cations in soils (U.S. EPA, 2008b). Major terrestrial effects include a decline in sensitive tree species, such as red spruce (*Picea rubens*) and sugar maple (*Acer saccharum*) (U.S. EPA, 2008b). Biological effects of acidification in terrestrial ecosystems are generally linked to aluminum toxicity and decreased ability of plant roots to take up base cations (U.S. EPA, 2008b). Decreases in the acid neutralizing capacity and increases in inorganic aluminum concentration contribute to declines in zooplankton, macro invertebrates, and fish species richness in aquatic ecosystems (U.S. EPA, 2008b).

Geology (particularly surficial geology) is the principal factor governing the sensitivity of terrestrial and aquatic ecosystems to acidification from nitrogen and sulfur deposition (U.S. EPA, 2008b). Geologic formations having low base cation supply generally underlie the watersheds of acid-sensitive lakes and streams. Other factors contribute to the sensitivity of soils and surface waters to acidifying deposition, including topography, soil chemistry, land use, and hydrologic flow path (U.S. EPA, 2008b).

5.5.3.3 Aquatic Ecosystems

Aquatic effects of acidification have been well studied in the U.S. and elsewhere at various trophic levels. These studies indicate that aquatic biota have been affected by acidification at virtually all levels of the food web in acid sensitive aquatic ecosystems. Effects have been most clearly documented for fish, aquatic insects, other invertebrates, and algae. Biological effects are primarily attributable to a combination of low pH and high inorganic aluminum concentrations. Such conditions occur more frequently during rainfall and snowmelt that cause high flows of water and less commonly during low-flow conditions, except where chronic acidity conditions are severe. Biological effects of episodes include reduced fish condition factor[16], changes in species composition and declines in aquatic species richness

[16] Condition factor is an index that describes the relationship between fish weight and length, and is one measure of sublethal acidification stress that has been used to quantify effects of acidification on an individual fish (U.S.EPA, 2008b).

across multiple taxa, ecosystems and regions. These conditions may also result in direct fish mortality (Van Sickle et al., 1996). Biological effects in aquatic ecosystems can be divided into two major categories: effects on health, vigor, and reproductive success; and effects on biodiversity. Surface water with ANC values greater than 50 µeq/L generally provides moderate protection for most fish (i.e., brook trout, others) and other aquatic organisms (U.S. EPA, 2009c). Table 5-15 provides a summary of the biological effects experienced at various ANC levels.

Table 5-15. Aquatic Status Categories

Acute Concern	<0 micro equivalent per Liter (µeq/L)	Near complete loss of fish populations is expected. Planktonic communities have extremely low diversity and are dominated by acidophilic forms. The number of individuals in plankton species that are present is greatly reduced.
Severe Concern	0–20 µeq/L	Highly sensitive to episodic acidification. During episodes of high acidifying deposition, brook trout populations may experience lethal effects. Diversity and distribution of zooplankton communities decline sharply.
Elevated Concern	20–50 µeq/L	Fish species richness is greatly reduced (i.e., more than half of expected species can be missing). On average, brook trout populations experience sublethal effects, including loss of health, reproduction capacity, and fitness. Diversity and distribution of zooplankton communities decline.
Moderate Concern	50–100 µeq/L	Fish species richness begins to decline (i.e., sensitive species are lost from lakes). Brook trout populations are sensitive and variable, with possible sublethal effects. Diversity and distribution of zooplankton communities also begin to decline as species that are sensitive to acidifying deposition are affected.
Low Concern	>100 µeq/L	Fish species richness may be unaffected. Reproducing brook trout populations are expected where habitat is suitable. Zooplankton communities are unaffected and exhibit expected diversity and distribution.

A number of national and regional assessments have been conducted to estimate the distribution and extent of surface water acidity in the U.S. (U.S. EPA, 2008b). As a result, several regions of the U.S. have been identified as containing a large number of lakes and streams that are seriously impacted by acidification. Figure 5-8 illustrates those areas of the U.S. where aquatic ecosystems are at risk from acidification.

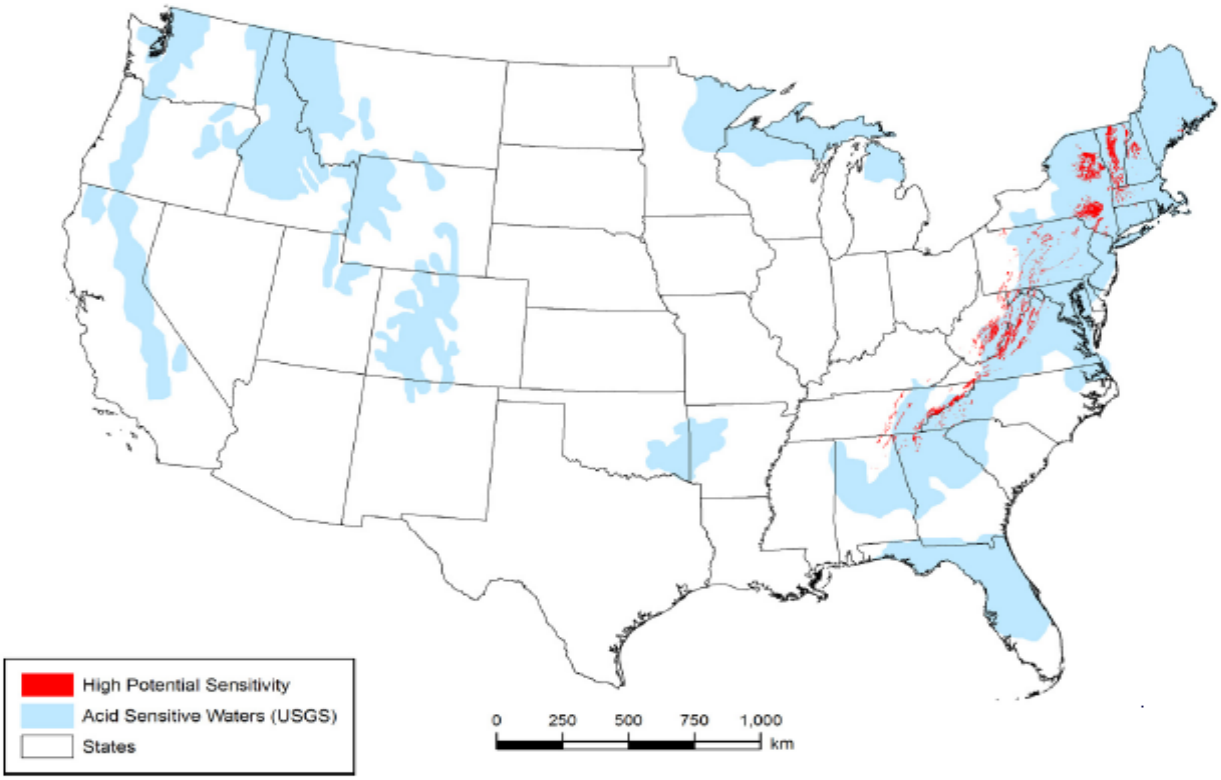

Figure 5-8. Areas Potentially Sensitive to Aquatic Acidification (U.S. EPA, 2008b)

Because acidification primarily affects the diversity and abundance of aquatic biota, it also affects the ecosystem services that are derived from the fish and other aquatic life found in these surface waters.

While acidification is unlikely to have serious negative effects on, for example, water supplies, it can limit the productivity of surface waters as a source of food (i.e., fish). In the northeastern United States, the surface waters affected by acidification are not a major source of commercially raised or caught fish; however, they are a source of food for some recreational and subsistence fishermen and for other consumers. For example, there is evidence that certain population subgroups in the northeastern United States, such as the Hmong and Chippewa ethnic groups, have particularly high rates of self-caught fish consumption (Hutchison and Kraft, 1994; Peterson et al., 1994). However, it is not known if and how their consumption patterns are affected by the reductions in available fish populations caused by surface water acidification.

Inland surface waters support several cultural services, including aesthetic and educational services and recreational fishing. Recreational fishing in lakes and streams is among

the most popular outdoor recreational activities in the northeastern United States. Based on studies conducted in the northeastern United States, Kaval and Loomis (2003) estimated average consumer surplus values per day of $36 for recreational fishing (in 2007 dollars); therefore, the implied total annual value of freshwater fishing in the northeastern United States was $5.1 billion in 2006.[17] For recreation days, consumer surplus value is most commonly measured using recreation demand, travel cost models.

Another estimate of the overarching ecological benefits associated with reducing lake acidification levels in Adirondacks National Park can be derived from the contingent valuation (CV) survey (Banzhaf et al., 2006), which elicited values for specific improvements in acidification-related water quality and ecological conditions in Adirondack lakes. The survey described a base version with minor improvements said to result from the program, and a scope version with large improvements due to the program and a gradually worsening status quo. After adapting and transferring the results of this study and converting the 10-year annual payments to permanent annual payments using discount rates of 3% and 5%, the WTP estimates ranged from $48 to $107 per year per household (in 2004 dollars) for the base version and $54 to $154 for the scope version. Using these estimates, the aggregate annual benefits of eliminating all anthropogenic sources of NO_x and SO_x emissions were estimated to range from $291 million to $829 million (U.S. EPA, 2009b).[18]

In addition, inland surface waters provide a number of regulating services associated with hydrological and climate regulation by providing environments that sustain aquatic food webs. These services are disrupted by the toxic effects of acidification on fish and other aquatic life. Although it is difficult to quantify these services and how they are affected by acidification, some of these services may be captured through measures of provisioning and cultural services.

5.5.3.4 Terrestrial Ecosystems

Acidifying deposition has altered major biogeochemical processes in the U.S. by increasing the nitrogen and sulfur content of soils, accelerating nitrate and sulfate leaching from soil to drainage waters, depleting base cations (especially calcium and magnesium) from soils, and increasing the mobility of aluminum. Inorganic aluminum is toxic to some tree roots. Plants affected by high levels of aluminum from the soil often have reduced root growth, which

[17] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

[18] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

restricts the ability of the plant to take up water and nutrients, especially calcium (U. S. EPA, 2008b). These direct effects can, in turn, influence the response of these plants to climatic stresses such as droughts and cold temperatures. They can also influence the sensitivity of plants to other stresses, including insect pests and disease (Joslin et al., 1992) leading to increased mortality of canopy trees. In the U.S., terrestrial effects of acidification are best described for forested ecosystems (especially red spruce and sugar maple ecosystems) with additional information on other plant communities, including shrubs and lichen (U.S. EPA, 2008b).

Certain ecosystems in the continental U.S. are potentially sensitive to terrestrial acidification, which is the greatest concern regarding nitrogen and sulfur deposition U.S. EPA (2008b). Figure 5-9 depicts the areas across the U.S. that are potentially sensitive to terrestrial acidification.

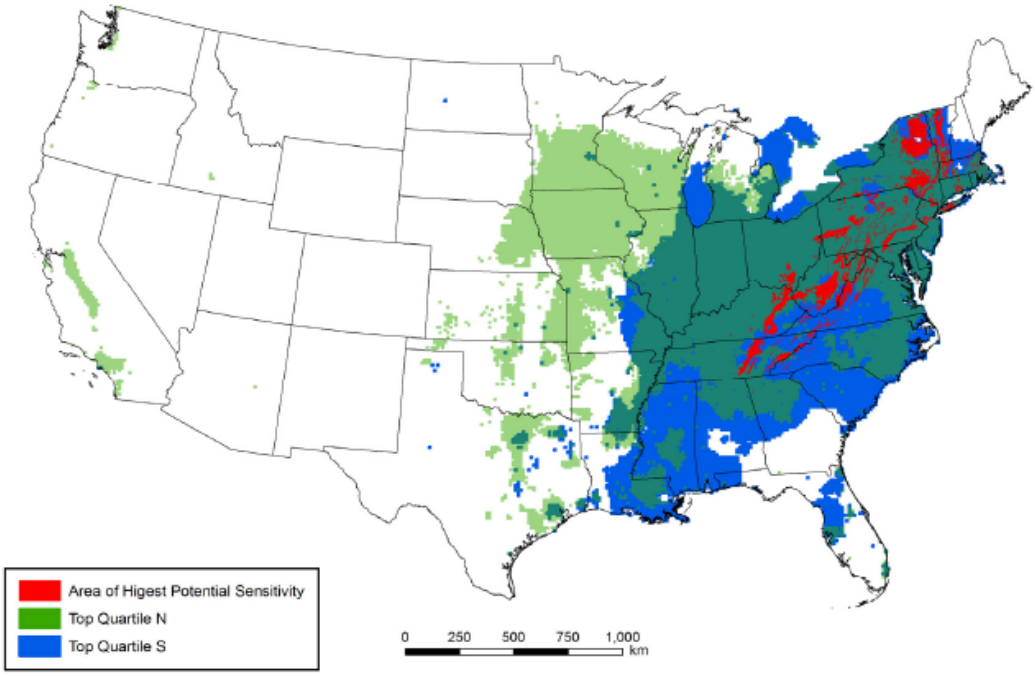

Figure 5-9. Areas Potentially Sensitive to Terrestrial Acidification (U.S. EPA, 2008b)

Both coniferous and deciduous forests throughout the eastern U.S. are experiencing gradual losses of base cation nutrients from the soil due to accelerated leaching from acidifying deposition. This change in nutrient availability may reduce the quality of forest nutrition over the long term. Evidence suggests that red spruce and sugar maple in some areas in the eastern U.S. have experienced declining health because of this deposition. For red spruce, (*Picea*

rubens) dieback or decline has been observed across high elevation landscapes of the northeastern U.S., and to a lesser extent, the southeastern U.S., and acidifying deposition has been implicated as a causal factor (DeHayes et al., 1999). Figure 5-10 shows the distribution of red spruce (brown) and sugar maple (green) in the eastern U.S.

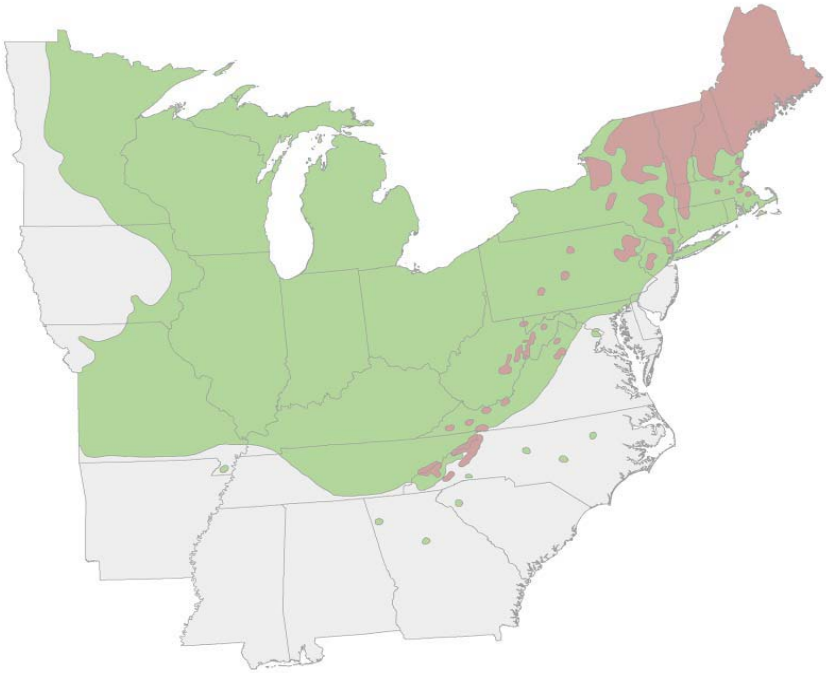

Figure 5-10. Distribution of Red Spruce (Pink) and Sugar Maple (Green) in the Eastern U.S. (U.S. EPA, 2008b)

Terrestrial acidification affects several important ecological endpoints, including declines in habitat for threatened and endangered species (cultural), declines in forest aesthetics (cultural), declines in forest productivity (provisioning), and increases in forest soil erosion and reductions in water retention (cultural and regulating).

Forests in the northeastern United States provide several important and valuable provisioning services in the form of tree products. Sugar maples are a particularly important commercial hardwood tree species, providing timber and maple syrup. In the United States, sugar maple saw timber was nearly 900 million board feet in 2006 (USFS, 2006), and annual production of maple syrup was nearly 1.4 million gallons, accounting for approximately 19% of worldwide production. The total annual value of U.S. production in these years was approximately $160 million (NASS, 2008). Red spruce is also used in a variety of products including lumber, pulpwood, poles, plywood, and musical instruments. The total removal of red

spruce saw timber from timberland in the United States was over 300 million board feet in 2006 (USFS, 2006).

Forests in the northeastern United States are also an important source of cultural ecosystem services—nonuse (i.e., existence value for threatened and endangered species), recreational, and aesthetic services. Red spruce forests are home to two federally listed species and one delisted species:

- Spruce-fir moss spider (*Microhexura montivaga*)—endangered
- Rock gnome lichen (*Gymnoderma lineare*)—endangered
- Virginia northern flying squirrel (*Glaucomys sabrinus fuscus*)—delisted, but important

Forestlands support a wide variety of outdoor recreational activities, including fishing, hiking, camping, off-road driving, hunting, and wildlife viewing. Regional statistics on recreational activities that are specifically forest based are not available; however, more general data on outdoor recreation provide some insights into the overall level of recreational services provided by forests. More than 30% of the U.S. adult population visited a wilderness or primitive area during the previous year and engaged in day hiking (Cordell et al., 2008). From 1999 to 2004, 16% of adults in the northeastern United States participated in off-road vehicle recreation, for an average of 27 days per year (Cordell et al., 2005). The average consumer surplus value per day of off-road driving in the United States was $25 (in 2007 dollars), and the implied total annual value of off-road driving recreation in the northeastern United States was more than $9 billion (Kaval and Loomis, 2003). More than 5% of adults in the northeastern United States participated in nearly 84 million hunting days (U.S. FWS and U.S. Census Bureau, 2007). Ten percent of adults in northeastern states participated in wildlife viewing away from home on 122 million days in 2006. For these recreational activities in the northeastern United States, Kaval and Loomis (2003) estimated average consumer surplus values per day of $52 for hunting and $34 for wildlife viewing (in 2007 dollars). The implied total annual value of hunting and wildlife viewing in the northeastern United States was, therefore, $4.4 billion and $4.2 billion, respectively, in 2006.

As previously mentioned, it is difficult to estimate the portion of these recreational services that are specifically attributable to forests and to the health of specific tree species. However, one recreational activity that is directly dependent on forest conditions is fall color viewing. Sugar maple trees, in particular, are known for their bright colors and are, therefore,

an essential aesthetic component of most fall color landscapes. A survey of residents in the Great Lakes area found that roughly 30% of residents reported at least one trip in the previous year involving fall color viewing (Spencer and Holecek, 2007). In a separate study conducted in Vermont, Brown (2002) reported that more than 22% of households visiting Vermont in 2001 made the trip primarily for viewing fall colors.

Two studies estimated values for protecting high-elevation spruce forests in the southern Appalachian Mountains. Kramer et al. (2003) conducted a contingent valuation study estimating households' WTP for programs to protect remaining high-elevation spruce forests from damages associated with air pollution and insect infestation. Median household WTP was estimated to be roughly $29 (in 2007 dollars) for a smaller program, and $44 for the more extensive program. Jenkins et al. (2002) conducted a very similar study in seven Southern Appalachian states on a potential program to maintain forest conditions at status quo levels. The overall mean annual WTP for the forest protection programs was $208 (in 2007 dollars). Multiplying the average WTP estimate from these studies by the total number of households in the seven-state Appalachian region results in an aggregate annual range of $470 million to $3.4 billion for avoiding a significant decline in the health of high-elevation spruce forests in the Southern Appalachian region.[19]

Forests in the northeastern United States also support and provide a wide variety of valuable regulating services, including soil stabilization and erosion control, water regulation, and climate regulation. The total value of these ecosystem services is very difficult to quantify in a meaningful way, as is the reduction in the value of these services associated with total nitrogen and sulfur deposition. As terrestrial acidification contributes to root damages, reduced biomass growth, and tree mortality, all of these services are likely to be affected; however, the magnitude of these impacts is currently very uncertain.

5.5.4 Ecological Effects Associated with Gaseous Sulfur Dioxide

Uptake of gaseous sulfur dioxide in a plant canopy is a complex process involving adsorption to surfaces (leaves, stems, and soil) and absorption into leaves. SO_2 penetrates into leaves through to the stomata, although there is evidence for limited pathways via the cuticle. Pollutants must be transported from the bulk air to the leaf boundary layer in order to get to the stomata. When the stomata are closed, as occurs under dark or drought conditions, resistance to gas uptake is very high and the plant has a very low degree of susceptibility to

[19] These estimates reflect the marginal value of the service for the hypothetical program described in the survey, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

injury. In contrast, mosses and lichens do not have a protective cuticle barrier to gaseous pollutants or stomates and are generally more sensitive to gaseous sulfur and nitrogen than vascular plants (U.S. EPA, 2008b). Acute foliar injury usually happens within hours of exposure, involves a rapid absorption of a toxic dose, and involves collapse or necrosis of plant tissues. Another type of visible injury is termed chronic injury and is usually a result of variable SO_2 exposures over the growing season. Besides foliar injury, chronic exposure to low SO_2 concentrations can result in reduced photosynthesis, growth, and yield of plants (U.S. EPA, 2008b). These effects are cumulative over the season and are often not associated with visible foliar injury. As with foliar injury, these effects vary among species and growing environment. SO_2 is also considered the primary factor causing the death of lichens in many urban and industrial areas (Hutchinson et al., 1996).

In addition to the role of sulfate deposition on methylation, the technologies installed to reduce emissions of NO_x and SO_2 associated with this rule would also reduce mercury emissions. EPA recently commissioned an information collection request that will soon provide greatly improved power industry mercury emissions estimates that will enable the Agency to better estimate mercury emissions changes from its air emissions control actions. For this reason, the Agency did not estimate Hg changes in this rule and will instead wait for these new data which will be available in the near future. Due to time and resource limitations, we were unable in any event to model mercury dispersion, deposition, methylation, bioaccumulation in fish tissue, and human consumption of mercury-contaminated fish that would be needed in order to estimate the human health benefits from reducing these mercury emissions.

5.5.5 Nitrogen Enrichment

5.5.5.1 Aquatic Enrichment

One of the main adverse ecological effects resulting from N deposition, particularly in the Mid-Atlantic region of the United States, is the effect associated with nutrient enrichment in estuarine waters. A recent assessment of 141 estuaries nationwide by the National Oceanic and Atmospheric Administration (NOAA) concluded that 19 estuaries (13%) suffered from moderately high or high levels of eutrophication due to excessive inputs of both N and phosphorus, and a majority of these estuaries are located in the coastal area from North Carolina to Massachusetts (NOAA, 2007). For estuaries in the Mid-Atlantic region, the contribution of atmospheric distribution to total N loads is estimated to range between 10% and 58% (Valigura et al., 2001).

Eutrophication in estuaries is associated with a range of adverse ecological effects. The conceptual framework developed by NOAA emphasizes four main types of eutrophication effects—low dissolved oxygen (DO), harmful algal blooms (HABs), loss of submerged aquatic vegetation (SAV), and low water clarity. Low DO disrupts aquatic habitats, causing stress to fish and shellfish, which, in the short-term, can lead to episodic fish kills and, in the long-term, can damage overall growth in fish and shellfish populations. Low DO also degrades the aesthetic qualities of surface water. In addition to often being toxic to fish and shellfish, and leading to fish kills and aesthetic impairments of estuaries, HABs can, in some instances, also be harmful to human health. SAV provides critical habitat for many aquatic species in estuaries and, in some instances, can also protect shorelines by reducing wave strength; therefore, declines in SAV due to nutrient enrichment are an important source of concern. Low water clarity is the result of accumulations of both algae and sediments in estuarine waters. In addition to contributing to declines in SAV, high levels of turbidity also degrade the aesthetic qualities of the estuarine environment.

Estuaries in the eastern United States are an important source of food production, in particular fish and shellfish production. The estuaries are capable of supporting large stocks of resident commercial species, and they serve as the breeding grounds and interim habitat for several migratory species. To provide an indication of the magnitude of provisioning services associated with coastal fisheries, from 2005 to 2007, the average value of total catch was $1.5 billion per year. It is not known, however, what percentage of this value is directly attributable to or dependent upon the estuaries in these states.

In addition to affecting provisioning services through commercial fish harvests, eutrophication in estuaries may also affect the demand for seafood. For example, a well-publicized toxic pfiesteria bloom in the Maryland Eastern Shore in 1997, which involved thousands of dead and lesioned fish, led to an estimated $56 million (in 2007 dollars) in lost seafood sales for 360 seafood firms in Maryland in the months following the outbreak (Lipton, 1999).

Estuaries in the United States also provide an important and substantial variety of cultural ecosystem services, including water-based recreational and aesthetic services. The water quality in the estuary directly affects the quality of these experiences. For example, there were 26 million days of saltwater fishing coastal states from North Carolina to Massachusetts in 2006 (FWA and Census, 2007). Assuming an average consumer surplus value for a fishing day at

$36 (in 2007 dollars) in the Northeast and $87 in the Southeast (Kaval and Loomis, 2003), the aggregate value was approximately $1.3 billion (in 2007 dollars).[20] In addition, almost 6 million adults participated in motorboating in coastal states from North Carolina to Massachusetts, for a total of nearly 63 million days annually during 1999–2000 (Leeworthy and Wiley, 2001). Using a national daily value estimate of $32 (in 2007 dollars) for motorboating (Kaval and Loomis (2003), the aggregate value of these coastal motorboating outings was $2 billion per year.[21] Almost 7 million participated in birdwatching for 175 million days per year, and more than 3 million participated in visits to non-beach coastal waterside areas.

Estuaries and marshes have the potential to support a wide range of regulating services, including climate, biological, and water regulation; pollution detoxification; erosion prevention; and protection against natural hazards from declines in SAV (MEA, 2005). SAV can help reduce wave energy levels and thus protect shorelines against excessive erosion, which increases the risks of episodic flooding and associated damages to near-shore properties or public infrastructure or even contribute to shoreline retreat.

5.5.5.2 Terrestrial Enrichment

Terrestrial enrichment occurs when terrestrial ecosystems receive N loadings in excess of natural background levels, either through atmospheric deposition or direct application. Evidence presented in the Integrated Science Assessment (U.S. EPA, 2008b) supports a causal relationship between atmospheric N deposition and biogeochemical cycling and fluxes of N and carbon in terrestrial systems. Furthermore, evidence summarized in the report supports a causal link between atmospheric N deposition and changes in the types and number of species and biodiversity in terrestrial systems. Nitrogen enrichment occurs over a long time period; as a result, it may take as much as 50 years or more to see changes in ecosystem conditions and indicators. This long time scale also affects the timing of the ecosystem service changes.

One of the main provisioning services potentially affected by N deposition is grazing opportunities offered by grasslands for livestock production in the Central U.S. Although N deposition on these grasslands can offer supplementary nutritive value and promote overall grass production, there are concerns that fertilization may favor invasive grasses and shift the species composition away from native grasses. This process may ultimately reduce the

[20] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

[21] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

productivity of grasslands for livestock production. Losses due to invasive grasses can be significant; for example, based on a bioeconomic model of cattle grazing in the upper Great Plains, Leitch, Leistritz, and Bangsund (1996) and Leistritz, Bangsund, and Hodur (2004) estimated $130 million in losses due to a leafy spurge infestation in the Dakotas, Montana, and Wyoming. [22] However, the contribution of N deposition to these losses is still uncertain.

5.5.6 Benefits of Reducing Ozone Effects on Vegetation and Ecosystems

Ozone causes discernible injury to a wide array of vegetation (U.S. EPA, 2006c; Fox and Mickler, 1996). In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts (U.S. EPA, 2006c). Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function (De Steiguer et al., 1990; Pye, 1988).

When ozone is present in the air, it can enter the leaves of plants, where it can cause significant cellular damage. Like carbon dioxide (CO_2) and other gaseous substances, ozone enters plant tissues primarily through the stomata in leaves in a process called "uptake" (Winner and Atkinson, 1986). Once sufficient levels of ozone (a highly reactive substance), or its reaction products, reaches the interior of plant cells, it can inhibit or damage essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns (U.S. EPA, 2006c; Tingey and Taylor, 1982). With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance, leading to reduced growth and/or reproduction. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, or more susceptible to disease, pest infestation, harsh weather (e.g., drought, frost) and other environmental stresses, which can all produce a loss in plant vigor in ozone-sensitive species that over time may lead to premature plant death. Furthermore, there is evidence that ozone can interfere with the formation of mycorrhiza, essential symbiotic fungi associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont (U.S. EPA, 2006c).

[22] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

This ozone damage may or may not be accompanied by visible injury on leaves, and likewise, visible foliar injury may or may not be a symptom of the other types of plant damage described above. Foliar injury is usually the first visible sign of injury to plants from ozone exposure and indicates impaired physiological processes in the leaves (Grulke, 2003). When visible injury is present, it is commonly manifested as chlorotic or necrotic spots, and/or increased leaf senescence (accelerated leaf aging). Because ozone damage can consist of visible injury to leaves, it can also reduce the aesthetic value of ornamental vegetation and trees in urban landscapes, and negatively affects scenic vistas in protected natural areas.

Ozone can produce both acute and chronic injury in sensitive species depending on the concentration level and the duration of the exposure. Ozone effects also tend to accumulate over the growing season of the plant, so that even lower concentrations experienced for a longer duration have the potential to create chronic stress on sensitive vegetation. Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of ozone uptake through closure of stomata) (U.S. EPA, 2006c; Winner, 1994). After injuries have occurred, plants may be capable of repairing the damage to a limited extent (U.S. EPA, 2006c). Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and response to ozone, it is not possible to identify threshold values above which ozone is consistently toxic for all plants.

Because plants are at the base of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone impacts at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors (U.S. EPA, 2006c). In most instances, responses to chronic or recurrent exposure in forested ecosystems are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems (U.S. EPA, 2006c, McBride et al., 1985; Miller et al., 1982). It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States (U.S. EPA, 2006c).

5.5.6.1 Ozone Effects on Forests

Air pollution can affect the environment and affect ecological systems, leading to changes in the ecological community and influencing the diversity, health, and vigor of individual species (U.S. EPA, 2006c). Ozone has been shown in numerous studies to have a strong effect on the health of many plants, including a variety of commercial and ecologically important forest tree species throughout the United States (U.S. EPA, 2007).

In the U.S., this data comes from the U.S. Department of Agriculture (USDA) Forest Service Forest Inventory and Analysis (FIA) program. As part of its Phase 3 program, formerly known as Forest Health Monitoring, FIA examines ozone injury to ozone-sensitive plant species at ground monitoring sites in forestland across the country (excluding woodlots and urban trees). FIA looks for damage on the foliage of ozone-sensitive forest plant species at each site that meets certain minimum criteria. Because ozone injury is cumulative over the course of the growing season, examinations are conducted in July and August, when ozone injury is typically highest.

Monitoring of ozone injury to plants by the USDA Forest Service has expanded over the last 10 years from monitoring sites in 10 states in 1994 to nearly 1,000 monitoring sites in 41 states in 2002. The data underlying the indictor in Figure 5-11 are based on averages of all observations collected in 2002, the latest year for which data are publicly available at the time the study was conducted, and are broken down by U.S. EPA Regions. Ozone damage to forest plants is classified using a subjective five-category biosite index based on expert opinion, but designed to be equivalent from site to site. Ranges of biosite values translate to no injury, low or moderate foliar injury (visible foliar injury to highly sensitive or moderately sensitive plants, respectively), and high or severe foliar injury, which would be expected to result in tree-level or ecosystem-level responses, respectively (U.S. EPA, 2006c; Coulston, 2004). The highest percentages of observed high and severe foliar injury, which are most likely to be associated with tree or ecosystem-level responses, are primarily found in the Mid-Atlantic and Southeast regions.

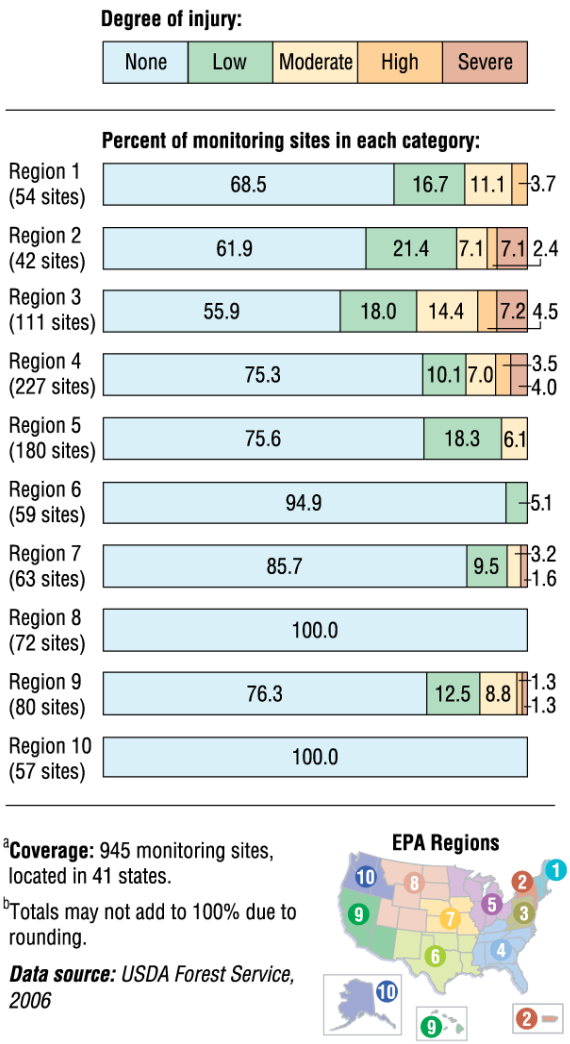

Figure 5-11. Ozone Injury to Forest Plants in U.S. by EPA Regions, 2002

Assessing the impact of ground-level ozone on forests in the eastern United States involves understanding the risks to sensitive tree species from ambient ozone concentrations and accounting for the prevalence of those species within the forest. As a way to quantify the risks to particular plants from ground-level ozone, scientists have developed ozone-exposure/tree-response functions by exposing tree seedlings to different ozone levels and measuring reductions in growth as "biomass loss." Typically, seedlings are used because they are easy to manipulate and measure their growth loss from ozone pollution. The mechanisms of susceptibility to ozone within the leaves of seedlings and mature trees are identical, and the decreases predicted using the seedlings should be related to the decrease in overall plant fitness for mature trees, but the magnitude of the effect may be higher or lower depending on the tree species (Chappelka and Samuelson, 1998). In areas where certain ozone-sensitive

species dominate the forest community, the biomass loss from ozone can be significant. Significant biomass loss can be defined as a more than 2% annual biomass loss, which would cause long term ecological harm as the short-term negative effects on seedlings compound to affect long-term forest health (Heck, 1997).

Some of the common tree species in the United States that are sensitive to ozone are black cherry (*Prunus serotina*), tulip-poplar (*Liriodendron tulipifera)*, and eastern white pine (*Pinus strobus)*. Ozone-exposure/tree-response functions have been developed for each of these tree species, as well as for aspen (*Populus tremuliodes*), and ponderosa pine (*Pinus ponderosa*) (U.S. EPA, 2007). Other common tree species, such as oak (*Quercus* spp.*)* and hickory (*Carya* spp.), are not as sensitive to ozone. Consequently, with knowledge of the distribution of sensitive species and the level of ozone at particular locations, it is possible to estimate a "biomass loss" for each species across their range. As shown in Figure 5-12, current ambient levels of ozone are associated with significant biomass loss across large geographic areas (U.S. EPA, 2009b). However, this information is unavailable for this rule.

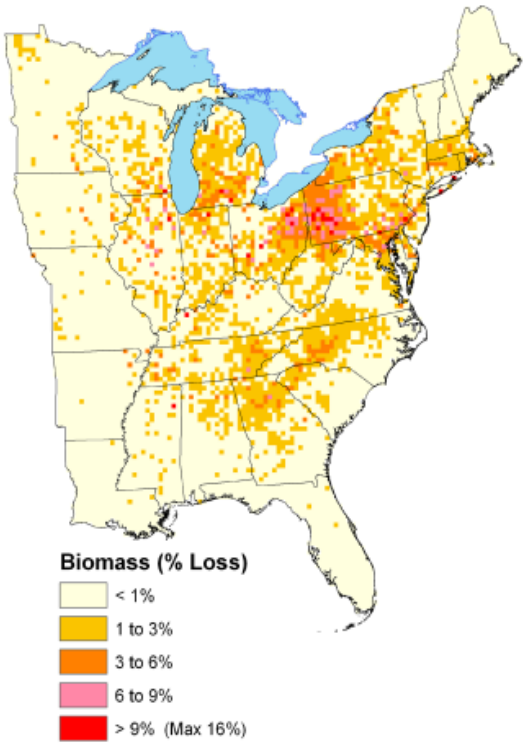

Figure 5-12. Estimated Black Cherry, Yellow Poplar, Sugar Maple, Eastern White Pine, Virginia Pine, Red Maple, and Quaking Aspen Biomass Loss due to Current Ozone Exposure, 2006-2008 (U.S. EPA, 2009b)

To estimate the biomass loss for forest ecosystems across the eastern United States, the biomass loss for each of the seven tree species was calculated using the three-month, 12-hour W126 exposure metric at each location, along with each tree's individual C-R functions. The W126 exposure metric was calculated using monitored ozone data from CASTNET and AQS sites, and a three-year average was used to mitigate the effect of variations in meteorological and soil moisture conditions. The biomass loss estimate for each species was then multiplied by its prevalence in the forest community using the U.S. Department of Agriculture (USDA) Forest Service IV index of tree abundance calculated from Forest Inventory and Analysis (FIA) measurements (Prasad, 2003). Sources of uncertainty include the ozone-exposure/plant-response functions, the tree abundance index, and other factors (e.g., soil moisture). Although these factors were not considered, they can affect ozone damage (Chappelka, 1998).

Ozone damage to the plants including the trees and understory in a forest can affect the ability of the forest to sustain suitable habitat for associated species particularly threatened and endangered species that have existence value—a nonuse ecosystem service—for the public. Similarly, damage to trees and the loss of biomass can affect the forest's provisioning services in the form of timber for various commercial uses. In addition, ozone can cause discoloration of leaves and more rapid senescence (early shedding of leaves), which could negatively affect fall-color tourism because the fall foliage would be less available or less attractive. Beyond the aesthetic damage to fall color vistas, forests provide the public with many other recreational and educational services that may be impacted by reduced forest health including hiking, wildlife viewing (including bird watching), camping, picnicking, and hunting. Another potential effect of biomass loss in forests is the subsequent loss of climate regulation service in the form of reduced ability to sequester carbon (Felzer et al., 2005).

5.5.6.2 Ozone Effects on Crops and Urban Ornamentals

Laboratory and field experiments have also shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). Damage to crops from ozone exposures includes yield losses (i.e., in terms of weight, number, or size of the plant part that is harvested), as well as changes in crop quality (i.e., physical appearance, chemical composition, or the ability to withstand storage) (U.S. EPA, 2007). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the United States" (U.S. EPA, 2006c). In addition, economic studies have shown reduced economic benefits as a result of predicted reductions in crop yields, directly affecting

the amount and quality of the provisioning service provided by the crops in question, associated with observed ozone levels (Kopp et al., 1985; Adams et al., 1986; Adams et al., 1989). According to the Ozone Staff Paper, there has been no evidence that crops are becoming more tolerant of ozone (U.S. EPA, 2007). Using the Agriculture Simulation Model (AGSIM) (Taylor, 1994) to calculate the agricultural benefits of reductions in ozone exposure, U.S. EPA estimated that meeting a W126 standard of 21 ppm-hr would produce monetized benefits of approximately $160 million to $300 million (inflated to 2006 dollars) (U.S. EPA, 2007). [23]

Urban ornamentals are an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels. Because ozone causes visible foliar injury, the aesthetic value of ornamentals (such as petunia, geranium, and poinsettia) in urban landscapes would be reduced (U.S. EPA, 2007). Sensitive ornamental species would require more frequent replacement and/or increased maintenance (fertilizer or pesticide application) to maintain the desired appearance because of exposure to ambient ozone (U.S. EPA, 2007). In addition, many businesses rely on healthy-looking vegetation for their livelihoods (e.g., horticulturalists, landscapers, Christmas tree growers, farmers of leafy crops, etc.) and a variety of ornamental species have been listed as sensitive to ozone (Abt Associates, 2010). The ornamental landscaping industry is valued at more than $30 billion (inflated to 2006 dollars) annually, by both private property owners/tenants and by governmental units responsible for public areas (Abt Associates, 2010). Therefore, urban ornamentals represent a potentially large unquantified benefit category. This aesthetic damage may affect the enjoyment of urban parks by the public and homeowners' enjoyment of their landscaping and gardening activities. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, we cannot conduct a quantitative analysis to estimate these effects.

5.5.7 Unquantified SO_2 and NO_2-Related Human Health Benefits

Following an extensive evaluation of health evidence from epidemiologic and laboratory studies, the Integrated Science Assessment for Sulfur Dioxide concluded that there is a causal relationship between respiratory health effects and short-term exposure to SO_2 (U.S. EPA, 2008c). The immediate effect of SO_2 on the respiratory system in humans is bronchoconstriction. Asthmatics are more sensitive to the effects of SO_2 likely resulting from preexisting inflammation associated with this disease. A clear concentration-response

[23] These estimates illustrate the value of vegetation effects from a substantial reduction of ozone concentrations, not the marginal change in ozone concentrations anticipated a result of the emission reductions achieved by this rule.

relationship has been demonstrated in laboratory studies following exposures to SO_2 at concentrations between 20 and 100 ppb, both in terms of increasing severity of effect and percentage of asthmatics adversely affected. Based on our review of this information, we identified four short-term morbidity endpoints that the SO_2 ISA identified as a "causal relationship": asthma exacerbation, respiratory-related emergency department visits, and respiratory-related hospitalizations. The differing evidence and associated strength of the evidence for these different effects is described in detail in the SO_2 ISA. The SO_2 ISA also concluded that the relationship between short-term SO_2 exposure and premature mortality was "suggestive of a causal relationship" because it is difficult to attribute the mortality risk effects to SO_2 alone. Although the SO_2 ISA stated that studies are generally consistent in reporting a relationship between SO_2 exposure and mortality, there was a lack of robustness of the observed associations to adjustment for pollutants. We did not quantify these benefits due to time constraints.

Epidemiological researchers have associated NO_2 exposure with adverse health effects in numerous toxicological, clinical and epidemiological studies, as described in the Integrated Science Assessment for Oxides of Nitrogen - Health Criteria (Final Report) (U.S. EPA, 2008c). The NO_2 ISA provides a comprehensive review of the current evidence of health and environmental effects of NO_2. The NO_2 ISA concluded that the evidence "is sufficient to infer a likely causal relationship between short-term NO_2 exposure and adverse effects on the respiratory system" (ISA, section 5.3.2.1). These epidemiologic and experimental studies encompass a number of endpoints including [Emergency Department (ED)] visits and hospitalizations, respiratory symptoms, airway hyperresponsiveness, airway inflammation, and lung function. Effect estimates from epidemiologic studies conducted in the United States and Canada generally indicate a 2-20% increase in risks for ED visits and hospital admissions and higher risks for respiratory symptoms (ISA, section 5.4). The NO_2 ISA concluded that the relationship between short-term NO_2 exposure and premature mortality was "suggestive but not sufficient to infer a causal relationship" because it is difficult to attribute the mortality risk effects to NO_2 alone. Although the NO_2 ISA stated that studies consistently reported a relationship between NO_2 exposure and mortality, the effect was generally smaller than that for other pollutants such as PM. We did not quantify these co-benefits due to time constraints.

5.6 Social Cost of Carbon and Greenhouse Gas Co-Benefits

EPA has assigned a dollar value to reductions in carbon dioxide (CO_2) emissions using recent estimates of the "social cost of carbon" (SCC). The SCC is an estimate of the monetized damages associated with an incremental increase in carbon emissions in a given year. It is

intended to include (but is not limited to) changes in net agricultural productivity, human health, property damages from increased flood risk, and the value of ecosystem services due to climate change. The SCC estimates used in this analysis were developed through an interagency process that included EPA and other executive branch entities, and concluded in February 2010. EPA first used these SCC estimates in the benefits analysis for the final joint EPA/DOT Rulemaking to establish Light-Duty Vehicle Greenhouse Gas Emission Standards and Corporate Average Fuel Economy Standards; see the rule's preamble for discussion about application of SCC (75 FR 25324; 5/7/10). The SCC Technical Support Document (SCC TSD) provides a complete discussion of the methods used to develop these SCC estimates.[24]

The interagency group selected four SCC values for use in regulatory analyses, which we have applied in this analysis: $5.9, $24.3, $39, and $74.4 per metric ton of CO_2 emissions[25] in 2016, in 2007 dollars. The first three values are based on the average SCC from three integrated assessment models, at discount rates of 2.5%, 3%, and 5%, respectively. SCCs at several discount rates are included because the literature shows that the SCC is quite sensitive to assumptions about the discount rate, and because no consensus exists on the appropriate rate to use in an intergenerational context. The fourth value is the 95th percentile of the SCC from all three models at a 3% discount rate. It is included to represent higher-than-expected impacts from temperature change further out in the tails of the SCC distribution. Low probability, high impact events are incorporated into all of the SCC values through explicit consideration of their effects in two of the three models as well as the use of a probability density function for equilibrium climate sensitivity. Treating climate sensitivity probabilistically results in more high temperature outcomes, which in turn lead to higher projections of damages.

The SCC increases over time because future emissions are expected to produce larger incremental damages as physical and economic systems become more stressed in response to greater climatic change. Note that the interagency group estimated the growth rate of the SCC directly using the three integrated assessment models rather than assuming a constant annual

[24] Docket ID EPA-HQ-OAR-2009-0472-114577, *Technical Support Document: Social Cost of Carbon for Regulatory Impact Analysis Under Executive Order 12866,* Interagency Working Group on Social Cost of Carbon, with participation by Council of Economic Advisers, Council on Environmental Quality, Department of Agriculture, Department of Commerce, Department of Energy, Department of Transportation, Environmental Protection Agency, National Economic Council, Office of Energy and Climate Change, Office of Management and Budget, Office of Science and Technology Policy, and Department of Treasury (February 2010). Also available at http://www.epa.gov/otaq/climate/regulations.htm

[25] Note that upstream and downstream emission changes were not considered for this rule. For example, there may be changes in greenhouse gas emissions (in particular, methane) due to changes in fossil fuel extraction and transport in response to this proposal, but those emission changes were not quantified.

growth rate. This helps to ensure that the estimates are internally consistent with other modeling assumptions. The SCC estimates for the analysis year of 2016 in 2007$ are provided in Table 5-16.

Table 5-16. Social Cost of Carbon (SCC) Estimates (per tonne of CO_2) for 2016 (in 2007$)[a]

Discount Rate and Statistic		SCC Estimate, $
5%	Average	5.9
3%	Average	24.3
2.5%	Average	39.0
3%	95th percentile	74.4

[a] The SCC values are dollar-year and emissions-year specific. SCC values represent only a partial accounting of climate impacts.

When attempting to assess the incremental economic impacts of carbon dioxide emissions, the analyst faces a number of serious challenges. A recent report from the National Academies of Science (NRC 2009) points out that any assessment will suffer from uncertainty, speculation, and lack of information about (1) future emissions of greenhouse gases, (2) the effects of past and future emissions on the climate system, (3) the impact of changes in climate on the physical and biological environment, and (4) the translation of these environmental impacts into economic damages. As a result, any effort to quantify and monetize the harms associated with climate change will raise serious questions of science, economics, and ethics and should be viewed as provisional.

The interagency group noted a number of limitations to the SCC analysis, including the incomplete way in which the integrated assessment models capture catastrophic and non-catastrophic impacts, their incomplete treatment of adaptation and technological change, uncertainty in the extrapolation of damages to high temperatures, and assumptions regarding risk aversion. Current integrated assessment models do not assign value to all of the important physical, ecological, and economic impacts of climate change because models understandably lag behind the most recent research. The limited amount of research linking climate impacts to economic damages makes the interagency modeling exercise even more difficult. The interagency group hopes that over time researchers and modelers will work to fill these gaps and that the SCC estimates used for regulatory analysis by the federal government will continue to evolve with improvements in modeling. Additional details on these limitations are discussed in the SCC TSD.

In light of these limitations, the interagency group has committed to updating the current estimates as the science and economic understanding of climate change and its impacts on society improves over time. Specifically, the interagency group has set a preliminary goal of revisiting the SCC values in the next few years or at such time as substantially updated models become available, and to continue to support research in this area.

Applying the global SCC estimates shown in Table 5-16 to the estimated reductions in annual CO_2 emissions of 15 million metric tons for the policy scenario, we estimate the dollar value of the climate related co-benefits captured by the models for 2016 using three discount rates 5%, 3%, and 2.5% rather than 3% and 7%.[26] These climate co-benefit estimates are provided in Table 5-17. The CO_2 emission reductions associated with the policy scenario were developed using IPM and result largely from projected increases in electricity generation from natural gas sources and reductions in coal-fired generation by 2016. Even within the coal generation fleet, there are likely some modest generation shifts away from the least efficient units towards units that are more efficient to operate. These CO_2 emission reductions are net of any CO_2 emission increases associated with the energy usage for control technologies required by the rule.

Table 5-17. Monetized Co-Benefits of CO_2 Emissions Reductions in 2016 (in millions of 2007$)[a,b,c,d]

Discount Rate and Statistic		SCC Estimate, $
5%	Average	89
3%	Average	360
2.5%	Average	590
3%	95th percentile	1,100

[a] All estimates have been rounded.

[b] The SCC values are dollar-year and emissions-year specific.

[c] SCC values represent only a partial accounting of climate impacts.

[d] Three discount rates are used to estimate the dollar value of the climate related co-benefits.

As noted above, there are a number of limitations associated with the SCC and its use to assess the climate benefits of regulations. Beyond the SCC's incomplete treatment of impacts associated with CO_2 emissions, it is important to note that SCC is limited to assessing the

[26] See SCC TSD for more information about discount rate selection. Also, it is possible that other benefits or costs of proposed regulations unrelated to CO_2 emissions will be discounted at rates that differ from those used to develop the SCC estimates.

climate benefits associated with changes in CO_2 emissions only. However this rule will have an impact on the emissions of other pollutants that will affect the climate. These other pollutants include other greenhouse gases, aerosols and aerosols precursors such as black carbon, organic carbon, sulfur dioxide and nitrogen oxides, and ozone precursors such as nitrogen oxides and volatile organic carbon compounds. Changes in these pollutants (both increases and decreases) can be a direct result of changes in electricity generation, including but not limited to the changes in SO_2, NO_x, and filterable particulate matter identified in Chapter 3 of the RIA, but can also result from upstream changes in emissions due to changes in fossil fuel extraction and transport or downstream emission changes for secondary market impacts (not calculated for this rule). Reductions in black carbon or ozone precursors would lead to further cooling, but reductions in the other aerosol species and precursors would lead to warming. Therefore, changes in non-CO_2 pollutants could potentially augment or offset the climate benefits calculated here. These pollutants can act in different ways and on different timescales than carbon dioxide. For example, aerosols reflect (and in the case of black carbon, absorb) incoming radiation, whereas greenhouse gases absorb outgoing infrared radiation. These aerosols can also affect climate indirectly by altering properties of clouds. Black carbon can also deposit on snow and ice, darkening these surfaces and accelerating melting. In terms of lifetime, while carbon dioxide emissions can increase concentrations in the atmosphere for hundreds to thousands of years, many of these other pollutants are short lived and remain in the atmosphere for short periods of time ranging from days to weeks and can therefore exhibit large spatial and temporal variability. The climate impacts of these other pollutants can be complex and have not been calculated for this rule.

5.7 Co-Benefits Results

Applying the impact and valuation functions described previously in this chapter to the estimated changes in ambient PM yields estimates of the changes in physical damages (e.g., premature deaths, hospital admissions). Since the air quality modeling performed for this RIA does not reflect the changes in emissions of $PM_{2.5}$ precursors associated with the final emissions control requirements of the rule, we extrapolate the co-benefits of the final rule from the co-benefits of the air quality modeled emissions (see Appendices 5A and 5B). From these modeled co-benefits, we calculate BPT values for SO_2 and direct PM (carbonaceous and crustal), separately for Eastern and Western states, following the general methodology described by Fann et al. (2009). We then apply the BPT values to the final emission changes associated with the revised policy scenario. Since the geographic distribution of emission changes did not change dramatically from the modeled emission scenarios to the final policy scenario, extrapolating co-benefits using the BPT approach reasonably approximates the co-benefits of

the final policy scenario. However, there is additional uncertainty in the extrapolated benefits estimates relative to the benefits estimated for the air quality modeled emissions.

This section summarizes the health co-benefits estimated for the final policy scenario in 2016. Co-benefits associated with the modeled air quality changes are described in Appendix 5C. Although extrapolating recreational visibility impacts to the final revised policy scenario is not possible, we estimate that visibility co-benefits add $1.1 billion to the total monetized benefits of the modeled interim policy scenario (see Appendix 5C). Visibility benefits are not included in the co-benefits estimate for the final policy. Table 5-18 presents health impacts among eastern and western states. Monetized values for both health and welfare endpoints are presented in Table 5-19. All monetary benefits are in constant-year 2007$.

Not all known health and welfare co-benefits for non-HAP pollutants could be quantified or monetized in this analysis. The monetized value of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized co-benefits is thus equal to the subset of monetized PM- and CO_2-related health and welfare co-benefits plus B, the sum of the non-monetized health and welfare; this B represents both uncertainty and a bias in this analysis, as it reflects those co-benefits categories that we are unable quantify in this analysis.

This assessment estimates that in 2016 MATS will result in between 4,200 and 11,000 $PM_{2.5}$-related avoided premature deaths annually. The total monetized health and climate co-benefits of MATS in 2016 are between $37 billion and $90 billion using a 3% discount rate and between $33 billion and $81 using a 7% discount rate. As shown in Appendix 5C, 95% of the health co-benefits result from reduced exposure to sulfate particles. Mortality co-benefits account for approximately 93% to 97% of total monetized co-benefits depending on the $PM_{2.5}$ estimates used, in part because we are unable to quantify most of the non-health co-benefits. The next largest benefit is for reductions in chronic illness (CB and non-fatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Hospital admissions for respiratory and cardiovascular causes, visibility, MRADs and work loss days account for the majority of the remaining co-benefits. The remaining categories each account for a small percentage of total benefit; however, they represent a large number of avoided incidences affecting many individuals.

Figure 5-13 summarizes an array of $PM_{2.5}$-related monetized co-benefits estimates based on alternative epidemiology and expert-derived PM-mortality estimate. A comparison of the incidence table to the monetary co-benefits table reveals that there is not always a close

correspondence between the number of incidences avoided for a given endpoint and the monetary value associated with that endpoint. For example, there are over 100 times more work loss days than premature mortalities, yet work loss days account for only a very small fraction of total monetized co-benefits. This reflects the fact that many of the less severe health effects, while more common, are valued at a lower level than the more severe health effects. Also, some effects, such as hospital admissions, are valued using a proxy measure of WTP. As such, the true value of these effects may be higher than that reported in Table 5-19.

Table 5-18. Estimated Reduction in Incidence of Adverse Health Effects of the Mercury and Air Toxics Standards in 2016 (95% confidence intervals)[a,b]

Impact	Eastern U.S.[c]	Western U.S.	Total
Premature death			
Pope et al. (2002) (age >30)	4,100 (1,100 – 7,000)	130 (30 – 220)	4,200 (1,200 – 7,200)
Laden et al. (2006) (age >25)	10,000 (4,800 – 16,000)	320 (140 – 510)	11,000 (5,000 – 17,000)
Infant (< 1 year)	19 (-21 – 59)	1 (-1 – 2)	20 (-22 – 61)
Chronic bronchitis	2,700 (89 – 5,400)	100 (-1 – 210)	2,800 (88 – 5,600)
Non-fatal heart attacks (age > 18)	4,600 (1,200 – 8,100)	120 (25 – 210)	4,700 (1,200 – 8,300)
Hospital admissions—respiratory (all ages)	820 (320 – 1,300)	17 (6 – 27)	830 (330 – 1,300)
Hospital admissions—cardiovascular (age > 18)	1,800 (1,200 – 2,100)	42 (27 – 50)	1,800 (1,200 – 2,200)
Emergency room visits for asthma (age < 18)	3,000 (1,500 – 4,500)	110 (52 – 160)	3,100 (1,600 – 4,700)
Acute bronchitis (age 8-12)	6,000 (-1,400 – 13,000)	250 (-69 – 560)	6,300 (-1,400 – 14,000)
Lower respiratory symptoms (age 7-14)	77,000 (30,000 – 120,000)	3,100 (1,100 – 5,200)	80,000 (31,000 – 130,000)
Upper respiratory symptoms (asthmatics age 9-18)	58,000 (11,000 – 110,000)	2,400 (360 – 4,400)	60,000 (11,000 – 110,000)
Asthma exacerbation (asthmatics 6-18)	130,000 (4,500 – 430,000)	5,200 (-6 – 18,000)	130,000 (4,500 – 450,000)
Lost work days (ages 18-65)	520,000 (440,000 – 600,000)	21,000 (18,000 – 24,000)	540,000 (460,000 – 620,000)
Minor restricted-activity days (ages 18-65)	3,100,000 (2,500,000 – 3,700,000)	120,000 (99,000 – 150,000)	3,200,000 (2,600,000 – 3,800,000)

[a] Estimates rounded to two significant figures; column values will not sum to total value.

[b] The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts.

[c] Includes Texas and those states to the north and east.

Table 5-19. Estimated Economic Value of Health and Welfare co-benefits of the Mercury and Air Toxics Standards in 2016 (95% confidence intervals, billions of 2007$)[a]

Impact	Pollutant	Eastern U.S.[b]	Western U.S.	Total
Adult premature death (Pope et al. 2002 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$33 ($2.6 - $99)	$1.0 (<$0.01 - $3.1)	$34 ($2.6 - $100)
7% discount rate	$PM_{2.5}$	$30 ($2.3 - $90)	$0.9 (<$0.01 - $2.8)	$30($2.4 - $92)
Adult premature death (Laden et al. 2006 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$84 ($7.4 - $240)	$2.6 ($0.1 - $7.6)	$87 ($7.5 - $250)
7% discount rate	$PM_{2.5}$	$76 ($6.7 - $220)	$2.3 ($0.1 - $6.9)	$78 ($6.8 - $230)
Infant premature death	$PM_{2.5}$	$0.2 ($-0.2 – $0.8)	<$0.01	$0.2 ($-0.2 - $0.8)
Chronic Bronchitis	$PM_{2.5}$	$1.3 ($0.1 - $6.1)	$0.1 (<$0.01 - $0.2)	$1.4 ($0.1 - $6.4)
Non-fatal heart attacks				
3% discount rate	$PM_{2.5}$	$0.5 ($0.1 - $1.3)	<$0.01	$0.5 ($0.1 - $1.3)
7% discount rate	$PM_{2.5}$	$0.4 ($0.1 - $1.0)	<$0.01	$0.4 ($0.1 - $1.0)
Hospital admissions—respiratory	$PM_{2.5}$	$0.01 (<$0.01 - $0.02)	<$0.01	$0.01 ($0.01 - $0.02)
Hospital admissions—cardiovascular	$PM_{2.5}$	$0.03 (<$0.01 - $0.05)	<$0.01	$0.03 (<$0.01 - $0.05)
Emergency room visits for asthma	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Acute bronchitis	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lower respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Upper respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Asthma exacerbation	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lost work days	$PM_{2.5}$	$0.1 ($0.1 - $0.1)	<$0.01	$0.1 ($0.1 - $0.1)
Minor restricted-activity days	$PM_{2.5}$	$0.2 ($0.1 - $0.3)	<$0.01	$0.2 ($0.1 - $0.3)
CO_2-related co-benefits (3% discount rate)	CO_2			$0.36

(continued)

Table 5-19. Estimated Economic Value of Health and Welfare co-benefits of the Mercury and Air Toxics Standards in 2016 (95% confidence intervals, billions of 2007$)[a] (continued)

Total Monetized co-benefits (Pope et al. 2002 $PM_{2.5}$ mortality estimate)			
3% discount rate	$35+B ($2.8 - $110)	$1.1+B ($0.03 - $3.4)	$37+B ($3.2 - $110)
7% discount rate	$32+B ($2.5 - $98)	$1.0+B ($0.03 - $3.1)	$33+B ($2.9 - $100)
Total Monetized Benefits (Laden et al. 2006 $PM_{2.5}$ mortality estimate)			
3% discount rate	$87+B ($7.5 - $250)	$2.7+B ($0.1 - $7.9)	$90+B ($8.0 - $260)
7% discount rate	$78+B ($6.8 - $230)	$2.4+B ($0.1 - $7.2)	$81+B ($7.3 - $240)

[a] Estimates rounded to two significant figures. The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts. Confidence intervals reflect random sampling error and not the additional uncertainty associated with co-benefits scaling described above. The net present value of reduced CO_2 emissions are calculated differently than other co-benefits. The same discount rate used to discount the value of damages from future emissions (SCC at 5, 3, 2.5 percent) is used to calculate net present value of SCC for internal consistency. This table shows monetized CO_2 co-benefits at discount rates at 3 and 7 percent that were calculated using the global average SCC estimate at a 3% discount rate because the interagency workgroup on this topic deemed this marginal value to be the central value. In Section 5.6 we also report CO_2 co-benefits using discount rates of 5 percent (average), 2.5 percent (average), and 3 percent (95^{th} percentile).

[b] Includes Texas and those states to the north and east.

$PM_{2.5}$ mortality benefits represent a substantial proportion of total monetized co-benefits (over 90%), and these estimates have the following key assumptions and uncertainties.

1. The $PM_{2.5}$-related co-benefits were derived through a benefit per-ton approach, which does not fully reflect local variability in population density, meteorology, exposure, baseline health incidence rates, or other local factors that might lead to an over-estimate or under-estimate of the actual co-benefits of controlling PM precursors. In addition, differences in the distribution of emissions reductions across states between the modeled scenario and the final rule scenario add uncertainty to the final benefits estimates.

2. This rule is expected to reduce emissions of SO_2, NO_x, and directly emitted $PM_{2.5}$. We assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because $PM_{2.5}$ produced via transported precursors emitted from EGUs may differ significantly from direct $PM_{2.5}$ released from diesel engines and other industrial

sources, but the scientific evidence is not yet sufficient to allow differential effects estimates by particle type.

3. We assume that the health impact function for fine particles is linear within the range of ambient concentrations under consideration. Thus, the estimates include health co-benefits from reducing fine particles in areas with varied concentrations of $PM_{2.5}$, including both regions that are in attainment with fine particle standard and those that do not meet the standard down to the lowest modeled concentrations.

Based on our review of the current body of scientific literature, EPA estimated PM-related mortality without applying an assumed concentration threshold. EPA's Integrated Science Assessment for Particulate Matter (U.S. EPA, 2009a), which was reviewed by EPA's Clean Air Scientific Advisory Committee (U.S. EPA-SAB, 2009a; U.S. EPA-SAB, 2009b), concluded that the scientific literature consistently finds that a no-threshold log-linear model most adequately portrays the PM-mortality concentration-response relationship while also recognizing potential uncertainty about the exact shape of the concentration-response function. Consistent with this finding, we incorporated a "Lowest Measured Level" (LML) assessment, which is a method EPA has employed in several recent RIA's including the Cross-State Air Pollution Rule (U.S. EPA, 2011b).

This approach summarizes the distribution of avoided PM mortality impacts according to the baseline (i.e. pre-MATS) $PM_{2.5}$ levels experienced by the population receiving the $PM_{2.5}$ mortality benefit (Figures 5-14 and 5-15). We identify on this figure the lowest air quality levels measured in each of the two primary epidemiological studies EPA used to quantify PM-related mortality. This information allows readers to determine the portion of PM-related premature deaths avoided occurring at or above the LML of each study; in general, our confidence in the estimated PM-related premature deaths avoided decreases as we consider air quality levels further below the LML in the two epidemiological studies. While the LML analysis provides some insight into the level of uncertainty in the estimated PM mortality co-benefits, EPA does not view the LML as a threshold and continues to quantify PM-related mortality impacts using a full range of modeled air quality concentrations. For a summary of the scientific review statements regarding the lack of a threshold in the $PM_{2.5}$-mortality relationship, see the Technical Support Document (TSD) entitled *Summary of Expert Opinions on the Existence of a Threshold in the Concentration-Response Function for $PM_{2.5}$-related Mortality* (U.S. EPA, 2010e), which is provided in Appendix 5E of this RIA. While this figure describes the relationship between baseline $PM_{2.5}$ exposure and avoided premature deaths for the modeled air quality scenario, we expect the distribution of mortality impacts to be fairly similar between the two cases.

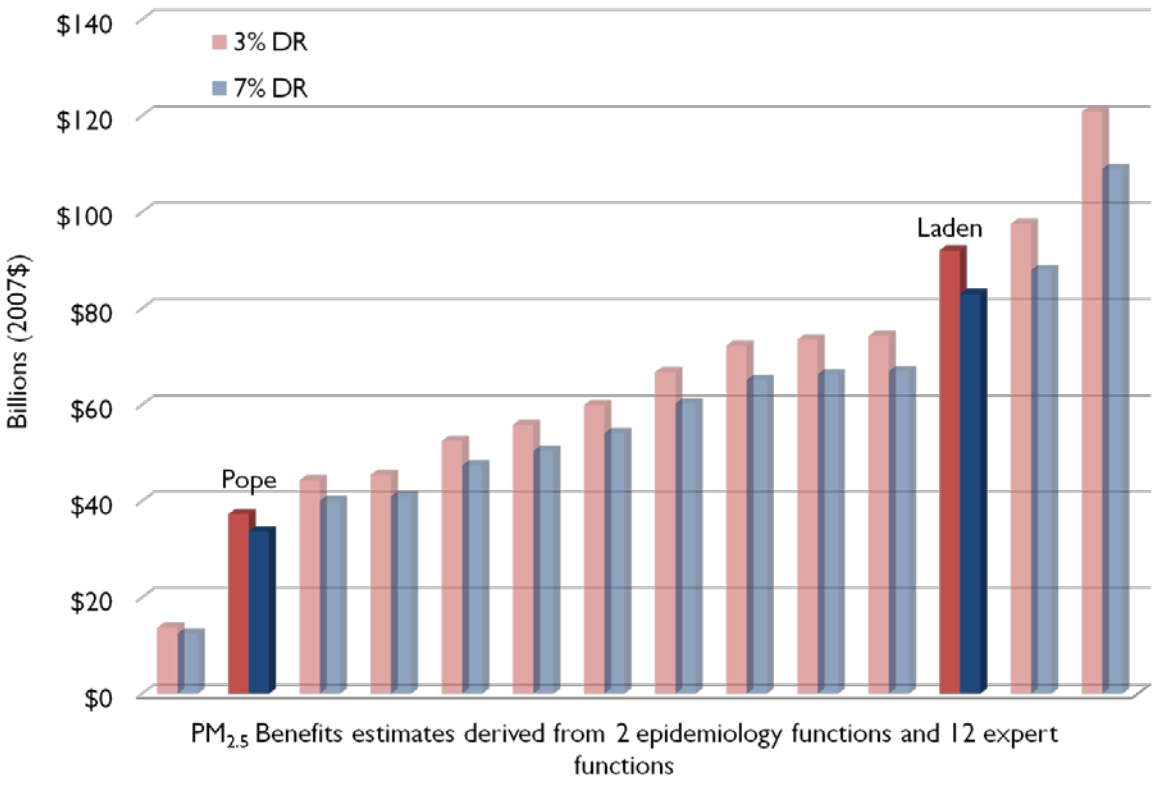

Figure 5-13. Economic Value of Estimated $PM_{2.5}$-Related Health co-benefits of the Mercury and Air Toxics Standards in 2016 According to Epidemiology or Expert-Derived PM Mortality Risk Estimate[a,b]

[a] Based on the modeled interim baseline, which is approximately equivalent to the final baseline (see Appendix 5A).

[b] Column total equals sum of $PM_{2.5}$-related mortality and morbidity co-benefits.

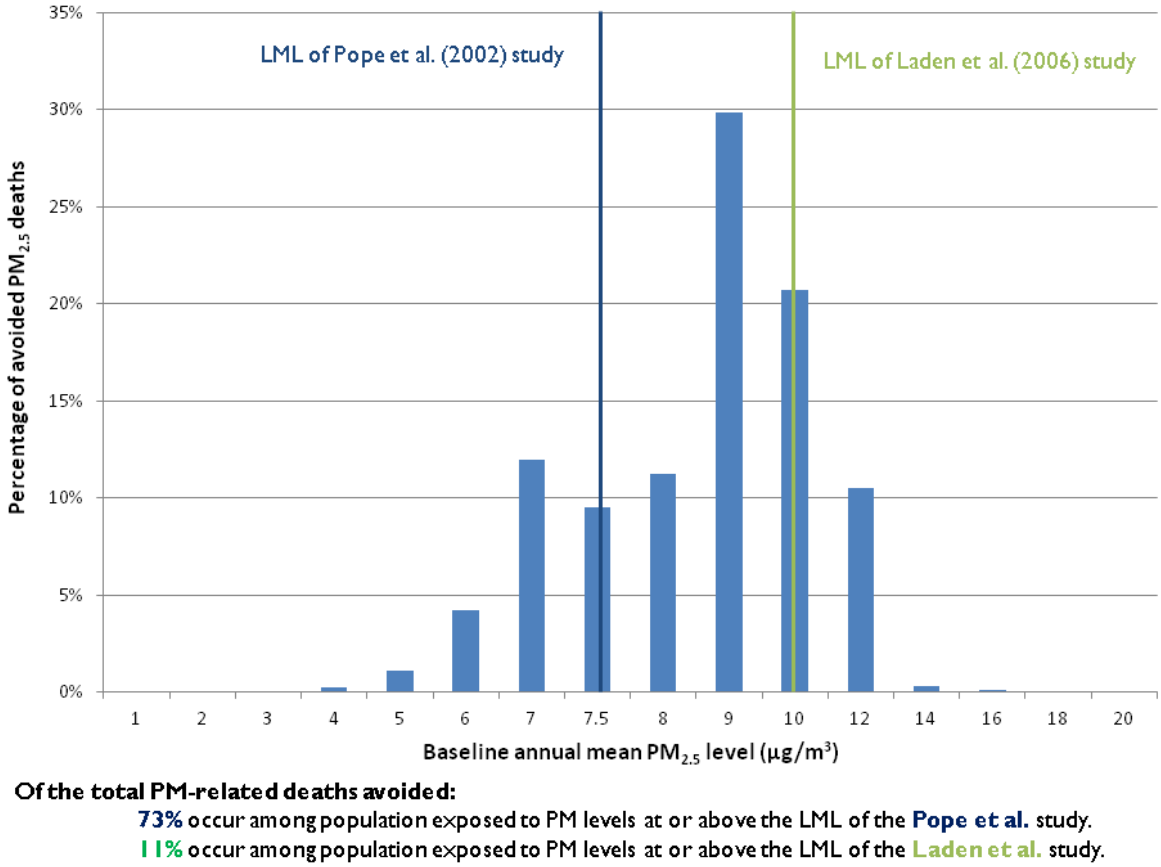

Figure 5-14. Percentage of Total PM-Related Mortalities of the Mercury and Air Toxics Standards in 2016 Avoided by Baseline Air Quality Level[a]

[a] Based on the modeled interim baseline, which is approximately equivalent to the final baseline (see Appendix 5A)

Some proportion of the avoided PM-related impacts we estimate in this analysis occur among populations exposed at or above the LML of the Laden et al. (2006) study, while a majority of the impacts occur at or above the LML of the Pope et al. (2002) study (Figure 5-14), increasing our confidence in the PM-related premature mortality analysis. Based on the modeled interim baseline which is approximately equivalent to the final baseline (see Appendix 5A), 11% and 73% of the estimated avoided premature deaths occur at or above an annual mean $PM_{2.5}$ level of 10 µg/m^3 (the LML of the Laden et al. 2006 study) and 7.5 µg/m^3 (the LML of the Pope et al. 2002 study), respectively. Using these percentages derived from the modeled interim baseline, Table 5-20 shows the allocation of reduced incidence above and below the LMLs of Laden et al. (2006) and Pope et al. (2002). As we model avoided premature deaths among populations exposed to levels of $PM_{2.5}$, we have lower confidence in levels below the LML for each study.

Table 5-20. Estimated Reduction in Incidence of Premature Adult Mortality due to the Mercury and Air Toxics Standards in 2016 Occurring Above and Below the Lowest Measured Levels in the Underlying Epidemiology Studies[a]

Study and Lowest Measured Level (LML)	Total Reduced Mortality Incidence	Allocation of Reduced Mortality Incidence	
		Below LML	At or Above LML
Pope et al. (2002), 7.5 μg/m^3	4,200	1,100	3,100
Laden et al. (2006), 10 μg/m^3	11,000	9,600	1,200

[a] These estimates are rounded to two significant digits. It is important to emphasize that although we have lower levels of confidence in levels below the LML for each study, the scientific evidence does not support the existence of a level below which health effects from exposure to $PM_{2.5}$ do not occur. See Appendix 5E for more information.

A large fraction of the $PM_{2.5}$-related benefits associated with this rule occur below the level of the National Ambient Air Quality Standard (NAAQS) for annual $PM_{2.5}$ at 15 μg/m^3, which was set in 2006. It is important to emphasize that NAAQS are not set at a level of zero risk. Instead, the NAAQS reflect the level determined by the Administrator to be protective of public health within an adequate margin of safety, taking into consideration effects on susceptible populations. While benefits occurring below the standard may be less certain than those occurring above the standard, EPA considers them to be legitimate components of the total benefits estimate.

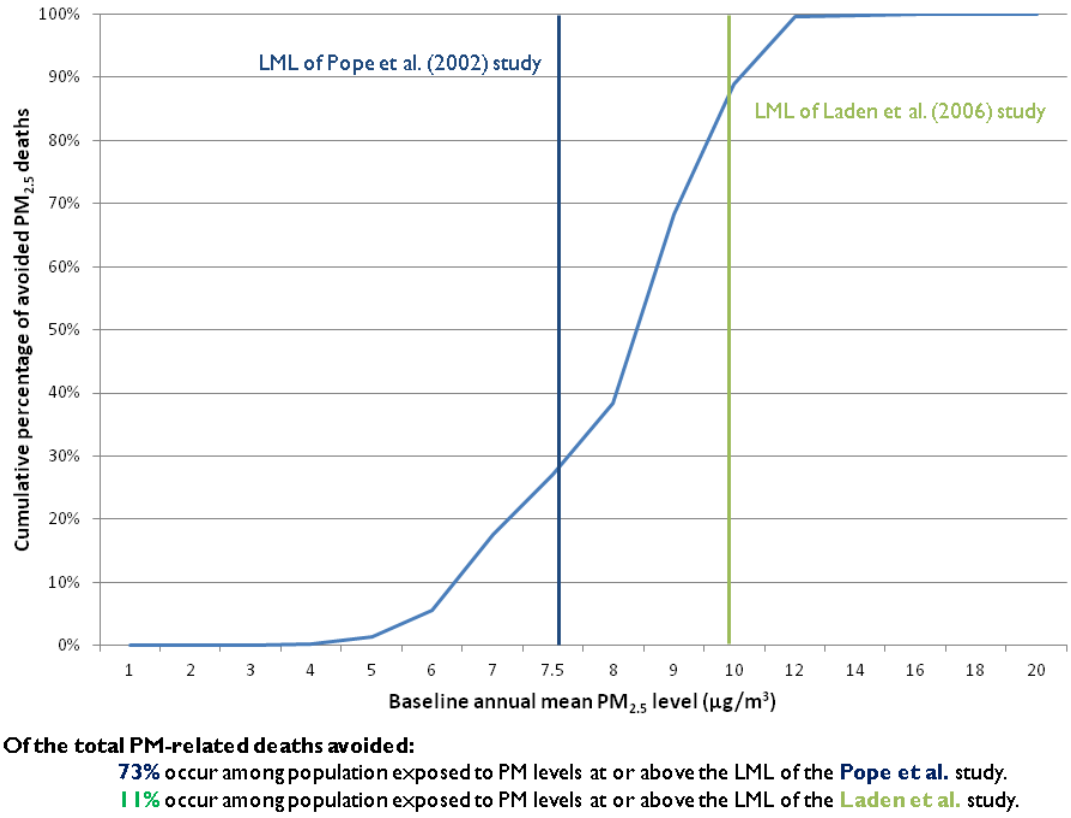

Figure 5-15. Cumulative Percentage of Total PM-Related Mortalities of the Mercury and Air Toxics Standards in 2016 Avoided by Baseline Air Quality Level[a]

[a] Based on the modeled interim baseline, which is approximately equivalent to the final baseline (see Appendix 5A)

While the LML of each study is important to consider when characterizing and interpreting the overall level $PM_{2.5}$-related co-benefits, as discussed earlier in this chapter, EPA believes that both cohort-based mortality estimates are suitable for use in air pollution health impact analyses. When estimating PM-related premature deaths avoided using risk coefficients drawn from the Laden et al. (2006) analysis of the Harvard Six Cities and the Pope et al. (2002) analysis of the American Cancer Society cohorts there are innumerable other attributes that may affect the size of the reported risk estimates—including differences in population demographics, the size of the cohort, activity patterns and particle composition among others. The LML assessment presented here provides a limited representation of one key difference between the two studies.

5.8 Discussion

This analysis demonstrates the significant health and welfare co-benefits of MATS. We estimate that in 2016 the rule will have reduced the number of $PM_{2.5}$-related premature deaths by between 4,200 and 11,000 and produce substantial non-mortality co-benefits. We estimate the monetized health and climate co-benefits of MATS to be $37 billion to $90 billion at a 3% discount rate and $33 billion to $81 billion at a 7% discount rate in 2016, depending on the epidemiological function used to estimate reductions in premature mortality. All estimates are in 2007$. Health co-benefits comprise approximately 99% of these total monetized co-benefits. This co-benefits assessment omits several important categories of co-benefits that we were unable to quantify, including health and ecological co-benefits from reducing exposure to ozone, ecosystem co-benefits for reducing nitrogen and sulfate deposition, the direct health co-benefits from reducing exposure to SO_2 and NO_2, and reduced visibility impairment in recreational areas. Inherent in any complex RIA such as this one are multiple sources of uncertainty. Some of these we characterized through our quantification of statistical error in the concentration response relationships and our use of the expert elicitation-derived $PM_{2.5}$ mortality functions. Others are unquantified, including the projection of atmospheric conditions and source-level emissions, the projection of baseline morbidity rates, incomes and technological development.

The emissions scenarios for the RIA reflects the Cross-State Air Pollution Rule (CSAPR) as finalized in July 2011 and the emissions reductions of SOx, NOx, directly emitted PM, and CO_2 are consistent with application of federal rules, state rules and statutes, and other binding, enforceable commitments in place by December 2010 for the analysis timeframe[27]. EPA has proposed minor modifications to the state level SO_2 budgets in the Cross State Air Pollution Rule (CSAPR; see http://www.epa.gov/airtransport/actions.html). These modifications are expected to result in small changes in the levels of SO_2 emission reductions expected in a number of states, with the largest impact expected in Texas. EPA expects that these changes will slightly reduce the benefits of CSAPR, and will have a small impact on the baseline emissions for MATS. Because of the change in the baseline SO_2 emissions for MATS, the MACT controls may result in slightly larger reductions in SO_2 and other emissions, and consequently slightly higher benefits. It is important to note that the total monetized benefits of both rules is not expected to change significantly, rather, the allocation of the SO_2 emissions reductions and

[27] Consistent with the mercury risk deposition modeling for MATS, EPA did not model non-federally enforceable mercury-specific emissions reduction rules in the base case or MATS policy case (see preamble Section III. A.). Note that this approach does not significantly affect SO_2 and NO_x projections underlying the cost and benefit results presented in this RIA.

benefits between the rules is changed, so that MATS accounts for slightly more of the total SO_2 emissions reductions and benefits, and CSAPR slightly less.

5.9 References

Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren, and P.K. Mills. 1995. Estimated Long-Term Ambient Concentrations of PM(10) and Development of Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health 50(2): 139-152.

Abbey, D.E., N. Nishino, W.F. McDonnell, R.J. Burchette, S.F. Knutsen, W. Lawrence Beeson, and J.X. Yang. 1999. Long-term inhalable particles and other air pollutants related to mortality in nonsmokers [see comments]. American Journal of Respiratory and Critical Care Medicine 159(2):373-382.

Abt Associates, Inc. April 2003. Proposed Nonroad Land-based Diesel Engine Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results. Prepared for Office of Air Quality Planning and Standards, U.S. EPA.

Abt Associates, Inc. 2005. U.S. EPA. Urban ornamental plants: sensitivity to ozone and potential economic losses. Memorandum to Bryan Hubbell and Zachary Pekar.

Abt Associates, Inc. 2010. Environmental Benefits and Mapping Program (Version 4.0). Bethesda, MD. Prepared for U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at <http://www.epa.gov/air/benmap>.

Adams PF, Hendershot GE, Marano MA. 1999. Current Estimates from the National Health Interview Survey, 1996. Vital Health Stat 10(200):1-212.

Adams, R. M., Glyer, J. D., Johnson, S. L., McCarl, B. A. 1989. A reassessment of the economic effects of ozone on U.S. agriculture. Journal of the Air Pollution Control Association, 39, 960-968.

Adams, R. M., Hamilton, S. A., McCarl, B. A. 1986. The benefits of pollution control: the case of ozone and U.S. agriculture. American Journal of Agricultural Economics, 34, 3-19.

Agency for Healthcare Research and Quality (AHRQ). 2000. HCUPnet, Healthcare Cost and Utilization Project.

American Lung Association (ALA). 2010. Trends in Asthma Morbidiy and Mortality. American Lung Association Epidemiology and Statistics Unit, Research and Program Services Division.

American Lung Association. 1999. Chronic Bronchitis. Available on the Internet at <http://www.lungusa.org/diseases/lungchronic.html>.

American Lung Association. 2002. Trends in Asthma Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit. Available on the Internet at <http://www.lungusa.org/data/asthma/ASTHMAdt.pdf>.

Banzhaf, S., D. Burtraw, D. Evans, and A. Krupnick. 2006. "Valuation of Natural Resource Improvements in the Adirondacks." Land Economics 82:445-464.

Berger, M.C., G.C. Blomquist, D. Kenkel, and G.S. Tolley. 1987. Valuing Changes in Health Risks: A Comparison of Alternative Measures. The Southern Economic Journal 53:977-984.

Brookshire, D.S., Thayer, M.A., Schulze, W.D. & D'Arge, R.C. 1982. "Valuing Public Goods: A Comparison of Survey and Hedonic Approaches." The American Economic Review. 72(1): 165-177.

Brown, L.H. 2002. Profile of the Annual Fall Foliage Tourist in Vermont: Travel Year 2001. Report prepared for the Vermont Department of Tourism and Marketing and the Vermont Tourism Data Center in association with the University of Vermont, Burlington, VT.

Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, et al. 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. Am J Epidemiol 153(5):444-452.

Carnethon, M.R., D. Liao, G.W. Evans, W.E. Cascio, L.E. Chambless, W.D. Rosamond, and G. Heiss. 2002. Does the Cardiac Autonomic Response to Postural Change Predict Incident Coronary Heart Disease and Mortality? The Atherosclerosis Risk in Communities Study.

Centers for Disease Control: Wide-ranging OnLine Data for Epidemiologic Research (CDC Wonder) (data from years 2004-2006), Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Available on the Internet at <http://wonder.cdc.gov>.

Centers for Disease Control and Prevention (CDC). 2009. *Summary Health Statistics for U.S. Children: National Health Interview Survey, 2008.* Vital and Health Statistics. Series 10, Number 244. U.S. Department of Health and Human Services. National Center for Health Statistics. December.

Chappelka, A.H., Samuelson, L.J. 1998. Ambient ozone effects on forest trees of the eastern United States: a review. New Phytologist, 139, 91-108.

Chen L, Jennison BL, Yang W, Omaye ST. 2000. Elementary school absenteeism and air pollution. Inhal Toxicol 12(11):997-1016.

Chestnut, L.G. and R.D. Rowe. 1990c. Economic Valuation of Changes in Visibility: A State of the Science Assessment for NAPAP. Section B5 in NAPAP State of Science and Technology Report 27.

Chestnut, L.G. April 15, 1997. Draft Memorandum: Methodology for Estimating Values for Changes in Visibility at National Parks.

Chestnut, L.G., and R.D. Rowe. 1990a. A New National Park Visibility Value Estimates. In Visibility and Fine Particles, Transactions of an AWMA/EPA International Specialty Conference, C.V. Mathai, ed. Air and Waste Management Association, Pittsburgh.

Chestnut, L.G., and R.D. Rowe. 1990b. Preservation Values for Visibility Protection at the National Parks: Draft Final Report. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC, and Air Quality Management Division, National Park Service, Denver, CO.

Cordell, H.K., C.J. Betz, G. Green, and M. Owens. 2005. Off-Highway Vehicle Recreation in the United States, Regions and States: A National Report from the National Survey on Recreation and the Environment (NSRE). Prepared for the U.S. Department of Agriculture Forest Service, Southern Research Station, National OHV Policy and Implementation Teams, Athens, GA. Available on the Internet at <http://www.fs.fed.us/recreation/programs/ohv/OHV_final_report.pdf>.

Cordell, K., B. Leeworthy, G.T. Green, C. Betz, B. Stephens. 2008. The National Survey on Recreation & the Environment. Research Work Unit 4953. Pioneering Research on Changing Forest Values in the South and Nation, U.S. Department of Agriculture Forest Service, Southern Research Station, Athens, GA. Available on the Internet at <http://www.srs.fs.fed.us/trends>.

Coulston, J.W., Riitters, K.H., Smith, G.C. 2004. A preliminary assessment of the Montreal process indicators of air pollution for the United States. Environmental Monitoring and Assessment, 95, 57-74.

Cropper, M. L. and A. J. Krupnick. 1990. The Social Costs of Chronic Heart and Lung Disease. Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.

Davidson K, Hallberg A, McCubbin D, Hubbell BJ. 2007. Analysis of $PM_{2.5}$ Using the Environmental Benefits Mapping and Analysis Program (BenMAP). J Toxicol Environ Health 70: 332—346.

De Steiguer, J., Pye, J., Love, C. 1990. Air Pollution Damage to U.S. Forests. Journal of Forestry, 88(8), 17-22.

DeHayes, D.H., P.G. Schaberg, G.J. Hawley, and G.R. Strimbeck. 1999. Acid rain impacts on calcium nutrition and forest health. Bioscience 49(10):789–800.

Dekker, J.M., R.S. Crow, A.R. Folsom, P.J. Hannan, D. Liao, C.A. Swenne, and E.G. Schouten. 2000. Low Heart Rate Variability in a 2-Minute Rhythm Strip Predicts Risk of Coronary Heart Disease and Mortality From Several Causes: The ARIC Study. Circulation 2000 102:1239-1244.

Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris, and F.E. Speizer. 1993. An Association between Air Pollution and Mortality in Six U.S. Cities. New England Journal of Medicine 329(24):1753-1759.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne, and F.E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children-Respiratory Symptoms. Environmental Health Perspectives 104(5):500-505.

Eisenstein, E.L., L.K. Shaw, K.J. Anstrom, C.L. Nelson, Z. Hakim, V. Hasselblad and D.B. Mark. 2001. Assessing the Clinical and Economic Burden of Coronary Artery Disease: 1986-1998. Medical Care 39(8):824-35.

Fann, N., C.M. Fulcher, B.J. Hubbell. 2009. The influence of location, source, and emission type in estimates of the human health benefits of reducing a ton of air pollution. Air Qual Atmos Health 2:169–176.

Felzer, B., J. Reilly, J. Melillo, D Kicklighter, M. Sarogim, C Wang, R. Prinn, Q. Zhuang. 2005. Future effects of Ozone on Carbon Sequestration and Climate Change Policy using a Global Biogeochemical Model. Climatic Change (2005) 73: 345–373

Fox, S., Mickler, R. A. (Eds.). 1996. Impact of Air Pollutants on Southern Pine Forests, Ecological Studies. (Vol. 118, 513 pp.) New York: Springer-Verlag.

Freeman(III), AM. 1993. The Measurement of Environmental and Resource Values: Theory and Methods. Washington, DC: Resources for the Future.

Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, et al. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. Epidemiology 12(1):43-54.

Gold, D.R., A. Litonjua, J. Schwartz, E. Lovett, A. Larson, B. Nearing, G. Allen, M. Verrier, R. Cherry., and R. Verrier. 2000. Ambient Pollution and Heart Rate Variability. Circulation 101(11):1267-73.

Grulke, N.E. 2003. The physiological basis of ozone injury assessment attributes in Sierran conifers. In A. Bytnerowicz, M.J. Arbaugh, & R. Alonso (Eds.), Ozone air pollution in the Sierra Nevada: Distribution and effects on forests. (pp. 55-81). New York, NY: Elsevier Science, Ltd.

Harrington, W., and P.R. Portney. 1987. Valuing the Benefits of Health and Safety Regulation. Journal of Urban Economics 22:101-112.

Heck, W.W. &Cowling E.B. 1997. The need for a long term cumulative secondary ozone standard – an ecological perspective. Environmental Management, January, 23-33.

Hollman, F.W., T.J. Mulder, and J.E. Kallan. January 2000. Methodology and Assumptions for the Population Projections of the United States: 1999 to 2100. Population Division Working Paper No. 38, Population Projections Branch, Population Division, U.S. Census Bureau, Department of Commerce.

Hubbell BJ, Fann N, Levy JI. 2009. Methodological Considerations in Developing Local-Scale Health Impact Assessments: Balancing National, Regional and Local Data. Air Qual Atmos Health doi: 10.1007/s11869-009-0037-z [online 31 March 2009].

Hubbell BJ, Hallberg A, McCubbin D, Post, E. 2005. Health-Related Benefits of Attaining the 8-Hr Ozone Standard. Environ Health Perspect 113: 73—82.

Hutchinson, J., D. Maynard, and L. Geiser. 1996. Air Quality and Lichens —A Literature Review Emphasizing the Pacific Northwest, USA. USDA Forest Service, Pacific Northwest Region Air Resource Management Program, U.S. Forest Service, U.S. Department of Agriculture.

Hutchison, R., and C.E. Kraft. 1994. Hmong Fishing Activity and Fish Consumption. Journal of Great Lakes Research 20(2):471–487.

Industrial Economics, Inc. 2006. Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between $PM_{2.5}$ Exposure and Mortality. Prepared for the U.S. EPA, Office of Air Quality Planning and Standards, September. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/Uncertainty/pm_ee_report.pdf>.

Industrial Economics, Incorporated (IEc). 1994. Memorandum to Jim DeMocker, Office of Air and Radiation, Office of Policy Analysis and Review, U.S. Environmental Protection Agency. March 31.

Intergovernmental Panel on Climate change (IPCC). 2007. Climate Change 2007 - Synthesis Report Contribution of Working Groups I, II and III to the Fourth Assessment Report of the IPCC.

Ito, K. 2003. Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan. In Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute, Boston, MA.

Jenkins, D.H., J. Sullivan, and G.S. Amacher. 2002. Valuing high altitude spruce-fir forest improvements: Importance of forest condition and recreation activity. Journal of Forest Economics 8:77–99.

Jerrett M, Burnett RT, Pope CA, III, et al. 2009. Long-Term Ozone Exposure and Mortality. N Engl J Med 360:1085-95.

Joslin, J.D., Kelly, J.M., van Miegroet, H. 1992. Soil chemistry and nutrition of North American spruce-fir stands: evidence for recent change. Journal of Environmental Quality, 21, 12-30.

Kaval, P., and J. Loomis. 2003. Updated Outdoor Recreation Use Values With Emphasis On National Park Recreation. Final Report October 2003, under Cooperative Agreement CA 1200-99-009, Project number IMDE-02-0070.

Kleckner, N., and J. Neumann. June 3, 1999. Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income. Memorandum to Jim Democker, U.S. EPA/OPAR.

Kochi, I., B. Hubbell, and R. Kramer. 2006. An Empirical Bayes Approach to Combining Estimates of the Value of Statistical Life for Environmental Policy Analysis. Environmental and Resource Economics. 34: 385-406.

Kopp, R. J., Vaughn, W. J., Hazilla, M., Carson, R. 1985. Implications of environmental policy for U.S. agriculture: the case of ambient ozone standards. Journal of Environmental Management, 20, 321-331.

Kramer, A., T. Holmes, and M. Haefel. 2003. Contingent valuation of forest ecosystem protection. Pp. 303–320 in Forests in a Market Economy. Edited by E.O. Sills and K.L. Abt. Dordrecht, The Netherlands: Kluwer Academic Publishers.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi, Y, et al. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. HEI Research Report, 140, Health Effects Institute, Boston, MA.

Krewski, D., R.T. Burnett, M.S. Goldbert, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz, and W.H. White. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute. Cambridge MA. July.

Krupnick, A.J., and M.L. Cropper. 1992. The Effect of Information on Health Risk Valuations. Journal of Risk and Uncertainty 5(2):29-48.

Kunzli, N., R. Kaiser, S. Medina, M. Studnicka, O. Chanel, P. Filliger, et al. 2000. Public-health imjpact of outdoor and traffic-related air pollution: A European Assessment. The Lancet 356(9232):795-801.

Kunzli, N., S. Medina, R. Kaiser, P. Quenel, F. Horak Jr, and M. Studnicka. 2001. Assessment of Deaths Attributable to Air Pollution: Should We Use Risk Estimates Based on Time Series or on Cohort Studies? American Journal of Epidemiology 153(11):1050-55.

Laden, F., J. Schwartz, F.E. Speizer, and D.W. Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality. American Journal of Respiratory and Critical Care Medicine 173:667-672.

Lave, L.B., and E.P. Seskin. 1977. Air Pollution and Human Health. Baltimore: Johns Hopkins University Press for Resources for the Future.

Leeworthy, V.R., and P.C. Wiley. 2001. Current Participation Patterns in Marine Recreation. Silver Spring, MD: U.S. Department of Commerce, National Oceanic and Atmospheric Administration, National Ocean Service, Special Projects. November. Available on the Internet at < http://www.naturetourismplanning.com/pdfs/NSRE_2.pdf>.

Leistritz, F. L., D. A. Bangsund, and N. M. Hodur. 2004. Assessing the Economic Impact of Invasive Weeds: The Case of Leafy Spurge (*Euphorbia esula*). Weed Technology 18:1392-1395.

Leitch, J. A, F. L Leistritz, and D. A Bangsund. 1996. Economic effect of leafy spurge in the upper Great Plains: methods, models, and results. Impact Assessment 14: 419–434.

Levy JI, Baxter LK, Schwartz J. 2009. Uncertainty and variability in health-related damages from coal-fired power plants in the United States. Risk Anal. doi: 10.1111/j.1539-6924.2009.01227.x [Online 9 Apr 2009]

Liao, D., J. Cai, W.D. Rosamond, R.W. Barnes, R.G. Hutchinson, E.A. Whitsel, P. Rautaharju, and G. Heiss. 1997. Cardiac Autonomic Function and Incident Coronary Heart Disease: A Population-Based Case-Cohort Study. The ARIC Study. Atherosclerosis Risk in Communities Study. American Journal of Epidemiology 145(8):696-706.

Liao, D., J. Creason, C. Shy, R. Williams, R. Watts, and R. Zweidinger. 1999. Daily Variation of Particulate Air Pollution and Poor Cardiac Autonomic Control in the Elderly. Environ Health Perspect 107:521-5.

Lipfert, F.W., H. Mitchell Perry Jr., J. Philip Miller, Jack D. Baty, Ronald E. Wyzg, and Sharon E. Carmody. 2000. The Washington University-EPRI Veterans' Cohort Mortality Study: Preliminary Results. Inhalation Toxicology 12:41-74.

Lipfert, F.W., S.C. Morris, and R.E. Wyzga. 1989. Acid Aerosols—the Next Criteria Air Pollutant. Environmental Science & Technology 23(11):1316-1322.

Lipfert, F.W.; Perry, H.M., Jr.; Miller, J.P.; Baty, J.D.; Wyzga, R.E.; Carmody, S.E. 2003. Air Pollution, Blood Pressure, and Their Long-Term Associations with Mortality. Inhalation Toxicology. 15, 493-512.

Lipfert, F.W.; Wyzga, R.E.; Baty, J.D.; Miller, J.P. 2006. Traffic Density as a Surrogate Measure of Environmental Exposures in Studies of Air Pollution Health Effects: Long- Term Mortality in a Cohort of US Veterans. Atmospheric Environment 40: 154-169.

Lipton, D. W. 1999. Pfiesteria's economic impact on seafood industry sales and recreational fishing, p. 35–38. In B. L. Gardner and L. Koch (ed.), Proceedings of the Conference, Economics of Policy Options for Nutrient Management and Pfiesteria. Center for Agricultural and Natural Resource Policy, University of Maryland, College Park.

Loehman, E.T., S. Park, and D. Boldt. 1994. "Willingness to Pay for Gains and Losses in Visibility and Health." Land Economics 70(4): 478-498.

Magari, S.R., R. Hauser, J. Schwartz, P.L. Williams, T.J. Smith, and D.C. Christiani. 2001. Association of Heart rate Variability with Occupational and Environmental Exposure to Particulate Air Pollution. Circulation 104(9):986-91.

Malm, WC. 1999. Introduction to visibility. Colorado State University, Fort Collins, CO, USA, 1983, Revised edition, 1999. Available on the Internet at <vista.cira.colostate.edu/improve/Education/IntroToVisinstr.htm>.

McBride, J.R., Miller, P.R., Laven, R.D. 1985. Effects of oxidant air pollutants on forest succession in the mixed conifer forest type of southern California. In: Air Pollutants Effects on Forest Ecosystems, Symposium Proceedings, St. P, 1985, p. 157-167.

McClelland, G., W. Schulze, D. Waldman, J. Irwin, D. Schenk, T. Stewart, L. Deck, and M. Thayer. September 1993. Valuing Eastern Visibility: A Field Test of the Contingent Valuation Method. Prepared for Office of Policy, Planning and Evaluation, U.S. Environmental Protection Agency. Available on the Internet at < http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0008-1.pdf/$file/EE-0008-1.pdf>.

Millennium Ecosystem Assessment Board (MEA). 2005. Ecosystems and Human Well-being: Synthesis. Washington, DC: World Resources Institute.

Miller, P.R., O.C. Taylor, R.G. Wilhour. 1982. Oxidant air pollution effects on a western coniferous forest ecosystem. Corvallis, OR: U.S. Environmental Protection Agency, Environmental Research Laboratory (EPA600-D-82-276).

Moolgavkar SH, Luebeck EG, Anderson EL. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis St. Paul and Birmingham. Epidemiology. 8(4):364-370.

Moolgavkar, S.H. 2000. Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas. Journal of the Air and Waste Management Association 50:1199-1206.

Moolgavkar, S.H. 2003. Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties. In Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston, MA: Health Effects Institute.

Mrozek, J.R., and L.O. Taylor. 2002. What Determines the Value of Life? A Meta-Analysis. Journal of Policy Analysis and Management 21(2):253-270.

National Agricultural Statistics Service (NASS). 2008. Maple Syrup – June 12, 2008: Maple Syrup Production Up 30 Percent Nationwide. U.S. Department of Agriculture, National Agricultural Statistics Service, New England Agricultural Statistics, Concord, NH.

National Center for Education Statistics (NCHS). 1996. The Condition of Education 1996, Indicator 42: Student Absenteeism and Tardiness. U.S. Department of Education. Washington, DC.

National Oceanic and Atmospheric Administration (NOAA). 2007. Annual Commercial Landing Statistics. August. Available on the Internet at <http://www.st.nmfs.noaa.gov/st1/commercial/landings/annual_landings.html>.

National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. Washington, DC: The National Academies Press.

National Research Council (NRC). 2008. Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. National Academies Press. Washington, DC.

National Research Council (2009). *Hidden Costs of Energy: Unpriced Consequences of Energy Production and Use*. National Academies Press. See docket ID EPA-HQ-OAR-2009-0472-11486.

Neumann, J.E., M.T. Dickie, and R.E. Unsworth. March 31, 1994. Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis—Draft Valuation Document. Industrial Economics Incorporated (IEc) Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review.

Norris, G., S.N. YoungPong, J.Q. Koenig, T.V. Larson, L. Sheppard, and J.W. Stout. 1999. An Association between Fine Particles and Asthma Emergency Department Visits for Children in Seattle. Environmental Health Perspectives 107(6):489-493.

Office of Management and Budget (OMB). 2003. *Circular A-4: Regulatory Analysis.* Washington, DC. Available on the Internet at http://www.whitehouse.gov/omb/circulars/a004/a-4.html.

Ostro, B., M. Lipsett, J. Mann, H. Braxton-Owens, and M. White. 2001. Air Pollution and Exacerbation of Asthma in African-American Children in Los Angeles. Epidemiology 12(2):200-208.

Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: A Specification Test. Journal of Environmental Economics Management 14:87-98.

Ostro, B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity: An Observational Study of Multiple Pollutants. Environmental Research 50:238-247.

Ozkaynak, H., and G.D. Thurston. 1987. Associations between 1980 U.S. Mortality Rates and Alternative Measures of Airborne Particle Concentration. Risk Analysis 7(4):449-461.

Peel, J. L., P. E. Tolbert, M. Klein, et al. 2005. Ambient air pollution and respiratory emergency department visits. Epidemiology. Vol. 16 (2): 164-74.

Peters, A., D.W. Dockery, J.E. Muller, and M.A. Mittleman. 2001. Increased Particulate Air Pollution and the Triggering of Myocardial Infarction. Circulation 103:2810-2815.

Peterson, D.E., M.S. Kanarek, M.A. Kuykendall, J.M. Diedrich, H.A. Anderson, P.L. Remington, and T.B. Sheffy. 1994. Fish Consumption Patterns and Blood Mercury Levels in Wisconsin Chippewa Indians. Archives of Environmental Health 49(1):53–58.

Pitchford, M. and W. Malm. 1993. Development and applications of a standard visual index. Atmospheric Environment 28(5):1049-1054.

Poloniecki, J.D., R.W. Atkinson., A.P. de Leon., and H.R. Anderson. 1997. Daily Time Series for Cardiovascular Hospital Admissions and Previous Day's Air Pollution in London, UK. Occupational and Environmental Medicine 54(8):535-540.

Pope, C.A., III, D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. Respiratory Health and PM_{10} Pollution: A Daily Time Series Analysis. American Review of Respiratory Diseases 144:668-674.

Pope, C.A., III, M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.W. Heath, Jr. 1995. Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults. American Journal of Respiratory Critical Care Medicine 151:669-674.

Pope, C.A., III, R.T. Burnett, G.D. Thurston, M.J. Thun, E.E. Calle, D. Krewski, and J.J. Godleski. 2004. Cardiovascular Mortality and Long-term Exposure to Particulate Air Pollution. Circulation 109: 71-77.

Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association 287:1132-1141.

Pope, CA III, E Majid, D Dockery. 2009. Fine Particle Air Pollution and Life Expectancy in the United States. New England Journal of Medicine 360: 376—386.

Prasad, A.M. and Iverson, L.R. 2003. Little's range and FIA importance value database for 135 eastern U.S. tree species. Northeastern Research Station, USDA Forest Service. Available on the Internet at http://www.fs.fed.us/ne/delaware/4153/global/littlefia/index.html

Pye, J.M. 1988. Impact of ozone on the growth and yield of trees: A review. Journal of Environmental Quality, 17, 347-360.

Rae, D.A. 1983. Benefits of Visual Air Quality in Cincinnati Results of a Contingent Ranking Survey. Prepared for Electric Power Research Institute. May.

Ransom, Michael, and C. Arden Pope. 1992. M.R. Ransom and C.A. Pope, III, Elementary school absences and PM_{10} pollution in Utah Valley. Environ. Res. 58, pp. 204–219.

Roman, Henry A., Katherine D. Walker, Tyra L. Walsh, Lisa Conner, Harvey M. Richmond, Bryan J. Hubbell, and Patrick L. Kinney. 2008. Expert Judgment Assessment of the Mortality Impact of Changes in Ambient Fine Particulate Matter in the U.S. Environ. Sci. Technol., 42(7):2268-2274.

Rowe, R.D., and L.G. Chestnut. 1986. Oxidants and Asthmatics in Los Angeles: A Benefits Analysis- Executive Summary. Prepared by Energy and Resource Consultants, Inc. Report to the U.S. Environmental Protection Agency, Office of Policy Analysis. EPA- 230-09-86-018. Washington, DC.

Rowlatt, P., Spackman, M., Jones, S., Jones-Lee, M., Loomes, G. 1998. Valuation of deaths from air pollution. A Report for the Department of Environment, Transport and the Regions and the Department of Trade and Industry. National Economic Research Associates (NERA), London.

Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel, and S.C. Hartz. 1998. Direct Medical Costs of Coronary Artery Disease in the United States. American Journal of Cardiology 81(9):1110-1115.

Samet, J.M., S.L. Zeger, F. Dominici, F. Curriero, I. Coursac, D.W. Dockery, J. Schwartz, and A. Zanobetti. 2000. The National Morbidity, Mortality and Air Pollution Study: Part II: Morbidity, Mortality and Air Pollution in the United States. Research Report No. 94, Part II. Health Effects Institute, Cambridge MA. June.

Schwartz J. 1994a. PM(10) Ozone, and Hospital Admissions For the Elderly in Minneapolis St Paul, Minnesota. Arch Environ Health. 49(5):366-374.

Schwartz J. 1994b. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. Am J Respir Crit Care Med. 150(3):648-655.

Schwartz J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. Thorax. 50(5):531-538.

Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease. EnvironmentResearch 62:7-13.

Schwartz, J. 2005. How sensitive is the association between ozone and daily deaths to control for temperature? Am J Respir Crit Care Med. Vol. 171 (6): 627-31.

Schwartz, J., and L.M. Neas. 2000. Fine Particles are More Strongly Associated than Coarse Particles with Acute Respiratory Health Effects in Schoolchildren. Epidemiology 11:6-10.

Sheppard, L. 2003. Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994. In Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston, MA: Health Effects Institute.

Shogren, J., and T. Stamland. 2002. Skill and the Value of Life. Journal of Political Economy 110:1168-1197.

Sisler, J.F. 1996. Spatial and seasonal patterns and long-term variability of the composition of the haze in the United States: an analysis of data from the IMPROVE network. CIRA Report, ISSN 0737-5352-32, Colorado State University.

Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista, and W.B. Saunders. 1997. A National Estimate of the Economic Costs of Asthma. American Journal of Respiratory and Critical Care Medicine 156(3 Pt 1):787-793.

Smith, V.K., Pattanayak, S.K., and Van Houtven. 2006. Structural benefit transfer: An example using VSL estimates. Ecological Economics 60(2):361-371.

Smith, V.K., G. Van Houtven, and S.K. Pattanayak. 2002. Benefit Transfer via Preference Calibration. Land Economics 78:132-152.

Spencer, D.M., and D.F. Holecek. 2007. Basic characteristics of the fall tourism market. Tourism Management 28:491–504.

Standard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter. Web site: http://www2standardandpoors.com.

Stanford, R., T. McLaughlin and L. J. Okamoto. 1999. The cost of asthma in the emergency department and hospital. Am J Respir Crit Care Med. Vol. 160 (1): 211-5.

Tagaris E, Liao KJ, Delucia AJ, et al. 2009. Potential impact of climate change on air-pollution related human health effects. Environ. Sci. Technol. 43: 4979—4988.

Taylor R. 1994. Deterministic versus stochastic evaluation of the aggregate economic effects of price support programs. Agricultural Systems 44: 461-473.

Tingey, D.T., and Taylor, G.E. 1982 Variation in plant response to ozone: a conceptual model of physiological events. In M.H. Unsworth & D.P. Omrod (Eds.), Effects of Gaseous Air Pollution in Agriculture and Horticulture. (pp.113-138). London, UK: Butterworth Scientific.

Tolley, G., A. Randall, G. Blomquist, M. Brien, R. Fabian, G. Fishelson, A. Frankel, M. Grenchik, J. Hoehn, A. Kelly, R. Krumm, E. Mensah, and T. Smith. 1986. Establishing and Valuing the Effects of Improved Visibility in Eastern United States. Prepared for U.S. Environmental Protection Agency, Office of Policy, Planning and Evaluation. U.S. Environmental Protection Agency Grant #807768-01-0.

Tolley, G.S. et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. University of Chicago. Final Report for the U.S. Environmental Protection Agency. January

Tsuji, H., M.G. Larson, F.J. Venditti, Jr., E.S. Manders, J.C. Evans, C.L. Feldman, D. Levy. 1996. Impact of Reduced Heart Rate Variability on Risk for Cardiac Events. The Framingham Heart Study. Circulation 94(11):2850-2855.

U.S. Bureau of Census. 2000. Population Projections of the United States by Age, Sex, Race, Hispanic Origin and Nativity: 1999 to 2100. Population Projections Program, Population Division, U.S. Census Bureau, Washington, DC. Available on the Internet at <http://www.census.gov/population/projections/nation/summary/np-t.txt>.

U.S. Department of Agriculture - Forest Service (USDA Forest Service). 2006. Ozone bioindicator data. Accessed 2006. Available on the Internet at <http://nrs.fs.fed.us/fia/topics/ozone/data/>.

U.S. Environmental Protection Agency (U.S. EPA). 1999. The Benefits and Costs of the Clean Air Act 1990 to 2010: EPA Report to Congress. Office of Air and Radiation, Office of Policy, Washington, DC. November. EPA report no. EPA410-R-99-001. Available on the Internet at <http://www.epa.gov/air/sect812/1990-2010/fullrept.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2000. Guidelines for Preparing Economic Analyses. EPA 240-R-00-003. National Center for Environmental Economics, Office of Policy Economics and Innovation. Washington, DC. September. Available on the Internet at <http://yosemite.epa.gov/ee/epa/eed.nsf/webpages/Guidelines.html/$file/cover.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2004. Air Quality Criteria for Particulate Matter Volume II of II. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Research Triangle Park, NC EPA/600/P-99/002bF. Available on the Internet at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=87903>.

U.S. Environmental Protection Agency (U.S. EPA). 2006a. Regulatory Impact Analysis, 2006 National Ambient Air Quality Standards for Particulate Matter, Chapter 5. Office of Air Quality Planning and Standards, Research Triangle Park, NC. October. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/RIAs/Chapter%205—Benefits.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2006b. Ecological Benefits Assessment Strategic Plan. EPA-240-R-06-001. Office of the Administrator. Washington, DC. October. Available on the Internet at <http://yosemite.epa.gov/ee/epa/eed.nsf/webpages/EcologBenefitsPlan.html>.

U.S. Environmental Protection Agency (U.S. EPA). 2006c. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final). EPA/600/R-05/004aF-cF. Washington, DC: U.S. EPA. February. Available on the Internet at http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=149923.

U.S. Environmental Protection Agency (U.S. EPA). 2007. Review of the National Ambient Air Quality Standards for Ozone: Policy assessment of scientific and technical information. Staff paper. Office of Air Quality Planning and Standards. EPA-452/R-07-007a. July. Available on the Internet at http://www.epa.gov/ttn/naaqs/standards/ozone/data/2007_07_ozone_staff_paper.pdf

U.S. Environmental Protection Agency (U.S. EPA). 2008a. Regulatory Impact Analysis, 2008 National Ambient Air Quality Standards for Ground-level Ozone, Chapter 6. Office of Air Quality Planning and Standards, Research Triangle Park, NC. March. Available at <http://www.epa.gov/ttn/ecas/regdata/RIAs/6-ozoneriachapter6.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2008b. Integrated Science Assessment for Oxides of Nitrogen and Sulfur –Ecological Criteria National (Final Report). National Center for Environmental Assessment, Research Triangle Park, NC. EPA/600/R-08/139. December. Available on the Internet at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=201485>.

U.S. Environmental Protection Agency (U.S. EPA). 2008c. Integrated Science Assessment for Oxides of Nitrogen - Health Criteria (Final Report). National Center for Environmental Assessment, Research Triangle Park, NC. July. Available on the Internet at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=194645

U.S. Environmental Protection Agency (U.S. EPA). 2009a. Integrated Science Assessment for Particulate Matter (Final Report). EPA-600-R-08-139F. National Center for Environmental Assessment – RTP Division. December. Available on the Internet at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546>.

U.S. Environmental Protection Agency (U.S. EPA). 2009b. Risk and Exposure Assessment for Review of the Secondary National Ambient Air Quality Standards for Oxides of Nitrogen and Oxides of Sulfur (Final). EPA-452/R-09-008a. Office of Air Quality Planning and Standards, Research Triangle Park, NC. September. Available on the Internet at <http://www.epa.gov/ttn/naaqs/standards/no2so2sec/data/NOxSOxREASep2009MainContent.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2010a. Final Regulatory Impact Analysis (RIA) for the SO_2 National Ambient Air Quality Standards (NAAQS). Office of Air Quality Planning and Standards, Research Triangle Park, NC. June. Available on the Internet at http://www.epa.gov/ttnecas1/regdata/RIAs/fso2ria100602full.pdf.

U.S. Environmental Protection Agency (U.S. EPA). 2010b. Final Regulatory Impact Analysis (RIA) for the NO_2 National Ambient Air Quality Standards (NAAQS). Office of Air Quality Planning and Standards, Research Triangle Park, NC. January. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/RIAs/FinalNO2RIAfulldocument.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2010c. Quantitative Health Risk Assessment for Particulate Matter. Office of Air Quality Planning and Standards, Research Triangle Park, NC. June. Available on the Internet at <http://www.epa.gov/ttnnaaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2010d. Guidelines for Preparing Economic Analyses. EPA 240-R-10-001. National Center for Environmental Economics, Office of Policy Economics and Innovation. Washington, DC. December. Available on the Internet at <http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0568-50.pdf/$file/EE-0568-50.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2010e. Technical Support Document: Summary of Expert Opinions on the Existence of a Threshold in the Concentration-Response Function for $PM_{2.5}$-related Mortality. Research Triangle Park, NC. June. Available on the Internet at <www.epa.gov/ttn/ecas/regdata/Benefits/**thresholds**tsd.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2011a. Proposed Regulatory Impact Analysis (RIA) for the Toxics Rule. Office of Air Quality Planning and Standards, Research Triangle Park, NC. March. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/RIAs/ToxicsRuleRIA.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2011b. Regulatory Impact Analysis for the Federal Implementation Plans to Reduce Interstate Transport of Fine Particulate Matter and Ozone in 27 States; Correction of SIP Approvals for 22 States. Office of Air Quality Planning and Standards, Research Triangle Park, NC. June. Available on the Internet at <http://www.epa.gov/airtransport/pdfs/FinalRIA.pdf>.

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 1999. An SAB Advisory: The Clean Air Act Section 812 Prospective Study Health and Ecological Initial Studies. Prepared by the Health and Ecological Effects Subcommittee (HEES) of the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington DC. EPA-SAB-COUNCIL-ADV-99-005.

U.S. Environmental Protection Agency – Science Advisory Board (U.S. EPA-SAB). 2000. An SAB Report on EPA's White Paper Valuing the Benefits of Fatal Cancer Risk Reduction. EPA-SAB-EEAC-00-013.

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2004a. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis – Benefits and Costs of the Clean Air Act, 1990-2020. EPA-SAB-COUNCIL-ADV-04- 002 March.

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2004b. Review of the Draft Analytical Plan for EPA's Second Prospective Analysis – Benefits and Costs of the Clean Air Act, 1990-2020: An Advisory by the Advisory Council for Clean Air Compliance Analysis. EPA-SAB-COUNCIL-ADV-04- 004. May. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/7CCBBFE15CD4C8B185256F17005E3079/$File/council_adv_04004.pdf>.

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2004c. Advisory Council on Clean Air Compliance Analysis Response to Agency Request on Cessation Lag. EPA-COUNCIL-LTR-05-001. December. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/0/39F44B098DB49F3C85257170005293E0/$File/council_ltr_05_001.pdf>.

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2007. Clean Air Scientific Advisory Committee's (CASAC) Review of the Agency's Final Ozone Staff Paper. EPA-CASAC-07-002. Available on the Internet at http://yosemite.epa.gov/sab/sabproduct.nsf/FE915E916333D776852572AC007397B5/$File/casac-07-002.pdf

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2009a. Consultation on EPA's Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment. EPA-COUNCIL-09-009. May. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/723FE644C5D758DF852575BD00763A32/$File/EPA-CASAC-09-009-unsigned.pdf>.

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2009b. Review of EPA's Integrated Science Assessment for Particulate Matter (First External Review Draft, December 2008). EPA-COUNCIL-09-008. May. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/73ACCA834AB44A10852575BD0064346B/$File/EPA-CASAC-09-008-unsigned.pdf>.

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2010. Review of EPA's DRAFT Health Benefits of the Second Section 812 Prospective Study of the Clean Air Act. EPA-COUNCIL-10-001. June. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/9288428b8eeea4c885257242006935a3/59e06b6c5ca66597852575e7006c5d09!OpenDocument&TableRow=2.3#2.>.

U.S. Fish and Wildlife Service and U.S. Census Bureau (FWS and Census). 2007. 2006 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation (FHWAR). FHW/06-NAT. U.S. Department of the Interior, U.S. Fish and Wildlife Service, Washington, DC, and U.S. Department of Commerce, U.S. Census Bureau, Washington, DC.

U.S. Food and Drug Administration (U.S. FDA). 2001. Fish and Fisheries Products Hazards and Controls Guidance: Chapter 10: Methyl Mercury. Third Edition. June. Available on the Internet at < http://www.fda.gov/Food/GuidanceComplianceRegulatoryInformation/GuidanceDocuments/Seafood/ucm092041.htm>.

U.S. Forest Service (USFS). 2006. Forest Inventory and Analysis National Program: Forest Inventory Data Online. Online database. U.S. Department of Agriculture Forest Service, Forest Inventory and Analysis, Arlington, VA. Available on the Internet at <http://fia.fs.fed.us/tools-data>.

Valigura, R.A., R.B. Alexander, M.S. Castro, T.P. Meyers, H.W. Paerl, P.E. Stacy, and R.E. Turner. 2001. Nitrogen Loading in Coastal Water Bodies: An Atmospheric Perspective. Washington, DC: American Geophysical Union

Van Sickle, J., Baker, J.P., Simonin, H.A., Baldigo, B.P., Kretser, W.A., Sharpe, W.E. 1996. Episodic acidification of small streams in the northeastern United States: Fish mortality in field bioassays. Ecological Applications, 6, 408-421.

Vedal, S., J. Petkau, R. White, and J. Blair. 1998. Acute Effects of Ambient Inhalable Particles in Asthmatic and Nonasthmatic Children. American Journal of Respiratory and Critical Care Medicine 157(4):1034-1043.

Viscusi, V.K., and J.E. Aldy. 2003. The Value of a Statistical Life: A Critical Review of Market Estimates throughout the World. Journal of Risk and Uncertainty 27(1):5-76.

Viscusi, W.K., W.A. Magat, and J. Huber. 1991. Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Trade-Offs for Chronic Bronchitis. Journal of Environmental Economics and Management 21:32-51.

Wilson, A. M., C. P. Wake, T. Kelly, et al. 2005. Air pollution, weather, and respiratory emergency room visits in two northern New England cities: an ecological time-series study. Environ Res. Vol. 97 (3): 312-21.

Winner, W.E. 1994. Mechanistic analysis of plant responses to air pollution. Ecological Applications, 4(4), 651-661.

Winner, W.E., and C.J. Atkinson. 1986. Absorption of air pollution by plants, and consequences for growth. Trends in Ecology and Evolution 1:15-18.

Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. Medical Costs of Coronary Artery Disease in the United States. American Journal of Cardiology 65(7):432-440.

Woodruff TJ, Parker JD, Schoendorf KC. 2006. Fine particulate matter ($PM_{2.5}$) air pollution and selected causes of postneonatal infant mortality in California. Environmental Health Perspectives 114(5):786-90.

Woods & Poole Economics Inc. 2008. Population by Single Year of Age CD. CD-ROM. Woods & Poole Economics, Inc. Washington, D.C.

World Health Organization (WHO). 1977. International Classification of Diseases, 9th revision (ICD-9). Geneva:World Health Organization.

APPENDIX 5A
IMPACT OF THE INTERIM POLICY SCENARIO ON EMISSIONS

5A.1 Introduction

This section summarizes the emissions inventories that are used to create emissions inputs to the air quality modeling performed for this rule. A summary of the emissions reductions that were modeled for this rule is provided. Note that the emissions processing and corresponding air quality modeling were used to develop BPT scaling factors for the benefits calculation as described in this RIA. More information on this approach can be found in Appendix 5C. The emissions inventories were processed into the form required by the Community Multi-scale Air Quality (CMAQ) model. CMAQ simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone, particulate matter and air toxics.

As part of the analysis for this rulemaking, the modeling system was used to calculate daily and annual $PM_{2.5}$ concentrations, 8-hr maximum ozone and visibility impairment. Model predictions of $PM_{2.5}$ and ozone are used in a relative sense to estimate scenario-specific, future-year design values of $PM_{2.5}$ and ozone. These are combined with monitoring data to estimate population-level exposures to changes in ambient concentrations for use in estimating health and welfare effects. In the remainder of this section we provide an overview of (1) the emissions components of the modeling platform, (2) the development of the 2005 base year emissions, (3) the development of the future year baseline emissions, and (4) the development of the future year control case emissions.

5A.2 Overview of Modeling Platform and Emissions Processing Performed

A modeling platform is the collection of the inputs to an air quality model, including the settings and data used for the model, including emissions data, meteorology, initial conditions, and boundary conditions. The 2005-based air quality modeling platform used for this RIA includes 2005 base year emissions and 2005 meteorology for modeling ozone and $PM_{2.5}$ with CMAQ. In support of this rule, EPA modeled the air quality in the Eastern and the Western United States using two separate model runs, each with a horizontal grid resolution of 12 km x 12 km. These 12 km modeling domains were “nested” within a modeling domain covering the remainder of the lower 48 states and surrounding areas using a grid resolution of 36 x 36 km. The results from the 36-km modeling were used to provide incoming “boundary” for the 12km grids. Additional details on the non-emissions portion of the 2005v4.3 modeling platform used for this RIA are described in the air quality modeling section (Appendix 5B).

The 2005-based air quality modeling platform used in support of this RIA is version 4.3 and is referred to as the 2005v4.3 platform. It is an update to the 2005-based platform, version 4.1 (i.e., 2005v4.1) used for the proposal modeling and for the appropriate and necessary finding. The Technical Support Document "Preparation of Emissions Inventories for the Version 4.1, 2005-based Platform" (see http://www.epa.gov/ttn/chief/emch/index.html#toxics) provides information on the platform used for the proposed version of this rule and for the appropriate and necessary finding. The 2005v4.3 platform builds upon the 2005-based platform, version 4.2 which was the version of the platform used for the final Cross-State Air Pollution Rule and incorporated changes made in response to public comments on the proposed version of that rule. Detailed documentation about the 2005v4.3 platform emissions inventories used for this rule can be found in the "Emissions Modeling for the Final Mercury and Air Toxics Standards Technical Support Document".

5A.3 Development of 2005 Base Year Emissions

Emissions inventory inputs representing the year 2005 were developed to provide a base year for forecasting future air quality. The emission source sectors and the basis for current and future-year inventories include Electric Generating Utility point sources, non-EGU point sources, and the following types of sources with inventories primarily at the county level: onroad mobile, nonroad mobile, nonpoint, and fires. The specific sectors used for modeling are listed and defined in detail in the "Emissions Modeling for the Final Mercury and Air Toxics Standards Technical Support Document". The inventories used include emissions of criteria pollutants, and for some sectors the pollutants benzene, formaldehyde, acetaldehyde and methanol are used to speciate VOC into the chemical species needed by CMAQ.

The 2005v4 platform was the initial starting point for the 2005v4.3 platform used for this modeling. There were two intermediate versions: the version used for the MATS proposal modeling (2005v4.1), and the version used for the final Cross-State Air Pollution Rule modeling (2005v4.2). The basis of the 2005v4 platform and subsequent versions is the U.S. inventory is the 2005 National Emission Inventory (NEI), version 2 from October 6, 2008 (http://www.epa.gov/ttn/chief/net/2005inventory.html). The 2005 NEI v2 includes 2005-specific data for point and mobile sources, while most nonpoint data were carried forward from version 3 of the 2002 NEI.

Emissions for point sources were primarily from the 2005 NEI v2 inventory, consisting mostly of 2005 values with some 2002 emissions values used where 2005 data were not available. The point sources are split into "EGU" (aka "ptipm") and "Non-EGU" (aka

"ptnonipm") sectors for modeling purposes based on the matching of the unit level data in the NEI units in the National Electric Energy Database System (NEEDS) version 4.10 database. All units that matched NEEDS were included in the EGU sector so that the future year emissions could easily be taken from the Integrated Planning Model (IPM) as its outputs are also based on the NEEDS units. Efforts made to ensure the quality of the 2005 EGU inventory included ensuring that there were not duplicate emissions (e.g., data pulled forward from earlier inventories), accounting for plants or units that shutdown prior to 2005, adding estimates for ethanol plants, and accounting for installed emissions control devices.

The 2005 annual NO_X and SO_2 emissions for sources in the EGU sector are based primarily on data from EPA's Clean Air Markets Division's Continuous Emissions Monitoring (CEM) program, with other pollutants estimated using emission factors and the CEM annual heat input. For EGUs without CEMs, emissions were obtained from the state-submitted data in the NEI. For the 2005 base year, the annual EGU NEI emissions were allocated to hourly emissions values needed for modeling based on the 2004, 2005, and 2006 CEM data. The NO_X CEM data were used to create NO_X-specific profiles, the SO_2 data were used to create SO_2-specific profiles, and the heat input data were used to allocate all other pollutants. The three years of data were used to create monthly profiles by state, while the 2005 data were used to create state-averaged profiles for allocating monthly emissions to daily. These daily values were input into SMOKE, which utilized state-averaged 2005-based hourly profiles to allocate to hourly values. This approach to temporal allocation was used for all base and control cases modeled to provide a temporal consistency between the years modeled without tying the temporalization to the events of a single year.

For nonpoint sources, the 2002 NEI v2 inventory was augmented with updated oil and gas exploration emissions for Texas and Oklahoma (for CO, NO_X, PM, SO_2, VOC). These oil and gas exploration emissions were in addition to oil and gas data previously available in the 2005 v4 platform that includes emissions within the following states: Arizona, Colorado, Montana, Nevada, New Mexico, North Dakota, Oregon, South Dakota, Utah, and Wyoming.

The commercial marine category 3 (C3) vessel emissions portion of the nonroad sector used point-based gridded 2005 emissions that reflect the final projections developed for the category 3 commercial marine Emissions Control Area (ECA) proposal to the International Maritime Organization (EPA-420-F-10-041, August 2010). These emissions include Canada as part of the ECA, and were updated using region-specific growth rates and thus contain Canadian province codes. The state/federal water boundaries were based on a file available

from the Mineral Management Service (MMS) that specifies boundaries ranging from three to ten nautical miles from the coast.

The onroad emissions were primarily based on the version of the Motor Vehicle Emissions Simulator (MOVES) (http://www.epa.gov/otaq/models/moves/) used for the Tier 3 proposed rule. The first step was to run MOVES to output emission factors for a set of counties with characteristics representative of the counties within the continental United States. Data for each representative county included county-specific fuels, vehicle age distribution, inspection and maintenance programs, temperatures and relative humidity. The emission factors produced by MOVES were then combined by SMOKE with county-based activity data (vehicle miles traveled, speed data, and vehicle population) and gridded temperature data to create hourly, gridded emissions. Additional information on this approach is available in the "Emissions Modeling for the Final Mercury and Air Toxics Standards Technical Support Document".

The nonroad emissions utilized the National Mobile Inventory Model (NMIM) to run the NONROAD model for all states to create county/month emissions, updated from the annual emissions in the 2005 NEI v2 with some improvements. For this case, NMIM was run using representing county mode, with improved fuels, an improved toxics emission factor (1,3-butadiene for 2-stroke snowmobiles), and a small coding change that enabled NONROAD to process 15% ethanol (E15) fuels.

Other emissions inventories used included average-year county-based inventories for emissions from wildfires and prescribed burning. These emissions are intended to be representative of both base and future years and are held constant for each. As a result, post-processing techniques minimize their impact on the modeling results. The 2005v4.3 platform utilizes the same 2006 Canadian inventory and a 1999 Mexican inventory as were used since the v4 platform, as these were the latest available data from these countries.

Once developed, the emissions inventories were processed to provide the hourly, gridded emissions for the model-species needed by CMAQ. Details on this processing are further described in the "Emissions Modeling for the Final Mercury and Air Toxics Standards Technical Support Document". Table 5A-1 provides summaries of the 2005 U.S. emissions inventories modeled for this rule by sector. Tables 5A-2 through 5A-3 provide state-level summaries of SO_2, and $PM_{2.5}$ by sector. Note that the nonroad columns include emissions from traditional nonroad sources that are found "on-land," along with commercial marine sources. The nonpoint columns include area fugitive dust, agriculture, and other nonpoint emissions.

Table 5A-1. 2005 US Emissions by Sector

Emissions Sector	2005 NO_X [tons/yr]	2005 SO_2 [tons/yr]	2005 $PM_{2.5}$ [tons/yr]	2005 PM_{10} [tons/yr]	2005 NH_3 [tons/yr]	2005 CO [tons/yr]	2005 VOC [tons/yr]
Agriculture					3,251,990		
Area fugitive dust			1,030,391	8,858,992			
Average fires	189,428	49,094	684,035	796,229	36,777	8,554,551	1,958,992
Commercial marine Category 3 (US)	130,164	97,485	10,673	11,628		11,862	4,570
EGU	3,729,161	10,380,883	496,877	602,236	21,995	603,788	41,089
Locomotive/marine	1,922,723	153,068	56,666	59,342	773	270,007	67,690
Non-EGU point	2,213,471	2,030,759	433,346	647,873	158,342	3,201,418	1,279,308
Nonpoint	1,696,902	1,216,362	1,079,906	1,349,639	133,962	7,410,946	7,560,061
Nonroad	2,031,527	196,277	201,406	210,767	1,971	20,742,873	2,806,422
Onroad	8,235,002	168,480	301,073	369,911	144,409	41,117,658	3,267,931
US Total	**20,148,378**	**14,292,410**	**4,294,373**	**12,906,616**	**3,750,218**	**81,913,104**	**16,986,064**

Table 5A-2. 2005 Base Year SO_2 Emissions (tons/year) for States by Sector

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Alabama	460,123	66,373	52,325	5,622	3,554	983	588,980
Arizona	52,733	23,966	2,571	6,151	3,622	2,888	91,931
Arkansas	66,384	13,039	27,260	5,678	1,918	728	115,008
California	601	33,097	77,672	40,222	4,526	6,735	162,852
Colorado	64,174	1,550	6,810	4,897	2,948	1,719	82,098

(continued)

Table 5A-2. 2005 Base Year SO_2 Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Connecticut	10,356	1,831	18,455	2,557	1,337	4	34,540
Delaware	32,378	34,859	1,030	2,657	486	6	71,416
District of Columbia	1,082	686	1,559	414	205	0	3,947
Florida	417,321	57,429	70,490	31,190	12,388	7,018	595,836
Georgia	616,063	52,827	56,829	9,224	6,939	2,010	743,893
Idaho	0	17,151	2,915	2,304	902	3,845	27,117
Illinois	330,382	131,357	5,395	19,305	6,881	20	493,339
Indiana	878,979	86,337	59,775	9,437	4,641	24	1,039,194
Iowa	130,264	41,010	19,832	8,838	2,036	25	202,004
Kansas	136,520	12,926	36,381	8,035	1,978	103	195,943
Kentucky	502,731	25,808	34,229	6,943	3,240	364	573,315
Louisiana	109,875	165,705	2,378	25,451	2,902	892	307,202
Maine	3,887	18,512	9,969	1,625	963	150	35,106
Maryland	283,205	34,988	40,864	9,353	3,016	32	371,458
Massachusetts	84,234	19,620	25,261	6,524	2,669	93	138,402
Michigan	349,877	76,510	42,066	14,626	8,253	91	491,423
Minnesota	101,678	24,603	14,747	10,409	2,934	631	155,002
Mississippi	75,047	29,892	6,796	5,930	2,590	1,051	121,306
Missouri	284,384	78,308	44,573	10,464	4,901	186	422,816
Montana	19,715	11,056	2,600	3,813	874	1,422	39,480
Nebraska	74,955	7,910	7,659	9,199	1,510	105	101,337
Nevada	53,363	2,253	12,477	2,880	656	1,346	72,975
New Hampshire	51,445	3,155	7,408	789	746	38	63,580
New Jersey	57,044	7,639	10,726	13,321	3,038	61	91,830
New Mexico	30,628	7,831	3,193	3,541	1,801	3,450	50,445
New York	180,847	58,426	125,158	15,666	6,258	113	386,468
North Carolina	512,231	59,433	22,020	8,766	6,287	696	609,433
North Dakota	137,371	9,582	6,455	5,986	533	66	159,994
Ohio	1,116,095	115,155	19,810	15,425	7,336	22	1,273,843

(continued)

Table 5A-2. 2005 Base Year SO_2 Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Oklahoma	110,081	40,482	8,556	5,015	3,039	469	167,642
Oregon	12,304	9,825	9,845	5,697	1,790	4,896	44,357
Pennsylvania	1,002,203	83,375	68,349	11,999	6,266	32	1,172,224
Rhode Island	176	2,743	3,365	816	254	1	7,354
South Carolina	218,781	31,495	13,489	7,719	3,589	646	275,719
South Dakota	12,215	1,702	10,347	3,412	623	498	28,797
Tennessee	266,148	65,693	32,714	6,288	5,538	277	376,659
Texas	534,949	223,625	115,192	34,944	16,592	1,178	926,480
Tribal	3	1,511	0	0	0	0	1,515
Utah	34,813	9,132	3,577	2,439	1,890	1,934	53,784
Vermont	9	902	5,385	385	342	49	7,073
Virginia	220,287	69,401	32,923	10,095	4,600	399	337,705
Washington	3,409	24,211	7,254	18,810	3,343	407	57,433
West Virginia	469,456	46,710	14,589	2,133	1,378	215	534,481
Wisconsin	180,200	66,807	6,369	7,163	3,647	70	264,256
Wyoming	89,874	22,321	6,721	2,674	721	1,106	123,417
Total	**10,380,883**	**2,030,759**	**1,216,362**	**446,831**	**168,480**	**49,094**	**14,292,410**

Table 5A-3. 2005 Base Year $PM_{2.5}$ Emissions (tons/year) for States by Sector

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Alabama	23,366	19,498	35,555	4,142	5,775	13,938	102,273
Arizona	7,418	3,940	21,402	4,486	6,920	37,151	81,316
Arkansas	1,688	10,820	34,744	3,803	3,102	10,315	64,472
California	347	21,517	94,200	22,815	26,501	97,302	262,682
Colorado	4,342	7,116	25,340	3,960	4,377	24,054	69,189
Connecticut	562	224	11,460	1,740	2,544	56	16,586
Delaware	2,169	1,810	1,590	818	922	87	7,397
District of Columbia	17	172	589	277	367	0	1,421

(continued)

Table 5A-3. 2005 Base Year $PM_{2.5}$ Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Florida	24,217	25,193	52,955	15,035	16,241	99,484	233,125
Georgia	28,057	12,666	63,133	6,504	12,449	24,082	146,892
Idaho	0	2,072	41,492	2,140	1,402	52,808	99,914
Illinois	16,585	15,155	74,045	12,880	12,574	277	131,516
Indiana	34,439	14,124	74,443	6,515	7,585	344	137,450
Iowa	8,898	6,439	54,312	6,969	3,468	349	80,436
Kansas	5,549	7,387	138,437	5,719	3,109	1,468	161,669
Kentucky	19,830	10,453	31,245	4,762	5,566	5,155	77,010
Louisiana	5,599	32,201	28,164	9,440	4,288	12,647	92,339
Maine	52	3,783	15,037	1,363	1,759	2,127	24,120
Maryland	15,417	6,768	23,323	3,410	5,504	531	54,952
Massachusetts	3,110	2,245	31,116	3,293	5,913	1,324	47,001
Michigan	11,022	12,926	47,722	8,561	13,006	1,283	94,520
Minnesota	3,262	10,538	73,990	8,541	6,842	8,943	112,116
Mississippi	2,029	10,602	34,217	4,133	4,195	14,897	70,074
Missouri	6,471	6,966	76,419	7,230	7,665	2,636	107,388
Montana	2,398	2,729	30,096	2,654	1,347	17,311	56,536
Nebraska	1,246	2,340	45,661	5,848	2,620	1,483	59,198
Nevada	3,341	4,095	9,920	2,212	1,290	19,018	39,876
New Hampshire	2,586	568	13,316	907	1,512	534	19,423
New Jersey	4,625	2,588	13,623	5,042	5,963	865	32,707
New Mexico	5,583	1,460	50,698	1,959	2,861	48,662	111,224
New York	9,648	4,994	48,540	8,607	11,139	1,601	84,529
North Carolina	16,967	12,665	49,551	6,272	8,939	9,870	104,264
North Dakota	6,397	598	41,504	4,552	976	934	54,962
Ohio	53,572	12,847	52,348	9,847	11,785	316	140,715
Oklahoma	1,411	6,246	90,047	3,765	4,559	6,644	112,672
Oregon	412	8,852	58,145	3,741	3,375	65,350	139,874
Pennsylvania	55,547	16,263	44,607	7,565	11,058	454	135,494

(continued)

Table 5A-3. 2005 Base Year $PM_{2.5}$ Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Rhode Island	10	256	1,289	394	577	14	2,540
South Carolina	14,455	4,779	26,598	3,491	5,061	9,163	63,548
South Dakota	390	2,982	33,678	2,910	1,056	7,062	48,079
Tennessee	12,856	21,912	32,563	5,072	8,514	3,934	84,851
Texas	21,464	37,563	194,036	21,361	29,859	21,578	325,861
Tribal	0	1,569	0	0	0	0	1,569
Utah	5,055	3,595	14,761	1,627	2,703	27,412	55,153
Vermont	37	337	6,943	479	605	696	9,098
Virginia	12,357	11,455	38,140	5,968	6,661	5,659	80,241
Washington	2,396	4,618	45,599	6,697	6,721	4,487	70,519
West Virginia	26,377	5,154	14,778	1,702	1,930	3,050	52,991
Wisconsin	5,233	7,967	37,277	6,083	6,783	994	64,337
Wyoming	8,068	10,298	31,645	1,455	1,103	15,686	68,254
Total	**496,877**	**433,346**	**2,110,298**	**268,745**	**301,073**	**684,035**	**4,294,373**

5A.4 Development of Future year baseline Emissions

The future year baseline scenario, also known as the “reference case”, represents predicted emissions including adjustments for known promulgated federal measures for all sectors as of the year 2017, which is assumed to be representative of 2016. The EGU and mobile sectors reflect projected economic and fuel usage changes. Emissions from non-EGU stationary sectors have previously been shown to not be well correlated with economic forecasts, and therefore economic impacts were not included for non-EGU stationary sources. Like the 2005 base case, these emissions cases include criteria pollutants and for some sectors, benzene, formaldehyde, acetaldehyde and methanol from the inventory is used in VOC speciation. The future year baseline scenario represents predicted emissions in the absence of any further controls beyond those Federal measures already promulgated. For EGUs, all state and other programs available at the time of modeling have been included. For mobile sources, all national measures promulgated at the time of modeling have been included. Additional details on the future year baseline (i.e., reference case) emissions modeling can be found in the

"Emissions Modeling for the Final Mercury and Air Toxics Standards Technical Support Document".

The future year baseline EGU emissions were obtained using version 4.10 Final of the Integrated Planning Model (IPM) (http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html). The IPM is a multiregional, dynamic, deterministic linear programming model of the U.S. electric power sector. Version 4.10 Final reflects state rules and consent decrees through December 1, 2010, information obtained from the 2010 Information Collection Request (ICR), and information from comments received on the IPM-related Notice of Data Availability (NODA) published on September 1, 2010. Notably, IPM 4.1 Final included the addition of over 20 GW of existing Activated Carbon Injection (ACI) for coal-fired EGUs reported to EPA via the ICR. Additional unit-level updates that identified existing pollution controls (such as scrubbers) were also made based on the ICR and on comments from the IPM NODA. Units with SO_2 or NO_X advanced controls (e.g., scrubber, SCR) that were not required to run for compliance with Title IV, New Source Review (NSR), state settlements, or state-specific rules were modeled by IPM to either operate those controls or not based on economic efficiency parameters. The IPM run for future year baseline case modeled with CMAQ assumed that 100% of the HCl found in the coal was emitted into the atmosphere. However, in the final IPM results for the rule, neutralization of 75% of the available HCl was included based on recent findings.

Further details on the future year baseline EGU emissions inventory used for this rule can be found in the IPM v.4.10 Documentation, available at http://www.epa.gov/airmarkets/progsregs/epa-ipm/transport.html. The future year baseline modeled in IPM for this rule includes estimates of emissions reductions that will result from the Cross-State Air Pollution Rule. However, reductions from the Boiler MACT rule were not represented this modeling because the rule was stayed at the time the modeling was performed. A complete list of state regulations, NSR settlements, and state settlements included in the IPM modeling is given in Appendices 3-2, 3-3, and 3-4 beginning on p. 68 of http://www.epa.gov/airmarkets/progsregs/epa-ipm/CSAPR/docs/DocSuppv410_FTransport.pdf. For the future year baseline EGU emissions, the IPM outputs for 2020, which are also representative of the year 2017, were used as part of the 2017 reference case modeling. These emissions were very similar to the year 2015 emissions output from the same IPM modeling case.

Inventories of onroad mobile emissions for the future year baseline and control cases were created using the MOVES model with an approach consistent with the 2005 base year. As with the 2005 emissions, the future year onroad emissions were based on emission factors

developed using the Tier 3 version of MOVES processed through the SMOKE-MOVES interface. Future-year vehicle miles travelled (VMT) were projected from the 2005 NEI v2 VMT using growth rates from the 2009 Annual Energy Outlook (AEO) data. The VMT for heavy duty diesel vehicles class 8a and 8b was updated based on data from Oak Ridge National Laboratory. The future year onroad emissions reflect control program implementation through 2017 and include the Light-Duty Vehicle Tier 2 Rule, the Onroad Heavy-Duty Rule, the Mobile Source Air Toxics (MSAT) final rule, and the Renewable Fuel Standard version 2 (RFS2).

Future year nonroad mobile emissions were created using NMIM to run NONROAD in a consistent manner as was done for 2005, but with future-year equipment population estimates, fuels, and control programs. The fuels in 2017 are assumed to be E10. Emissions for locomotives and category 1 and 2 (C1 and C2) commercial marine vessels were derived based on emissions published in the Final Locomotive Marine Rule, Regulatory Impact Assessment, Chapter 3 (see http://www.epa.gov/otaq/locomotives.htm#2008final). The future year baseline nonroad mobile emissions reductions include emissions reductions to locomotives, various nonroad engines including diesel engines and various marine engine types, fuel sulfur content, and evaporative emissions standards, including the category 3 marine residual and diesel fuelled engines and International Maritime Organization standards which include the establishment of emission control areas for these ships. A summary of the onroad and nonroad mobile source control programs included in the projected future year baseline is shown in Table 5A-4.

Table 5A-4. Summary of Mobile Source Control Programs Included in the Future Year Baseline

National Onroad Rules:
Tier 2 rule (Signature date: February 28, 2000)
Onroad heavy-duty rule (February 24, 2009)
Final mobile source air toxics rule (MSAT2) (February 9, 2007)
Renewable fuel standard Version 2 (March 26, 2010)
Light duty greenhouse gas standards (May, 2010)
Corporate Average Fuel Economy (CAFE) standards for 2008–2011
Local Onroad Programs:
National low emission vehicle program (NLEV) (March 2, 1998)
Ozone transport commission (OTC) LEV Program (January, 1995)

(continued)

Table 5A-4. Summary of Mobile Source Control Programs Included in the Future Year Baseline (continued)

National Nonroad Controls:
Tier 1 nonroad diesel rule (June 17, 2004)
Phase 1 nonroad SI rule (July 3, 1995)
Marine SI rule (October 4, 1996)
Nonroad large SI and recreational engine rule (November 8, 2002)
Clean Air Nonroad Diesel Rule—Tier 4 (June 29, 2004)
Locomotive and marine rule (May 6, 2008)
Nonroad SI rule (October 8, 2008)
Aircraft:
Itinerant (ITN) operations at airports adjusted to year 2017
Locomotives:
Locomotive Emissions Final Rulemaking (December 17, 1997)
Clean Air nonroad diesel final rule—Tier 4 (June 29, 2004)
Locomotive rule (April 16, 2008)
Locomotive and marine rule (May 6, 2008)
Commercial Marine:
Locomotive and marine rule (May 6, 2008)
EIA fuel consumption projections for diesel-fueled vessels
Clean Air Nonroad Diesel Final Rule – Tier 4
Emissions Standards for Commercial Marine Diesel Engines (December 29,1999)
Tier 1 Marine Diesel Engines (February 28, 2003)
Category 3 marine diesel engines Clean Air Act and International Maritime Organization standards (April, 30, 2010)

For non-EGU point sources, emissions were projected by including emissions reductions and increases from a variety of source data. Other than for certain large municipal waste combustors and airports, non-EGU point source emissions were not grown using economic growth projections, but rather were held constant at the emissions levels in 2005. Emissions reductions were applied to non-EGU point source to reflect final federal measures, known plant closures, and consent decrees. The starting inventories for this rule were the projected

emission inventories developed for the 2005v4.2 platform for the final Cross-State Air Pollution Rule (see http://www.epa.gov/ttn/chief/emch/index.html#final). The most significant updates to the emission projections for this rule are the addition of future year ethanol, biodiesel and cellulosic plants that account for increased ethanol production from the Renewable Fuel Standard Rule that is incorporated into the base case for 2017.

Since aircraft at airports were treated as point emissions sources in the 2005 NEI v2, we developed future year baseline emissions for airports by applying projection factors based on activity growth projected by the Federal Aviation Administration Terminal Area Forecast (TAF) system, published in January 2010 for these sources.

Emissions from stationary nonpoint sources were projected using procedures specific to individual source categories. Refueling emissions were projected using refueling emissions from MOVES inventory mode runs. Portable fuel container emissions were projected using estimates from previous rulemaking inventories compiled by the Office of Transportation and Air Quality (OTAQ). Emissions of ammonia and dust from animal operations were projected based on animal population data from the Department of Agriculture and EPA. Residential wood combustion emissions were projected by replacement of obsolete woodstoves with new woodstoves and a 1 percent annual increase in fireplaces. Landfill emissions were projected using MACT controls. Other nonpoint sources were held constant between the 2005 and future year scenarios.

A summary of all rules and growth assumptions impacting non-EGU stationary sources is provided in Table 5A-5, along with the affected pollutants. Note that reductions associated with the Boiler MACT are not included in the future year baseline.

Table 5A-5. Control Strategies and/or Growth Assumptions Included in the Future Year Baseline for Non-EGU Stationary Sources

MACT rules, national, VOC: national applied by SCC, MACT	VOC
Consent decrees and settlements, including refinery consent decrees, and settlements for: Alcoa, TX and Premcor (formerly MOTIVA), DE	All
Municipal waste combustor reductions—plant level	PM
Hazardous waste combustion	PM
Hospital/medical/infectious waste incinerator regulations	NO_x, PM, SO_2
Large municipal waste combustors—growth applied to specific plants	All

(continued)

Table 5A-5. Control Strategies and/or Growth Assumptions Included in the Future Year Baseline for Non-EGU Stationary Sources (continued)

MACT rules, plant-level, VOC: auto plants	VOC
MACT rules, plant-level, PM & SO_2: lime manufacturing	PM, SO_2
MACT rules, plant-level, PM: taconite ore	PM
Municipal waste landfills: projection factor of 0.25 applied	All
Livestock emissions growth from year 2002 to year 2017	NH_3, PM
Residential wood combustion growth and change-outs from years 2005 to year 2017	All
Gasoline Stage II growth and control via MOVES from year 2005 to year 2017	VOC
Portable fuel container mobile source air toxics rule 2: inventory growth and control from year 2005 to year 2017	VOC
NESHAP: Portland Cement (09/09/10)—plant level based on industrial sector integrated solutions (ISIS) policy emissions in 2013. The ISIS results are from the ISIS-cement model runs for the NESHAP and NSPS analysis of July 28, 2010 and include closures.	Hg, NO_X, SO_2, PM, HCl
New York ozone SIP standards	VOC, HAP VOC, NO_X
Additional plant and unit closures provided by state, regional, and EPA agencies	All
Emission reductions resulting from controls put on specific boiler units (not due to MACT) after 2005, identified through analysis of the control data gathered from the ICR from the ICI boiler NESHAP.	NO_X, SO_2, HCL
NESHAP: Reciprocating Internal Combustion Engines (RICE).	NO_X, CO, PM, SO_2
RICE controls applied to Phase II WRAP 2018 oil and gas emissions	VOC, SO_2, NO_X, CO
RICE controls applied to 2008 Oklahoma and Texas Oil and gas emissions	VOC, SO_2, NO_X, CO, PM
Ethanol plants that account for increased ethanol due to RFS2	All
State fuel sulfur content rules for fuel oil—effective in 2017, only in Maine, New Jersey, and New York	SO_2

In all future year cases, the same emissions were used for Canada and Mexico as were used in the 2005 base case because appropriate future year emissions for sources in these countries were not available. Future year emissions need to reflect expected percent reductions or increases between the base year and the future year to be considered appropriate for this type of modeling and such emissions were not available.

Table 5A-6 shows a summary of the 2005 and modeled future year baseline emissions for the lower 48 states. Tables 5A-7 and 5A-8 below provide summaries of SO_2 and $PM_{2.5}$ in the

2017 baseline for each sector by state. Table 5A-9 shows the future year baseline EGU emissions by state for all criteria air pollutants.

Table 5A-6. Summary of Modeled Base Case Annual Emissions (tons/year) for 48 States by Sector: SO_2 and $PM_{2.5}$

Source Sector SO_2 Emissions	2005	2017
EGU point	10,380,883	3,281,364
Non-EGU point	2,030,759	1,534,991
Nonpoint	1,216,362	1,125,985
Nonroad	446,831	15,759
On-road	168,480	29,288
Average fire	49,094	49,094
Total SO_2, all sources	**14,292,410**	**6,036,480**
Source Sector $PM_{2.5}$ Emissions	**2005**	**2017**
EGU point	496,877	276,430
Non-EGU point	433,346	411,437
Nonpoint	2,110,298	1,912,757
Nonroad	268,745	150,221
On-road	301,073	129,416
Average fire	684,035	684,035
Total $PM_{2.5}$, all sources	**4,294,373**	**3,564,296**

Table 5A-7. Future Year Baseline SO_2 Emissions (tons/year) for States by Sector

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Alabama	186,084	63,053	52,341	146	569	983	303,177
Arizona	36,996	24,191	2,467	59	724	2,888	67,324
Arkansas	92,804	12,160	26,801	123	314	728	132,929
California	5,346	21,046	67,846	3,311	2,087	6,735	106,370
Colorado	74,255	1,415	6,210	50	532	1,719	84,181
Connecticut	3,581	1,833	18,149	100	311	4	23,978

(continued)

Table 5A-7. Future Year Baseline SO_2 Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Delaware	2,835	4,770	1,018	500	91	6	9,220
District of Columbia	5	686	1,505	3	38	0	2,237
Florida	117,702	49,082	70,073	1,255	2,111	7,018	247,241
Georgia	96,712	44,248	55,946	192	1,158	2,010	200,266
Idaho	182	17,133	2,894	23	162	3,845	24,240
Illinois	118,217	81,683	5,650	295	1,107	20	206,971
Indiana	200,969	73,930	59,771	150	760	24	335,604
Iowa	85,178	22,865	19,929	86	324	25	128,407
Kansas	45,740	10,288	36,140	57	294	103	92,622
Kentucky	116,927	23,530	33,852	215	463	364	175,350
Louisiana	142,447	129,730	2,669	1,449	447	892	277,634
Maine	2,564	14,285	2,007	72	149	150	19,226
Maryland	29,786	33,562	40,642	494	593	32	105,110
Massachusetts	15,133	17,077	24,907	266	565	93	58,041
Michigan	163,168	48,697	42,185	448	995	91	255,584
Minnesota	52,380	24,742	14,635	220	558	631	93,164
Mississippi	34,865	24,284	6,635	208	396	1,051	67,440
Missouri	178,143	33,757	44,680	191	722	186	257,679
Montana	24,018	7,212	1,875	25	106	1,422	34,657
Nebraska	70,910	6,885	7,899	58	202	105	86,058
Nevada	14,140	2,132	12,028	27	200	1,346	29,873
New Hampshire	6,719	2,471	7,284	21	137	38	16,671
New Jersey	9,042	6,700	9,528	686	757	61	26,774
New Mexico	10,211	7,813	2,719	26	262	3,450	24,480
New York	14,653	45,222	71,060	659	1,466	113	133,173
North Carolina	71,113	58,517	21,713	197	890	696	153,125
North Dakota	105,344	9,915	5,559	36	71	66	120,991
Ohio	180,935	93,600	19,777	373	1,093	22	295,799

(continued)

Table 5A-7. Future Year Baseline SO_2 Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Oklahoma	141,433	27,873	7,731	49	501	469	178,056
Oregon	13,211	9,790	9,508	218	361	4,896	37,985
Pennsylvania	126,316	64,697	67,650	427	1,060	32	260,183
Rhode Island	0	2,745	3,338	33	85	1	6,202
South Carolina	103,694	28,536	13,310	294	500	646	146,980
South Dakota	29,711	1,655	10,301	23	86	498	42,273
Tennessee	33,080	59,145	32,624	154	757	277	126,037
Texas	249,748	129,667	108,633	1,146	2,483	1,178	492,855
Tribal	0	676	0	0	0	0	676
Utah	34,912	6,599	3,365	27	291	1,934	47,128
Vermont	264	902	5,283	8	129	49	6,634
Virginia	51,004	50,387	32,439	275	849	399	135,353
Washington	5,569	19,780	6,885	881	633	407	34,156
West Virginia	84,344	32,458	14,322	64	178	215	131,582
Wisconsin	50,777	61,080	6,260	122	633	70	118,941
Wyoming	48,198	20,491	5,944	18	87	1,106	75,844
Total	**3,281,364**	**1,534,991**	**1,125,985**	**15,759**	**29,288**	**49,094**	**6,036,480**

Table 5A-8. Future Year Baseline $PM_{2.5}$ Emissions (tons/year) for States by Sector

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Alabama	13,154	17,052	33,235	2,403	2,217	13,938	81,999
Arizona	3,889	3,809	20,214	2,674	2,762	37,151	70,498
Arkansas	2,838	10,527	33,486	2,042	1,242	10,315	60,450
California	475	20,693	73,607	14,875	13,492	97,302	220,443
Colorado	3,845	7,037	19,868	2,350	2,387	24,054	59,540
Connecticut	400	222	6,838	1,038	1,414	56	9,968
Delaware	434	772	1,207	383	375	87	3,259

(continued)

Table 5A-8. Future Year Baseline $PM_{2.5}$ Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
District of Columbia	1	172	536	139	154	0	1,002
Florida	12,723	24,620	50,472	8,100	7,652	99,484	203,050
Georgia	13,445	12,105	59,412	3,803	4,863	24,082	117,711
Idaho	36	2,076	40,288	1,186	714	52,808	97,108
Illinois	8,587	13,471	70,775	6,885	4,926	277	104,922
Indiana	22,354	13,570	72,501	3,491	3,380	344	115,640
Iowa	4,298	7,000	51,684	3,348	1,519	349	68,198
Kansas	3,199	6,895	136,633	2,872	1,268	1,468	152,335
Kentucky	12,078	10,353	26,811	2,717	2,059	5,155	59,173
Louisiana	3,093	30,865	27,082	5,107	1,673	12,647	80,467
Maine	355	3,543	8,213	881	750	2,127	15,869
Maryland	3,969	6,382	18,960	1,975	2,492	531	34,310
Massachusetts	1,465	2,123	23,729	1,914	2,590	1,324	33,145
Michigan	8,102	11,688	43,055	4,696	4,949	1,283	73,773
Minnesota	2,598	9,867	68,121	4,483	2,882	8,943	96,893
Mississippi	2,201	10,492	31,474	2,337	1,525	14,897	62,926
Missouri	7,061	6,384	69,722	3,954	3,059	2,636	92,816
Montana	3,870	2,562	28,479	1,332	492	17,311	54,048
Nebraska	2,358	2,834	44,904	2,967	919	1,483	55,465
Nevada	2,505	4,032	9,351	1,319	857	19,018	37,083
New Hampshire	1,130	464	8,981	576	663	534	12,348
New Jersey	2,452	2,520	8,559	2,929	3,244	865	20,569
New Mexico	3,153	1,442	49,789	1,148	1,103	48,662	105,298
New York	2,331	4,859	44,334	5,032	6,723	1,601	64,879
North Carolina	9,983	12,656	43,398	3,583	3,521	9,870	83,011
North Dakota	5,870	795	40,802	2,126	383	934	50,910
Ohio	18,920	12,353	47,811	5,302	5,013	316	89,715
Oklahoma	3,530	5,695	88,862	2,029	2,006	6,644	108,767

(continued)

Table 5A-8. Future Year Baseline $PM_{2.5}$ Emissions (tons/year) for States by Sector (continued)

State	EGU	Non-EGU	Nonpoint	Nonroad	Onroad	Fires	Total
Oregon	381	8,869	39,503	2,148	1,627	65,350	117,877
Pennsylvania	16,727	14,874	38,523	4,582	4,854	454	80,014
Rhode Island	4	256	1,070	222	383	14	1,949
South Carolina	9,997	4,527	23,430	1,932	1,929	9,163	50,978
South Dakota	737	2,399	32,697	1,339	416	7,062	44,650
Tennessee	5,053	21,553	28,449	2,939	3,057	3,934	64,985
Texas	21,677	34,648	187,604	11,901	9,289	21,578	286,698
Tribal	1	1,568	0	0	0	0	1,569
Utah	4,524	3,530	13,978	963	1,318	27,412	51,724
Vermont	67	336	4,930	307	653	696	6,989
Virginia	4,529	10,165	32,254	3,507	3,446	5,659	59,561
Washington	1,444	4,421	35,706	3,328	2,874	4,487	52,259
West Virginia	13,602	4,281	12,951	1,048	762	3,050	35,695
Wisconsin	5,323	7,853	27,656	3,161	3,148	994	48,135
Wyoming	5,662	10,225	30,812	850	392	15,686	63,626
Total	**276,430**	**411,437**	**1,912,757**	**150,221**	**129,416**	**684,035**	**3,564,296**

Table 5A-9. Future Year Baseline EGU CAP Emissions (tons/year) by State

State	CO	NO_X	VOC	SO_2	NH_3	PM_{10}	$PM_{2.5}$
Alabama	27,024	64,064	1,524	186,084	1,472	16,686	13,154
Arizona	16,797	36,971	825	36,996	1,163	5,038	3,889
Arkansas	9,925	36,297	658	92,804	560	3,507	2,838
California	45,388	20,910	1,031	5,346	2,519	580	475
Colorado	9,006	50,879	636	74,255	398	4,605	3,845
Connecticut	9,180	2,738	139	3,581	313	431	400
Delaware	4,256	2,452	132	2,835	119	580	434
District of Columbia	67	11	2	5	3	1	1

(continued)

Table 5A-9. Future Year Baseline EGU CAP Emissions (tons/year) by State (continued)

State	CO	NO_X	VOC	SO_2	NH_3	PM_{10}	$PM_{2.5}$
Florida	72,915	83,174	2,253	117,702	3,997	19,098	12,723
Georgia	16,537	43,778	1,293	96,712	903	18,668	13,445
Idaho	1,532	613	41	182	57	38	36
Illinois	51,862	56,128	3,091	118,217	1,437	9,926	8,587
Indiana	30,587	106,881	2,295	200,969	1,317	33,816	22,354
Iowa	8,316	42,698	791	85,178	452	5,735	4,298
Kansas	5,066	25,163	683	45,740	305	3,996	3,199
Kentucky	37,287	71,259	1,604	116,927	928	16,279	12,078
Louisiana	32,626	33,509	852	142,447	1,427	3,677	3,093
Maine	12,789	6,121	306	2,564	269	366	355
Maryland	13,446	17,933	533	29,786	301	5,322	3,969
Massachusetts	7,128	7,991	279	15,133	395	1,915	1,465
Michigan	25,856	66,846	1,497	163,168	874	11,056	8,102
Minnesota	9,365	36,867	746	52,380	460	3,034	2,598
Mississippi	9,704	27,319	440	34,865	469	3,113	2,201
Missouri	16,499	52,464	1,714	178,143	740	9,093	7,061
Montana	5,266	20,946	338	24,018	198	6,117	3,870
Nebraska	4,691	28,898	542	70,910	292	2,948	2,358
Nevada	9,677	15,627	438	14,140	953	3,095	2,505
New Hampshire	5,667	4,908	206	6,719	207	1,234	1,130
New Jersey	25,831	11,178	823	9,042	747	2,948	2,452
New Mexico	9,079	65,189	574	10,211	570	3,833	3,153
New York	19,731	21,172	731	14,653	1,076	3,248	2,331
North Carolina	17,367	44,141	1,076	71,113	654	13,368	9,983
North Dakota	7,437	53,778	867	105,344	383	6,757	5,870
Ohio	33,481	93,150	2,005	180,935	1,317	25,688	18,920
Oklahoma	26,165	47,454	957	141,433	1,073	4,457	3,530
Oregon	5,905	10,828	203	13,211	381	446	381

(continued)

Table 5A-9. Future Year Baseline EGU CAP Emissions (tons/year) by State (continued)

State	CO	NO_X	VOC	SO_2	NH_3	PM_{10}	$PM_{2.5}$
Pennsylvania	38,767	123,501	2,023	126,316	1,522	22,117	16,727
Rhode Island	1,748	456	44	0	136	7	4
South Carolina	10,305	37,516	726	103,694	515	14,469	9,997
South Dakota	742	14,293	129	29,711	48	764	737
Tennessee	10,693	16,982	862	33,080	406	6,313	5,053
Texas	78,317	145,182	4,975	249,748	5,304	31,404	21,677
Tribal	601	73	15	0	47	2	1
Utah	5,632	67,476	526	34,912	279	5,843	4,524
Vermont	1,868	458	52	264	25	69	67
Virginia	30,205	39,408	821	51,004	1,115	5,404	4,529
Washington	7,183	14,284	326	5,569	346	1,706	1,444
West Virginia	15,496	54,247	1,320	84,344	658	18,415	13,602
Wisconsin	19,247	35,179	1,137	50,777	649	6,503	5,323
Wyoming	9,087	71,380	970	48,198	481	7,385	5,662
Total	**873,344**	**1,930,769**	**46,050**	**3,281,364**	**40,259**	**371,101**	**276,430**

Note: Emission estimates apply to all fossil Electric Generating Units, including those with capacity < 25MW.

5A.5 Development of Future Year Control Case Emissions for Air Quality Modeling

For the future year control case (i.e., policy case) air quality modeling**,** the emissions for all sectors were unchanged from the base case modeling except for those from EGUs. The IPM model was used to prepare the future year policy case for EGU emissions. The air quality modeling for MATS relied on EGU emission projections from an interim IPM platform based on the Cross-state Air Pollution Rule version 4.10_FTransport, and was subsequently updated during the rulemaking process. The updates made include: updated assumptions regarding the removal of HCl by alkaline fly ash in subbituminous and lignite coals; an update to the fuel-based mercury emission factor for petroleum coke, which was corrected based on re-examination of the 1999 ICR data; updated capital cost for new nuclear capacity and nuclear life extension costs; corrected variable operating and maintenance cost (VOM) for ACI retrofits; adjusted coal rank availability for some units, consistent with EIA From 923 (2008); updated state rules in Washington and Colorado; and numerous unit-level revisions based on comments received through the notice and comment process. In particular, the policy case modeled with

CMAQ did not include the neutralization of 75% of HCl as did the final policy case. Additional details on the version of IPM used to develop the control case are available in Chapter 3.

The changes in EGU SO_2, and $PM_{2.5}$ emissions as a result of the policy case for the lower 48 states are summarized in Table 5A-10. Table 5A-11 shows the CAP emissions for the modeled MATS control case by State. State-specific difference summaries of EGU SO_2 and $PM_{2.5}$ for the sum of the lower 48 states are shown in Tables 5A-12 and 5A-13, respectively.

Table 5A-10. Summary of Emissions Changes for the MATS AQ Modeling in the Lower 48 States

Future Year EGU Emissions	SO_2	$PM_{2.5}$
Base case EGU emissions (tons)	3,281,364	276,430
Control case EGU emissions (tons)	1,866,247	223,320
Reductions to base case in control case (tons)	1,415,117	53,110
Percentage reduction of base EGU emissions	43%	19%
Total Man-Made Emissions[a]		
Total base case emissions (tons)	6,036,480	3,564,296
Total control case emissions (tons)	4,621,363	3,511,186
Percentage reduction of all man-made emissions	23%	1%

[a] In this table, man-made emissions includes average fires.

Table 5A-11. EGU Emissions Totals for the Modeled MATS Control Case in the Lower 48 States

State	CO	NO_X	VOC	SO_2	NH_3	PM_{10}	$PM_{2.5}$
Alabama	20,873	61,863	1,313	68,517	1,235	9,734	7,844
Arizona	13,238	34,804	749	23,459	921	4,264	3,494
Arkansas	9,036	35,788	642	35,112	490	1,696	1,593
California	56,360	27,159	1,307	5,041	2,548	1,057	942
Colorado	8,219	44,409	582	19,564	358	3,492	2,859
Connecticut	8,017	2,800	136	1,400	313	439	412
Delaware	1,312	2,527	67	4,160	93	3,056	1,455
District of Columbia							
Florida	66,378	61,676	2,055	64,791	3,482	16,434	11,377

(continued)

Table 5A-11. EGU Emissions Totals for the Modeled MATS Control Case in the Lower 48 States (continued)

State	CO	NO_X	VOC	SO_2	NH_3	PM_{10}	$PM_{2.5}$
Georgia	14,217	41,006	1,197	78,197	790	11,165	9,742
Idaho	1,523	609	41	182	56	38	36
Illinois	24,365	50,655	2,353	103,867	1,050	7,309	6,588
Indiana	17,061	102,045	1,872	156,781	1,110	29,683	20,388
Iowa	7,340	41,247	747	48,030	410	3,318	2,947
Kansas	4,683	22,136	623	22,767	282	2,504	2,263
Kentucky	25,911	70,126	1,476	125,430	882	12,544	10,635
Louisiana	28,171	31,655	767	30,509	1,261	2,003	1,899
Maine	10,992	5,683	302	1,372	267	342	331
Maryland	4,283	16,554	400	18,091	211	3,851	3,143
Massachusetts	5,408	7,211	226	5,033	344	1,702	1,267
Michigan	18,792	60,982	1,215	82,834	718	8,261	6,893
Minnesota	8,699	34,942	709	33,214	430	3,332	2,936
Mississippi	8,782	20,749	410	15,975	397	1,949	1,720
Missouri	12,249	52,755	1,605	95,965	686	5,216	4,809
Montana	2,223	19,758	264	6,399	133	2,637	1,727
Nebraska	4,493	28,180	533	34,631	277	2,152	1,828
Nevada	7,178	14,382	336	6,372	725	2,626	2,073
New Hampshire	6,781	4,862	232	2,102	232	1,336	1,264
New Jersey	8,350	7,699	315	6,404	546	2,020	1,583
New Mexico	7,987	64,922	545	9,984	554	2,961	2,750
New York	18,725	20,863	699	28,174	1,086	3,123	2,350
North Carolina	15,195	35,309	1,033	59,551	602	8,885	7,988
North Dakota	7,266	53,267	858	23,889	371	5,940	5,051
Ohio	29,956	85,565	1,852	139,208	1,229	19,599	15,823
Oklahoma	26,687	44,725	892	44,602	970	2,293	2,056
Oregon	6,002	9,671	198	3,565	379	241	233
Pennsylvania	24,865	104,906	1,645	93,606	1,349	17,330	14,080

(continued)

Table 5A-11. EGU Emissions Totals for the Modeled MATS Control Case in the Lower 48 States (continued)

State	CO	NO_X	VOC	SO_2	NH_3	PM_{10}	$PM_{2.5}$
Rhode Island	1,721	443	43	0	134	7	4
South Carolina	9,826	37,849	725	40,901	459	9,627	6,963
South Dakota	641	14,290	117	2,483	41	260	245
Tennessee	5,551	16,931	723	42,666	334	6,721	5,272
Texas	71,475	138,086	4,444	105,958	4,774	25,359	17,601
Tribal	266	32	7	0	21	1	1
Utah	4,003	65,286	474	17,007	241	4,755	3,896
Vermont	1,868	458	52	264	25	69	67
Virginia	26,778	37,255	707	33,704	748	5,306	4,506
Washington	6,334	3,834	179	854	254	183	176
West Virginia	13,923	47,836	1,263	66,857	632	14,321	11,572
Wisconsin	16,124	32,865	1,012	28,322	578	4,725	3,969
Wyoming	7,516	71,135	932	28,456	467	5,946	4,671
Total	**707,640**	**1,789,790**	**40,875**	**1,866,247**	**35,493**	**281,811**	**223,320**

Table 5A-12. State Specific Changes in Annual EGU SO_2 for the Lower 48 States

State	Future Year Baseline SO_2 (tons)	Future Year Policy Case SO_2 (tons)	EGU SO_2 Reduction (tons)	EGU SO_2 Reduction (%)
Alabama	186,084	68,517	117,568	63%
Arizona	36,996	23,459	13,537	37%
Arkansas	92,804	35,112	57,692	62%
California	5,346	5,041	305	6%
Colorado	74,255	19,564	54,690	74%
Connecticut	3,581	1,400	2,181	61%
Delaware	2,835	4,160	-1,324	-47%
District of Columbia	5	0	5	100%

(continued)

Table 5A-12. State Specific Changes in Annual EGU SO_2 for the Lower 48 States (continued)

State	Future Year Baseline SO_2 (tons)	Future Year Policy Case SO_2 (tons)	EGU SO_2 Reduction (tons)	EGU SO_2 Reduction (%)
Florida	117,702	64,791	52,911	45%
Georgia	96,712	78,197	18,515	19%
Idaho	182	182	0	0%
Illinois	118,217	103,867	14,350	12%
Indiana	200,969	156,781	44,189	22%
Iowa	85,178	48,030	37,148	44%
Kansas	45,740	22,767	22,973	50%
Kentucky	116,927	125,430	-8,503	-7%
Louisiana	142,447	30,509	111,938	79%
Maine	2,564	1,372	1,191	46%
Maryland	29,786	18,091	11,695	39%
Massachusetts	15,133	5,033	10,100	67%
Michigan	163,168	82,834	80,334	49%
Minnesota	52,380	33,214	19,165	37%
Mississippi	34,865	15,975	18,890	54%
Missouri	178,143	95,965	82,177	46%
Montana	24,018	6,399	17,618	73%
Nebraska	70,910	34,631	36,279	51%
Nevada	14,140	6,372	7,768	55%
New Hampshire	6,719	2,102	4,618	69%
New Jersey	9,042	6,404	2,638	29%
New Mexico	10,211	9,984	228	2%
New York	14,653	28,174	-13,521	-92%
North Carolina	71,113	59,551	11,562	16%
North Dakota	105,344	23,889	81,455	77%
Ohio	180,935	139,208	41,727	23%
Oklahoma	141,433	44,602	96,831	68%

(continued)

Table 5A-12. State Specific Changes in Annual EGU SO_2 for the Lower 48 States (continued)

State	Future Year Baseline SO_2 (tons)	Future Year Policy Case SO_2 (tons)	EGU SO_2 Reduction (tons)	EGU SO_2 Reduction (%)
Oregon	13,211	3,565	9,646	73%
Pennsylvania	126,316	93,606	32,710	26%
Rhode Island	0	0	0	N/A
South Carolina	103,694	40,901	62,793	61%
South Dakota	29,711	2,483	27,228	92%
Tennessee	33,080	42,666	-9,586	-29%
Texas	249,748	105,958	143,790	58%
Tribal	0	0	0	N/A
Utah	34,912	17,007	17,905	51%
Vermont	264	264	0	0%
Virginia	51,004	33,704	17,300	34%
Washington	5,569	854	4,716	85%
West Virginia	84,344	66,857	17,488	21%
Wisconsin	50,777	28,322	22,454	44%
Wyoming	48,198	28,456	19,742	41%
Total	**3,281,364**	**1,866,247**	**1,415,117**	

Table 5A-13. State Specific Changes in Annual EGU $PM_{2.5}$ for the Lower 48 States

State	Future Year Baseline $PM_{2.5}$ (tons)	Future Year Policy Case $PM_{2.5}$ (tons)	EGU $PM_{2.5}$ Reduction (tons)	EGU $PM_{2.5}$ Reduction (%)
Alabama	13,154	7,844	5,310	40%
Arizona	3,889	3,494	395	10%
Arkansas	2,838	1,593	1,246	44%
California	475	942	-467	-98%
Colorado	3,845	2,859	985	26%
Connecticut	400	412	-12	-3%

(continued)

Table 5A-13. State Specific Changes in Annual EGU $PM_{2.5}$ for the Lower 48 States (continued)

State	Future Year Baseline $PM_{2.5}$ (tons)	Future Year Policy Case $PM_{2.5}$ (tons)	EGU $PM_{2.5}$ Reduction (tons)	EGU $PM_{2.5}$ Reduction (%)
Delaware	434	1,455	-1,021	-235%
District of Columbia	1	0	1	100%
Florida	12,723	11,377	1,346	11%
Georgia	13,445	9,742	3,703	28%
Idaho	36	36	0	0%
Illinois	8,587	6,588	2,000	23%
Indiana	22,354	20,388	1,966	9%
Iowa	4,298	2,947	1,351	31%
Kansas	3,199	2,263	936	29%
Kentucky	12,078	10,635	1,443	12%
Louisiana	3,093	1,899	1,193	39%
Maine	355	331	24	7%
Maryland	3,969	3,143	826	21%
Massachusetts	1,465	1,267	198	14%
Michigan	8,102	6,893	1,210	15%
Minnesota	2,598	2,936	-339	-13%
Mississippi	2,201	1,720	481	22%
Missouri	7,061	4,809	2,252	32%
Montana	3,870	1,727	2,143	55%
Nebraska	2,358	1,828	530	22%
Nevada	2,505	2,073	432	17%
New Hampshire	1,130	1,264	-134	-12%
New Jersey	2,452	1,583	868	35%
New Mexico	3,153	2,750	403	13%
New York	2,331	2,350	-19	-1%
North Carolina	9,983	7,988	1,995	20%
North Dakota	5,870	5,051	819	14%

(continued)

Table 5A-13. State Specific Changes in Annual EGU $PM_{2.5}$ for the Lower 48 States (continued)

State	Future Year Baseline $PM_{2.5}$ (tons)	Future Year Policy Case $PM_{2.5}$ (tons)	EGU $PM_{2.5}$ Reduction (tons)	EGU $PM_{2.5}$ Reduction (%)
Ohio	18,920	15,823	3,097	16%
Oklahoma	3,530	2,056	1,474	42%
Oregon	381	233	148	39%
Pennsylvania	16,727	14,080	2,646	16%
Rhode Island	4	4	0	2%
South Carolina	9,997	6,963	3,033	30%
South Dakota	737	245	492	67%
Tennessee	5,053	5,272	-219	-4%
Texas	21,677	17,601	4,077	19%
Tribal	1	1	1	56%
Utah	4,524	3,896	627	14%
Vermont	67	67	0	0%
Virginia	4,529	4,506	24	1%
Washington	1,444	176	1,268	88%
West Virginia	13,602	11,572	2,031	15%
Wisconsin	5,323	3,969	1,354	25%
Wyoming	5,662	4,671	991	17%
Total	**276,430**	**223,320**	**53,110**	

APPENDIX 5B
IMPACT OF THE INTERIM POLICY SCENARIO ON AIR QUALITY

5B.1 Air Quality Modeling Platform

This document describes the air quality modeling performed by EPA in support of the final National Emissions Standard for Hazardous Air Pollutants (NESHAP) related to electrical generating utilities. A national scale air quality modeling analysis was performed to estimate the impact of the sector emissions changes on future year annual and 24-hour $PM_{2.5}$ concentrations, 8-hr maximum ozone, as well as visibility impairment. Air quality benefits are estimated with the Community Multi-scale Air Quality (CMAQ) model. CMAQ simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone, particulate matter and other air pollutants. In addition to the CMAQ model, the modeling platform includes the emissions, meteorology, and initial and boundary condition data which are inputs to this model.

Emissions and air quality modeling decisions are made early in the analytical process. For this reason, it is important to note that the inventories used in the air quality modeling may be slightly different than the final utility sector inventories presented in the RIA. However, the air quality inventories and the final rule inventories are generally consistent, so the air quality modeling adequately reflects the effects of the rule. Similarly, the projected future year inventory used for this analysis is generally representative of several years around 2017 such as 2016.

Photochemical grid models use state of the science numerical algorithms to estimate pollutant formation, transport, and deposition over a variety of spatial scales that range from urban to continental. Emissions of precursor species are injected into the model where they react to form secondary species such as ozone and then transport around the modeling domain before ultimately being removed by deposition or chemical reaction. The 2005-based CMAQ modeling platform was used as the basis for the air quality modeling for this rule. This platform represents a structured system of connected modeling-related tools and data that provide a consistent and transparent basis for assessing the air quality response to projected changes in emissions. The base year of data used to construct this platform includes emissions and meteorology for 2005. This modeling platform is described in more detail in the modeling technical support document for this rule (USEPA, 2011).

5.B.1.1 Photochemical Model Background

The Community Multi-scale Air Quality (CMAQ) model v4.7.1 (www.cmaq-model.org) is a state of the science three-dimensional Eularian "one-atmosphere" photochemical transport model used to estimate air quality (Appel et al., 2008; Appel et al., 2007; Byun and Schere, 2006). CMAQ simulates the formation and fate of photochemical oxidants, ozone, primary and secondary PM concentrations, and air toxics over regional and urban spatial scales for given input sets of meteorological conditions and emissions. CMAQ is applied with the AERO5 aerosol module, which includes the ISORROPIA inorganic chemistry (Nenes et al., 1998) and a secondary organic aerosol module (Carlton et al., 2010). The CMAQ model is applied with sulfur and organic oxidation aqueous phase chemistry (Carlton et al., 2008) and the carbon-bond 2005 (CB05) gas-phase chemistry module (Gery et al., 1989).

5.B.1.2 Model Setup, Application, and Post-Processing

The modeling analyses were performed for a domain covering the continental United States, as shown in Figure 5B-1. This domain has a parent horizontal grid of 36 km with two finer-scale 12 km grids over portions of the eastern and western U.S. The model extends vertically from the surface to 100 millibars (approximately 15 km) using a sigma-pressure coordinate system. Air quality conditions at the outer boundary of the 36 km domain were taken from a global model and vary in time and space. The 36 km grid was only used to establish the incoming air quality concentrations along the boundaries of the 12 km grids. Only the finer grid data were used in determining the impacts of the emissions changes. Table 5B-1 provides geographic information about the photochemical model domains.

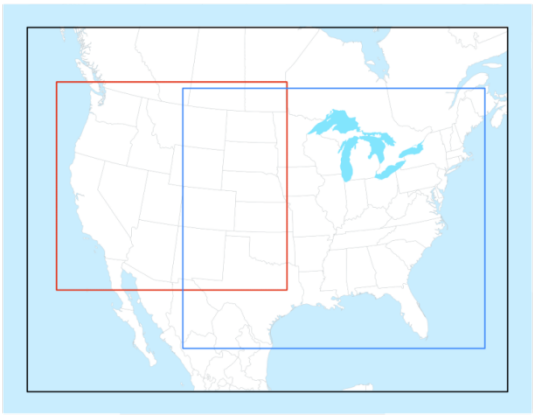

Figure 5B-1. Map of the Photochemical Modeling Domains. The black outer box denotes the 36 km national modeling domain; the red inner box is the 12 km western U.S. grid; and the blue inner box is the 12 km eastern U.S. grid.

Table 5B-1. Geographic Elements of Domains Used in Photochemical Modeling

	Photochemical Modeling Configuration		
	National Grid	**Western U.S. Fine Grid**	**Eastern U.S. Fine Grid**
Map Projection	Lambert Conformal Projection		
Grid Resolution	36 km	12 km	12 km
Coordinate Center	97 deg W, 40 deg N		
True Latitudes	33 deg N and 45 deg N		
Dimensions	148 x 112 x 14	213 x 192 x 14	279 x 240 x 14
Vertical extent	14 Layers: Surface to 100 millibar level		

The 36 km and both 12 km modeling domains were modeled for the entire year of 2005. Data from the entire year were utilized when looking at the estimation of $PM_{2.5}$ and visibility impacts from the regulation. Data from April through October is used to estimate ozone impacts.

As part of the analysis for this rulemaking, the modeling system was used to calculate daily and annual $PM_{2.5}$ concentrations, 8-hr maximum ozone, and visibility impairment. Model predictions are used to estimate future-year design values of $PM_{2.5}$ and ozone. Specifically, we compare a 2017 reference scenario to a 2017 control scenario. This is done by calculating the simulated air quality ratios between any particular future year simulation and the 2005 base. These predicted ratios are then applied to ambient base year design values. The design value projection methodology used here followed EPA guidance for such analyses (USEPA, 2007).

5.B.1.3 Emissions Input Data

The emissions data used in the base year and future reference and future emissions adjustment case are based on the 2005 v4.1 platform. Emissions are processed to photochemical model inputs with the SMOKE emissions modeling system (Houyoux et al., 2000). The 2017 reference case is intended to represent the emissions associated with growth and controls in that year projected from the 2005 simulation year. The United States EGU point source emissions estimates for the future year reference and control case are based on an Integrated Planning Model (IPM) run for criteria pollutants. Both control and growth factors were applied to a subset of the 2005 non-EGU point and non-point emissions to create the 2017 reference case. The 2005 v4 platform projection factors were the starting point for most

of the 2017 SMOKE-based projections. The estimated total anthropogenic emissions and emissions for the utility sector used in this modeling assessment are shown in Appendix 5A. Other North American emissions are based on a 2006 Canadian inventory and 1999 Mexican inventory. Both inventories are not grown or controlled when used as part of future year inventories. Global emissions of criteria pollutants are included in the modeling system through boundary condition inflow. More details on these emissions are available in Appendix 5A.

5B.2 Impacts of Sector on Future Annual $PM_{2.5}$ Levels

This section summarizes the results of our modeling of annual average $PM_{2.5}$ air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2017 baseline scenario to a 2017 control scenario. The modeling assessment indicates a decrease up to 1.03 $\mu g/m^3$ in annual $PM_{2.5}$ design values is possible given an area's proximity to controlled sources. The median reduction in annual $PM_{2.5}$ design value over all monitor locations is 0.36 $\mu g/m^3$. The change in future year projected design value is shown in Figure 5B-2. Negative changes indicate an improvement in air quality.

An annual $PM_{2.5}$ design value is the concentration that determines whether a monitoring site meets the annual NAAQS for $PM_{2.5}$. The full details involved in calculating an annual $PM_{2.5}$ design value are given in appendix N of 40 CFR part 50. Projected air quality benefits are estimated using procedures outlined by United States Environmental Protection Agency modeling guidance (USEPA, 2007).

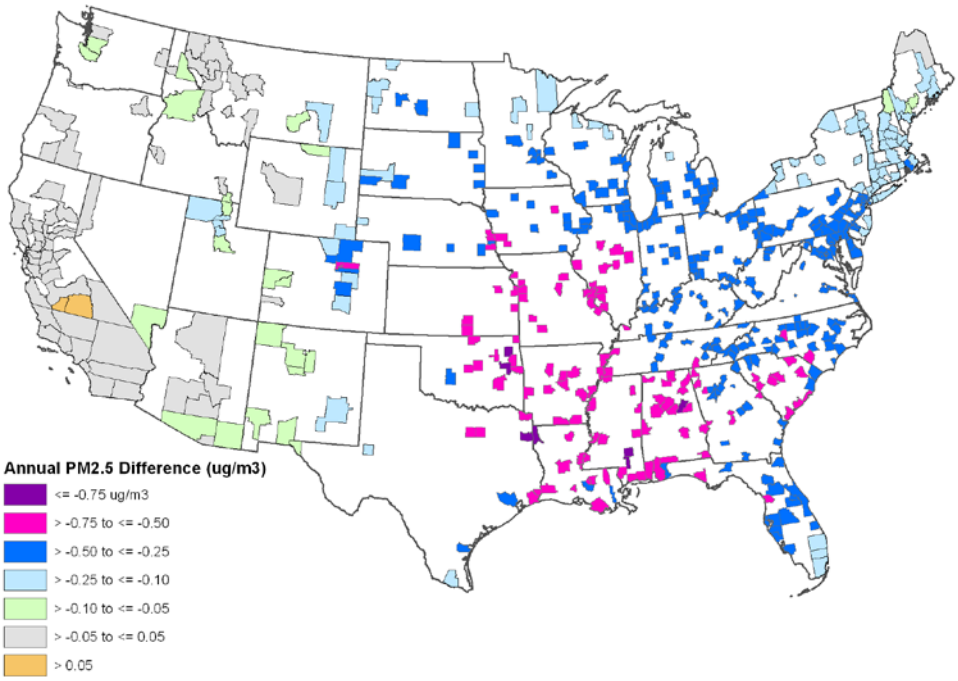

Figure 5B-2. Change in Design Values Between the 2017 Baseline and 2017 Control Simulations. Negative numbers indicate lower (improved) design values in the control case compared to the baseline

5B.3 Impacts of Sector on Future 24-hour $PM_{2.5}$ Levels

This section summarizes the results of our modeling of 24-hr average $PM_{2.5}$ air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2017 baseline scenario to a 2017 control scenario. A decrease up to 1.9 µg/m^3 in 24-hr average $PM_{2.5}$ design value at monitor locations in the United States is possible given an area's proximity to controlled sources and the amount of reduced emissions from those sources. A median decrease of 0.6 µg/m^3 in 24-hr average $PM_{2.5}$ design value at monitor locations in the United States is possible given an area's proximity to controlled sources and the amount of reduced emissions from those sources. The change in future year projected design value is shown in Figure 5B-3. Negative changes indicate an improvement in air quality.

A 24-hour $PM_{2.5}$ design value is the concentration that determines whether a monitoring site meets the 24-hour NAAQS for $PM_{2.5}$. The full details involved in calculating a 24-hour $PM_{2.5}$ design value are given in appendix N of 40 CFR part 50. Projected air quality benefits are estimated using procedures outlined by United States Environmental Protection Agency modeling guidance (USEPA, 2007).

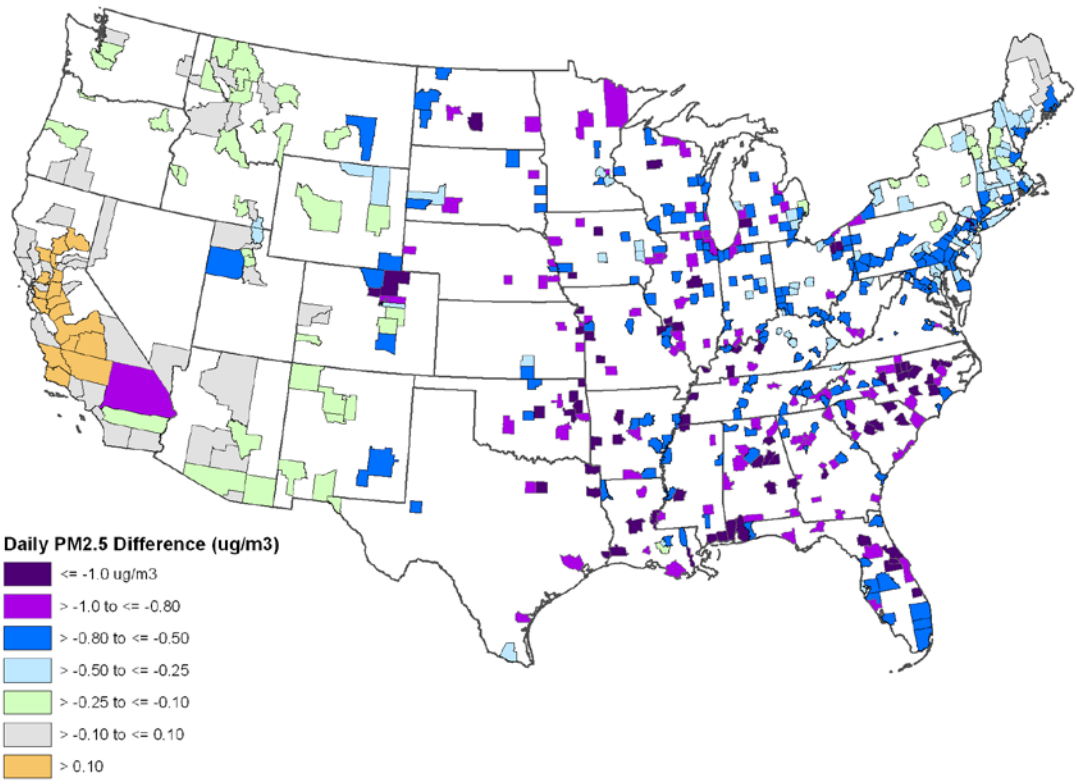

Figure 5B-3. Change in Design Values Between the 2017 Base Case and 2017 Control Simulations. Negative numbers indicate lower (improved) design values in the control case compared to the baseline.

5B.4 Impacts of Sector on Future Visibility Levels

Air quality modeling conducted for this rule was used to project visibility conditions in 138 mandatory Class I federal areas across the U.S. in 2017 (USEPA, 2007). The level of visibility impairment in an area is based on the light-extinction coefficient and a unitless visibility index, called a "deciview," which is used in the valuation of visibility. The deciview metric provides a scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. Higher deciview values are indicative of worse visibility. Thus, an improvement in visibility is a decrease in deciview value.

The modeling assessment indicates a median visibility improvement of 0.09 deciviews in annual 20% worst visibility days over all Class I area monitors. An improvement in visibility up to 0.97 deciviews on the 20% worst visibility days at Class I monitor locations in the United States is possible given an area's proximity to controlled sources and the amount of reduced emissions

from these sources. The change in future year projected visibility is shown in Figure 5B-4. Negative changes indicate an improvement in air quality.

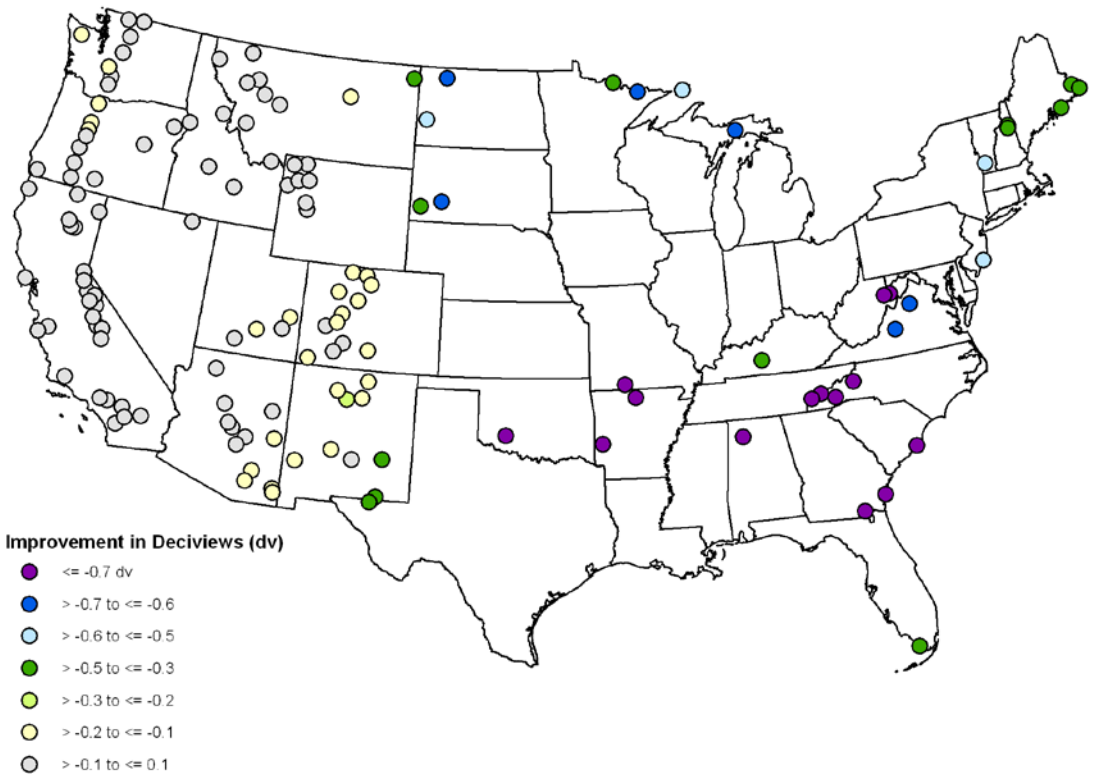

Figure 5B-4. Change in 20% Worst Days Between the 2017 Baseline and 2017 Control Simulations. Negative numbers indicate lower (improved) visibility expressed in deciviews in the control case compared to the baseline

5B.5 Impacts of Sector on Future Ozone Levels

This section summarizes the results of our modeling of 8-hr maximum ozone air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2017 baseline scenario to a 2017 control scenario. The modeling assessment indicates a decrease of up to 3.5 ppb in 8-hr averaged ozone design value is possible given an area's proximity to controlled sources and the amount of reduced emissions from these sources. A median decrease across all monitors of 0.20 ppb in 8-hr averaged ozone design value is possible given an area's proximity to controlled sources and the amount of reduced emissions from these sources. The change in future year projected design value is shown in Figure 5B-5. Negative changes indicate an improvement in air quality. The full details involved in calculating

design value are given in appendix P of 40 CFR part 50. Projected air quality benefits are estimated using procedures outlined by United States Environmental Protection Agency modeling guidance (USEPA, 2007).

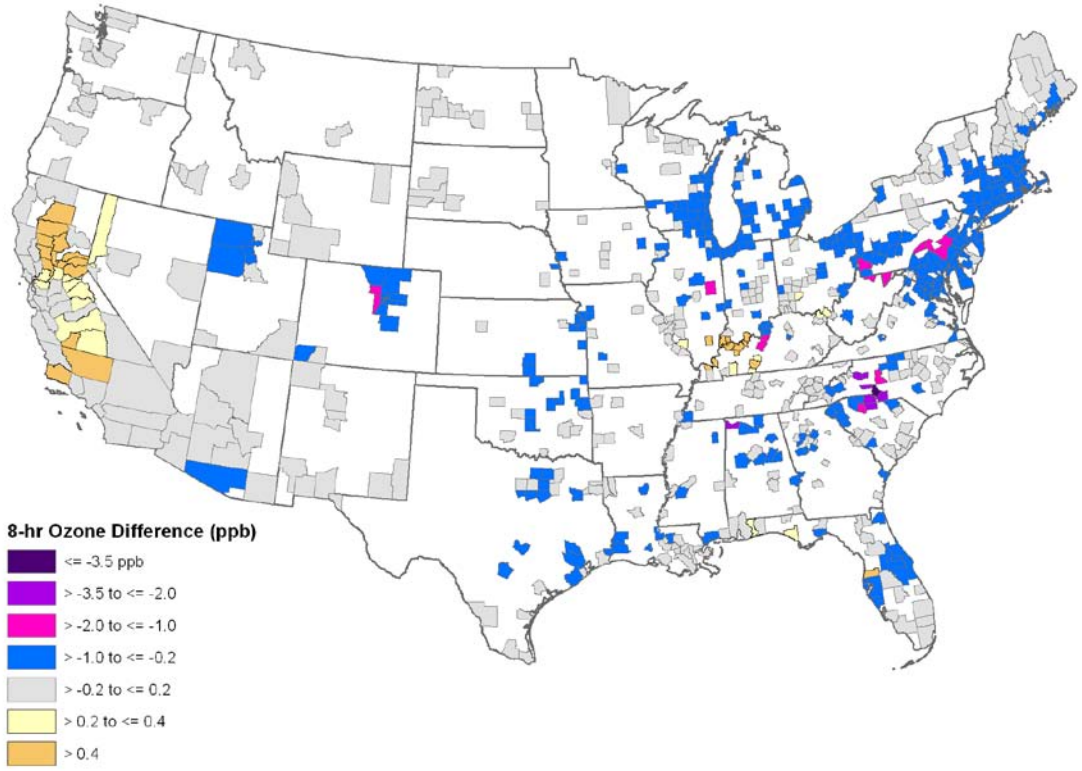

Figure 5B-5. Change in Design Values Between the 2017 Baseline and 2017 Control Simulations. Negative numbers indicate lower (improved) design values in the control case compared to the baseline

5B.6 References

Appel, K.W., Bhave, P.V., Gilliland, A.B., Sarwar, G., Roselle, S.J., 2008. Evaluation of the community multiscale air quality (CMAQ) model version 4.5: Sensitivities impacting model performance; Part II—particulate matter. Atmospheric Environment 42, 6057-6066.

Appel, K.W., Gilliland, A.B., Sarwar, G., Gilliam, R.C., 2007. Evaluation of the Community Multiscale Air Quality (CMAQ) model version 4.5: Sensitivities impacting model performance Part I—Ozone. Atmospheric Environment 41, 9603-9615.

Byun, D., Schere, K.L., 2006. Review of the governing equations, computational algorithms, and other components of the models-3 Community Multiscale Air Quality (CMAQ) modeling system. Applied Mechanics Reviews 59, 51-77.

Carlton, A.G., Bhave, P.V., Napelenok, S.L., Edney, E.D., Sarwar, G., Pinder, R.W., Pouliot, G.A., Houyoux, M., 2010. Model Representation of Secondary Organic Aerosol in CMAQv4.7. Environmental Science & Technology 44, 8553-8560.

Carlton, A.G., Turpin, B.J., Altieri, K.E., Seitzinger, S.P., Mathur, R., Roselle, S.J., Weber, R.J., 2008. CMAQ Model Performance Enhanced When In-Cloud Secondary Organic Aerosol is Included: Comparisons of Organic Carbon Predictions with Measurements. Environmental Science & Technology 42, 8798-8802.

Gery, M.W., Whitten, G.Z., Killus, J.P., Dodge, M.C., 1989. A Photochemical Kinetics Mechanism for Urban and Regional Scale Computer Modeling. Journal of Geophysical Research-Atmospheres 94, 12925-12956.

Houyoux, M.R., Vukovich, J.M., Coats, C.J., Wheeler, N.J.M., Kasibhatla, P.S., 2000. Emission inventory development and processing for the Seasonal Model for Regional Air Quality (SMRAQ) project. Journal of Geophysical Research-Atmospheres 105, 9079-9090.

Nenes, A., Pandis, S.N., Pilinis, C., 1998. ISORROPIA: A new thermodynamic equilibrium model for multiphase multicomponent inorganic aerosols. Aquatic Geochemistry 4, 123-152.

USEPA, 2007. Guidance on the Use of Models and Other Analyses for Demonstrating Attainment of Air Quality Goals for Ozone, $PM_{2.5}$, and Regional Haze, RTP.

USEPA, 2011. Air Quality Modeling Technical Support Document: Final EGU NESHAP (EPA-454/R-11-009), Research Triangle Park, North Carolina.

APPENDIX 5C
HEALTH AND WELFARE CO-BENEFITS OF THE MODELED INTERIM POLICY SCENARIO

In this appendix to the co-benefits chapter we report the estimates of the benefits of reductions in emissions of SO_2 and directly emitted $PM_{2.5}$ based on air quality modeling of an interim policy scenario.

As noted in Chapter 5 of the RIA, the air quality modeling performed for the RIA does not reflect the emission changes associated with the final rule requirements. To estimate the benefits of those emissions changes for the final rule, we developed BPT estimates for SO_2 and directly emitted $PM_{2.5}$ based on air quality modeling of an interim policy scenario. These BPT values were used to adjust benefits estimates for changes in the emission reductions resulting from the final policy scenario. This appendix reports the results of the benefits analysis associated with the modeled interim policy scenario described in Appendix 5A and 5B, along with the derivation of BPT values used to estimate the health benefits of the final policy scenario.

As described in the benefits chapter, the chief difference between the modeled and revised scenarios relates to the magnitude and distribution of SO_2 emission reductions (Figure 5C-1). In general, the modeled and revised policy cases achieve roughly similar levels of SO_2 reductions (1.42 versus 1.33 million tons, respectively) with a similar distribution among states. However, for some states (notably Alabama, Colorado, Louisiana, Michigan, Missouri, North Dakota, Oklahoma, and Texas), SO_2 emission reductions were lower for the final case versus the interim case. By far, the greatest difference in SO_2 emission reductions was in Michigan where the final case emission reduction was 70% lower than for the interim case. In a few states (notably Arkansas, Ohio, and South Carolina), SO_2 emission reductions were slightly larger for the final case versus the interim case. Since differences between the interim and final cases are not concentrated in any particular region of the country and the overall distribution of emission reductions is similar, we conclude that it is reasonable to apply BPT values derived from the interim case to the final case. While NO_x emissions reductions decreased by 70% between the interim and final cases (141,000 vs. 46,000 tons), the impact of NO_x on $PM_{2.5}$ concentrations and mortality is very minor relative to the impact of SO_2 emission reductions. Therefore, differences in the magnitude and distribution of NO_x emission reductions are likely to have only a minor effect on results.

5C.1 $PM_{2.5}$-Related Health Impacts and Monetized Benefits of Reductions in Emissions of SO_2 and Directly Emitted $PM_{2.5}$ for the Air Quality Modeled Interim Policy Scenario

Health benefits of the interim policy scenario are calculated using the modeled changes in $PM_{2.5}$ concentration described in Appendix 5B, which result from the emission changes described in Appendix 5A. Concentration changes are input into BenMAP to calculate the changes in incidence of an array of health endpoints, along with their associated monetary value. BenMAP is described in more detail in Chapter 5. In addition, more information can be found at http://www.epa.gov/air/benmap/.

Tables 5C-1 and 5C-2 summarize the $PM_{2.5}$-related health impacts and monetized benefits of the air quality modeled interim policy scenario. Not all known PM- and ozone-related health and welfare effects could be quantified or monetized. The monetized value of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized health benefits is thus equal to the subset of monetized PM- and ozone-related health and welfare benefits plus B, the sum of the nonmonetized health and welfare benefits; this B represents both uncertainty and a bias in this analysis, as it reflects those benefits categories that we are unable quantify in this analysis. Figure 5C-2 illustrates the distribution of avoided PM-related deaths by county across the U.S.

Methods for quantifying recreational visibility are described in Section 5.5.1. Visibility benefits are calculated for the modeled interim policy scenario only since there is no analogous approach for estimating visibility benefits using the BPT approach. However, the magnitude of SO_2 emission reductions did not significantly change in the visibility study areas between the interim and final emissions scenarios. Therefore, we expect the visibility benefit for the final policy scenario would be similar to that calculated for the interim policy scenario ($1.1 billion in total for the U.S., using 2007$). These benefits are not included in the co-benefits estimate of the final policy.

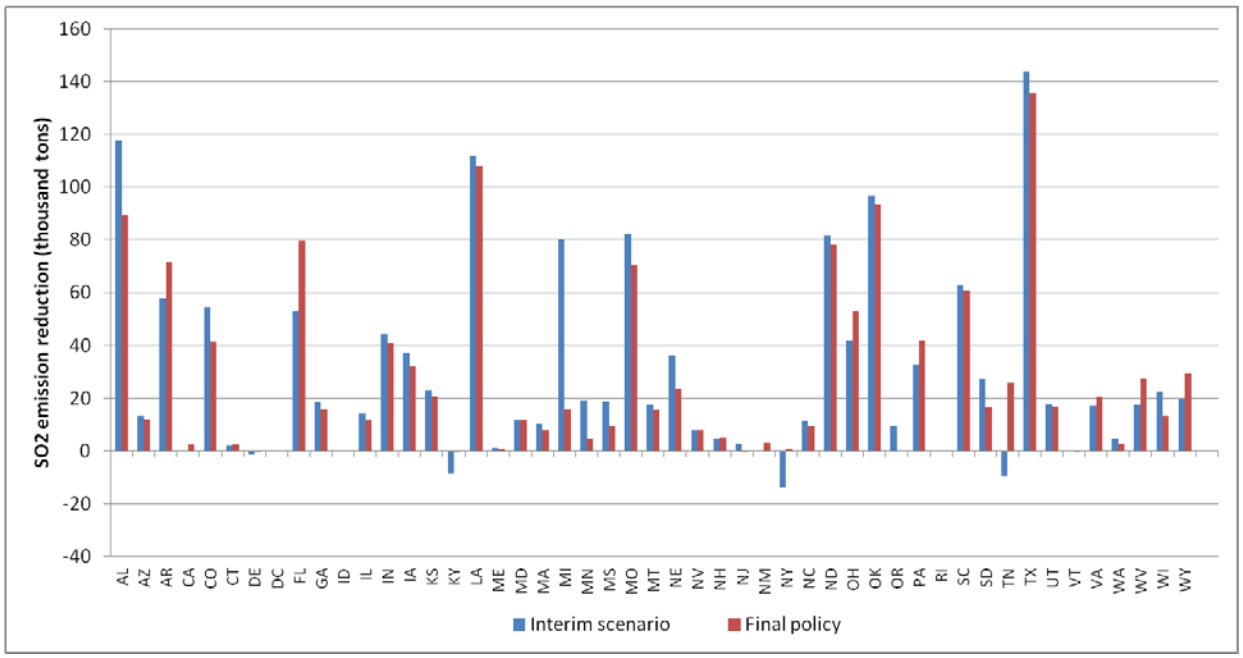

Figure 5C-1. Comparison of state-level SO_2 emission changes between the interim modeled scenario and the final policy.

Table 5C-1. Estimated Reduction in Incidence of Adverse Health Effects of the Interim Modeled Mercury and Air Toxics Standards in 2016 (95% confidence intervals)[a,b]

Impact	Eastern U.S.[c]	Western U.S.	Total
Premature Mortality			
Pope et al. (2002) (age >30)	4,200 (1,200—7,300)	120 (19—220)	4,400 (1,200—7,500)
Laden et al. (2006) (age >25)	11,000 (5,000—17,000)	310 (110—510)	11,000 (5,100—17,000)
Infant (< 1 year)	20 (-22—61)	1 (-1—2)	20 (-23—63)
Chronic Bronchitis	2,800 (94—5,500)	100 (-12—210)	2,900 (82—5,800)
Non-fatal heart attacks (age > 18)	4,800 (1,200—8,400)	110 (13—220)	4,900 (1,200—8,600)
Hospital admissions—respiratory (all ages)	840 (340—1,300)	16 (5—27)	860 (340—1,400)
Hospital admissions—cardiovascular (age > 18)	1,800 (1,200—2,200)	41 (26—51)	1,900 (1,300—2,200)
Emergency room visits for asthma (age < 18)	3,100 (1,600—4,700)	100 (44—160)	3,200 (1,700—4,800)
Acute bronchitis (age 8-12)	6,200 (-1,400—14,000)	240 (-100—570)	6,500 (-1,500—14,000)
Lower respiratory symptoms (age 7-14)	80,000 (31,000—130,000)	3,000 (860—5,200)	83,000 (32,000—130,000)
Upper respiratory symptoms (asthmatics age 9-18)	60,000 (11,000—110,000)	2,300 (130—4,400)	62,000 (11,000—110,000)
Asthma exacerbation (asthmatics 6-18)	130,000 (4,700—450,000)	5,000 (-520—17,000)	140,000 (4,200—460,000)
Lost work days (ages 18-65)	540,000 (460,000—620,000)	20,000 (16,000—24,000)	560,000 (470,000—640,000)
Minor restricted-activity days (ages 18-65)	3,200,000 (2,600,000—3,800,000)	120,000 (93,000—140,000)	3,300,000 (2,700,000—3,900,000)

[a] Estimates rounded to two significant figures; column values will not sum to total value.

[b] The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts.

[c] Includes Texas and those states to the north and east.

Table 5C-2. Estimated Economic Value of Health and Welfare Benefits of the Interim Modeled Mercury and Air Toxics Standards in 2016 (95% confidence intervals, billions of 2007$)

Impact	Pollutant	Eastern U.S.[a]	Western U.S.	Total
Premature mortality (Pope et al. 2002 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$34 ($2.7—$100)	$1.0 ($0.1—$3.1)	$35 ($2.8—$100)
7% discount rate	$PM_{2.5}$	$31 ($2.4—$93)	$0.9 ($0.1—$2.8)	$32 ($2.5—$96)
Premature mortality (Laden et al. 2006 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$87 ($7.7—$250)	$2.5 ($0.2—$7.5)	$90 ($7.9—$260)
7% discount rate	$PM_{2.5}$	$79 ($6.9—$230)	$2.3 ($0.2—$6.7)	$81 ($7.1—$240)
Infant mortality	$PM_{2.5}$	$0.2 ($-0.2—$0.8)	<$0.01	$0.2 ($-0.2—$0.8)
Chronic bronchitis	$PM_{2.5}$	$1.4 ($0.1—$6.4)	$0.05 (<$0.01—$0.23)	$1.4 ($0.1—$6.6)
Non-fatal heart attacks				
3% discount rate	$PM_{2.5}$	$0.5 ($0.1—$1.3)	$0.01 (<$0.01—$0.03)	$0.6 ($0.1—$1.3)
7% discount rate	$PM_{2.5}$	$0.4 ($0.1—$1.0)	$0.01 (<$0.01—$0.03)	$0.4 ($0.1—$1.0)
Hospital admissions—respiratory	$PM_{2.5}$	$0.01 ($0.01—$0.02)	<$0.01	$0.01 ($0.01—$0.02)
Hospital admissions—cardiovascular	$PM_{2.5}$	$0.03 (<$0.01—$0.05)	<$0.01	$0.03 (<$0.01—$0.06)
Emergency room visits for asthma	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Acute bronchitis	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lower respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Upper respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Asthma exacerbation	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lost work days	$PM_{2.5}$	$0.1 ($0.1—$0.1)	<$0.01	$0.1 ($0.1—$0.1)
Minor restricted-activity days	$PM_{2.5}$	$0.2 ($0.1—$0.3)	<$0.01	$0.2 ($0.1—$0.3)
Recreational visibility, Class I areas	$PM_{2.5}$	$0.9	$0.2	$1.1
Social cost of carbon (3% discount rate, 2016 value)	CO_2			

(continued)

Table 5C-2. Estimated Economic Value of Health and Welfare Benefits of the Interim Modeled Mercury and Air Toxics Standards in 2016 (95% confidence intervals, billions of 2007$) (continued)

Impact	Pollutant	Eastern U.S.	Western U.S.	Total
Total Monetized benefits (Pope et al. 2002 $PM_{2.5}$ mortality estimates)				
3% discount rate		$37+B ($3.7—$110)	$1.2+B ($0.2—$3.6)	$39+B ($4.0—$120)
7% discount rate		$34+B ($3.5—$100)	$1.1+B ($0.2—$3.3)	$35+B ($3.7—$110)
Total Monetized benefits (Laden et al. 2006 $PM_{2.5}$ mortality estimates)				
3% discount rate		$91+B ($8.7—$260)	$2.8+B ($0.4—$8.0)	$93+B ($9.1—$270)
7% discount rate		$82+B ($7.9—$240)	$2.5+B ($0.4—$7.2)	$84+B ($8.3—$240)

[a] Includes Texas and those states to the north and east.

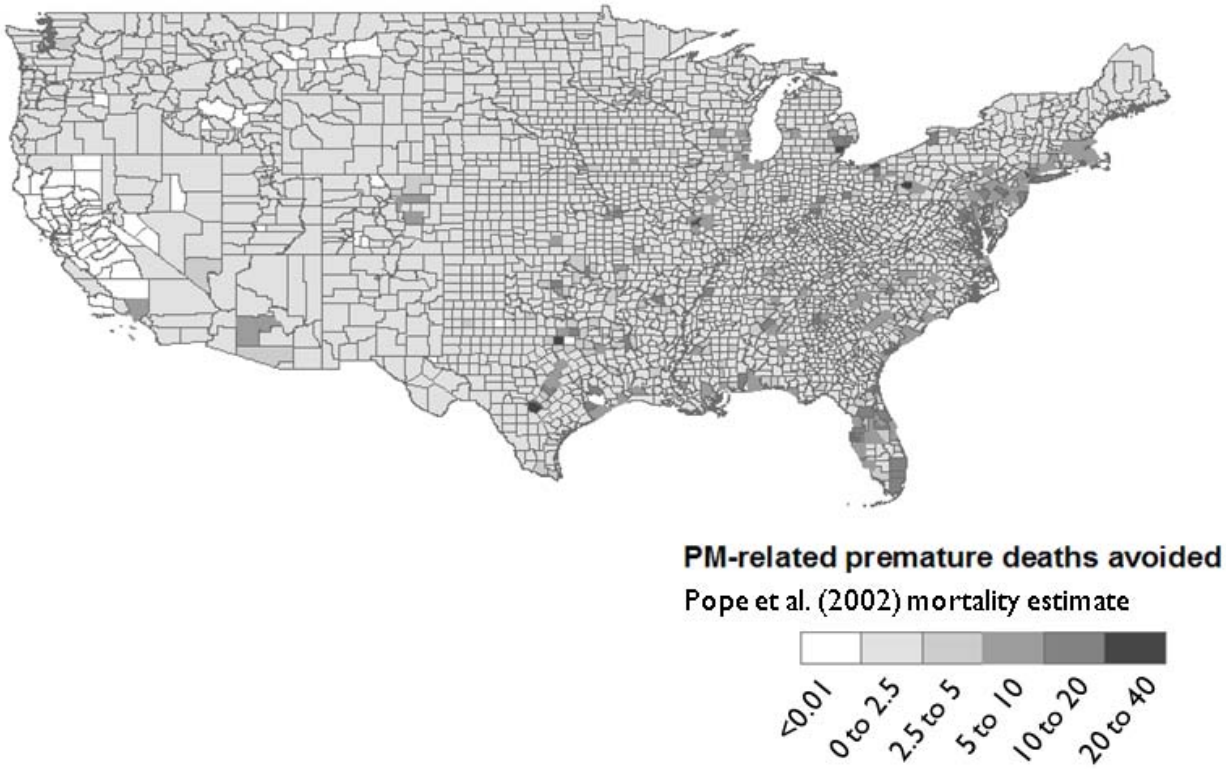

Figure 5C-2. Estimated Reduction in Excess $PM_{2.5}$-Related Premature Deaths Estimated to Occur in Each County in 2016 as a Result of the Interim Modeled Mercury and Air Toxics Standards

5C.2 Derivation of the BPT Values Used to Calculate the Health Benefits of the Final Policy Scenario

The health benefits summarized in Tables C-1 and C-2 include impacts of changes in sulfate, nitrate, and direct $PM_{2.5}$. To quantify the health benefits of the final policy scenario reported in Chapter 5, we calculate the health benefits per ton of emission reduced, separately for eastern and western states, and separately for SO_2, directly emitted carbonaceous $PM_{2.5}$, and directly emitted crustal $PM_{2.5}$. This calculation is shown by Equation 1:

$$BPT_{i,j,k} = \frac{TotalBenefits_{i,j,k,1}}{\Delta Emissions_{i,j,1}}$$

where BPT is the BPT for a particular pollutant i (SO_2, directly emitted carbonaceous $PM_{2.5}$, or directly emitted crustal $PM_{2.5}$), region j (Eastern U.S. or Western U.S.), health endpoint k (e.g. adult mortality, infant mortality, etc.), and the interim baseline and policy scenario (denoted as "1"). As described in Chapter 5, we do not generate BPT values for NO_x. Nitrate increases in the modeled policy scenario were two orders of magnitude smaller than the sulfate decreases and were not included in the BPT estimates. Including nitrate in the SO_2 BPT estimate would reduce the SO_2 BPT by 1-2%, with a corresponding impact on the total health benefits of the rule. Furthermore, as described in Appendix 5A, NO_x emission changes resulting from this rule were 75% smaller for the final policy scenario relative to the interim modeled policy scenario. Therefore, excluding the impacts of NO_x emission changes is unlikely to materially impact the final benefit results.

Table 5C-3 reports the economic value of the adult mortality benefits resulting from reductions in SO_2, directly emitted carbonaceous $PM_{2.5}$, and directly emitted crustal $PM_{2.5}$ for the modeled interim policy scenario, along with the BPT values derived from these benefits. Only adult mortality benefits are shown here as they contribute 93-97% of the total health benefits, however, BPT values were calculated and applied separately for each health endpoint. Since premature mortality is discounted after BPT values are applied to the final emissions, the values reported in Table 5C-3 are not discounted. Sulfate reductions resulting from SO_2 emission reductions contribute approximately 95% of the benefits of SO_2 and directly emitted $PM_{2.5}$ combined. In some locations, directly emitted carbonaceous $PM_{2.5}$ increased slightly in the Western U.S. for the interim policy scenario relative to the interim baseline, which overall resulted in negative BPT values for the West. However, since the magnitudes of the emission and concentration changes are small relative to the changes in SO_2 emissions and sulfate concentrations, the resulting increase in premature mortality is only 0.04% of the total health impact of the rule.

Table 5C-3. Estimated Economic Value of Adult Mortality Benefits by Pollutant, in Total and Per Ton of Emissions Reduced Interim Modeled Mercury and Air Toxics Standard in 2016 (95% confidence intervals, 2007$)

Pollutant and Source of Adult Mortality Estimate	Total Monetized Benefits (billions)		BPT (thousands)	
	Eastern U.S.[a]	Western U.S.	Eastern U.S.[a]	Western U.S.
SO_2 emissions (tons)[b]	1,268,961	146,155		
Pope et al. (2002) estimate	$36 ($2.9—$110)	$1.2 ($0.1—$3.7)	$29 ($2.3-$87)	$8.3 ($0.1-$25)
Laden et al. (2006) estimate	$93 ($8.2—$270)	$3.1 ($0.3—$9.0)	$73 ($6.4-$210)	$21 ($1.9-$62)
Carbonaceous $PM_{2.5}$ emissions (tons)[b]	5,860	231		
Pope et al. (2002) estimate[c]	$1.3 ($0.1—$3.9)	<-$0.01	$220 ($17-$670)	-$66 (-$450-$210)
Laden et al. (2006) estimate[c]	$3.3 ($0.3—$9.6)	<-$0.01	$560 ($49-$1,600)	-$170 (-$960-$350)
Crustal $PM_{2.5}$ emissions (tons)[b]	34,742	29,148		
Pope et al. (2002) estimate	$0.6 (<$0.01—$1.9)	$0.1 (<$0.01—$0.2)	$18 ($1.4-$55)	$9.6 ($0.1-$31)
Laden et al. (2006) estimate	$1.6 ($0.1—$4.7)	$0.1 (<$0.01—$0.4)	$47 ($4.1-$140)	$25 ($2.1-$74)

[a] Includes Texas and those states to the north and east.

[b] Emission reductions are reported for the modeled interim policy case, from which the BPT values were generated.

[C] Directly emitted carbonaceous $PM_{2.5}$ increased slightly in some locations in the Western U.S. for the interim policy scenario relative to the interim baseline, which overall resulted in negative BPT values for the West. However, since the magnitudes of the emission and concentration changes are small relative to the changes in SO_2 emissions and sulfate concentrations, the resulting increase in premature mortality is only 0.04% of the total health impact of the rule.

The BPT values reported in Table 5C-3, along with those calculated for the other health endpoints listed in Table 5C-2, are applied to the final emission changes described in Chapter 3, resulting in the final benefit values summarized in Chapter 5. This calculation is shown by Equation 2:

$$TotalBenefits_{i,j,k,} = \Delta Emissions_{i,j,2} \; x \; BPT_{i,j,k}$$

where 2 refers to the final baseline and policy scenarios.

APPENDIX 5D
$PM_{2.5}$ CO-BENEFITS OF THE FINAL RULE BY STATE

5D.1 Introduction

This appendix describes the distribution of the health-related $PM_{2.5}$ co-benefits associated with this rule by state. We describe our approach for allocating the national-level $PM_{2.5}$-related mortality and monetized benefits to the state-level. We also summarize the results of this analysis and describe the limitations and uncertainties associated with our approach. This rule is expected to achieve $PM_{2.5}$-related health benefits in all states, resulting from both emission reductions in that state and reduced transport of $PM_{2.5}$ between states. A key limitation of our approach is that it does not account for differences in the distribution of SO_2 and direct $PM_{2.5}$ emission reductions between the modeled interim scenario and the final policy (see Appendix 5C). $PM_{2.5}$-related co-benefits may therefore be under- or over-estimated for certain states.

5D.2 Methods

As described in Appendix 5C, the $PM_{2.5}$ health co-benefits of the final rule are calculated using a BPT approach. The BPT values are derived from air quality modeling of an interim emissions scenario. Since the distribution of the SO_2 emission reductions in the interim modeled scenario and the final policy were generally consistent, applying BPT values from the interim modeled scenario to the final policy reasonably approximates the total monetized benefits of the final policy. However, this approach requires aggregation of benefits in the interim scenario to larger spatial scales to account for transport of pollution across state boundaries. Therefore, the final rule benefits described in Appendix 5C are estimated for the eastern and western US, the same resolution at which BPT values were generated from the interim scenario.

Since spatially resolved estimates of the co-benefits are useful for understanding how the expected benefits of this rule are distributed across the U.S., we developed an approximating approach for allocating national-level $PM_{2.5}$ co-benefits estimated for the final policy to the state level. This approach follows three steps. First, we quantified the state-level mortality and monetized health co-benefits of the air quality modeled scenario using the BenMAP software. From these results, we calculated the percentage of national health benefits occurring in each state. Finally, these percentages were used to scale the national health benefits of the final policy down to the state level.

As another approach, EPA considered scaling the health co-benefits of the final policy scenario by the percentage of the national total SO_2 emission change occurring in each state, since the distribution of emission changes across the U.S. changed between the modeled interim scenario and the final policy. However, such an approach would not account for the population in each state which is a main driver for air pollution health impacts, nor would it account for transport of pollution across state lines. Therefore, EPA judged that scaling national health co-benefits of the final rule by the state distribution of the co-benefits of the interim modeled scenario is a more appropriate approach.

5D.3 Limitations and uncertainties

The method described above adds unique uncertainties and limitations beyond those already described in detail in Chapter 5. A key limitation of this approach is that the distribution of SO_2 and direct $PM_{2.5}$ emissions changed between the modeled interim scenario and the final policy (see Appendix 5C). Differences in the emission changes would have an effect on the percentage of health co-benefits occurring in each state for the final policy. However, our approach necessarily assumes that the state distribution of health co-benefits for the final policy is equivalent to that of the modeled interim scenario. $PM_{2.5}$-related health co-benefits for this rule could therefore be under- or over-estimated for certain states.

5D.4 Results

The reduction in incidence of adult premature $PM_{2.5}$-related mortality for the final rule by state is shown in Table 5D-1. Additional non-mortality heath benefits are also expected in each state but are not included here. The greatest percentage of interim mortality benefits in any one state is in Texas (10.8%), followed by Florida (6.7%). For the final policy, 460 to 1,200 avoided premature deaths are estimated to be avoided in Texas, and 280 to 750 in Florida, depending on the concentration-response factor. Although SO_2 emissions in some states (e.g. Kentucky, New York, Tennessee) increase between the interim baseline and interim policy scenario (see Appendix 5A), mortality decreases in these states due to reduced transport of pollution from other states. All states, therefore, experience health benefits from the interim scenario and the final rule. Table 5D-2 shows the estimated economic value of health and welfare benefits by state for the final rule. Approximately $4.0 to $9.7 billion (2007$, 3% discount rate) in benefits are expected to occur in Texas, and $2.4 to $6.0 billion in Florida, depending on the concentration-response function used for adult mortality.

Table 5D-1. Estimated Reduction in Incidence of Premature Adult Mortality for the Mercury and Air Toxics Standards in 2016 by State[a,b]

State	Percent of total interim benefits	Final policy benefits – adult mortality	
		Pope et al. (2002) estimate	Laden et al. (2006) estimate
Alabama	3.34	140	360
Arizona	0.32	14	35
Arkansas	2.28	96	250
California	0.13	6	14
Colorado	1.26	53	140
Connecticut	0.84	35	90
Delaware	0.30	13	32
DC	0.14	6	15
Florida	6.73	280	730
Georgia	4.53	190	490
Idaho	0.06	3	6
Illinois	5.31	220	570
Indiana	2.64	110	290
Iowa	1.45	61	160
Kansas	1.43	60	160
Kentucky	1.97	83	210
Louisiana	2.68	110	290
Maine	0.19	8	20
Maryland	1.99	84	220
Massachusetts	1.23	52	130
Michigan	3.78	160	410
Minnesota	1.34	57	150
Mississippi	2.21	93	240
Missouri	3.79	160	410
Montana	0.07	3	8
Nebraska	0.67	28	72
Nevada	0.09	4	10
New Hampshire	0.23	10	25
New Jersey	2.96	130	320
New Mexico	0.22	9	24
New York	4.11	170	440
North Carolina	4.42	190	480
North Dakota	0.17	7	19
Ohio	5.19	220	560
Oklahoma	2.82	120	300
Oregon	0.11	5	12
Pennsylvania	4.91	210	530
Rhode Island	0.27	11	29
South Carolina	3.01	130	330
South Dakota	0.25	11	27
Tennessee	3.38	140	370
Texas	10.82	460	1200
Utah	0.20	8	22
Vermont	0.09	4	10
Virginia	2.76	120	300
Washington	0.28	12	31

West Virginia	0.89	38	96
Wisconsin	2.06	87	220
Wyoming	0.05	2	6
National Total		4,200	11,000

[a] State level benefits of the final rule are scaled by the distribution of mortality benefits simulated from the interim scenario described in Appendices 5A, 5B, and 5C.

[b] Estimates rounded to two significant figures; column values will not sum to total value. These estimates do not include confidence intervals.

Table 5D-2. Estimated Economic Value of Health Benefits of the Mercury and Air Toxics Standard in 2016 by State (billions of 2007$, 3% discount rate)[a,b]

State	Percent of total interim benefits	Health benefits (billions of 2007$, 3% discount rate)	
		Pope et al. (2002) estimate	Laden et al. (2006) estimate
Alabama	3.32	$1.20	$3.00
Arizona	0.32	$0.12	$0.29
Arkansas	2.27	$0.82	$2.00
California	0.13	$0.05	$0.12
Colorado	1.28	$0.46	$1.10
Connecticut	0.84	$0.30	$0.75
Delaware	0.30	$0.11	$0.27
DC	0.14	$0.05	$0.12
Florida	6.68	$2.40	$6.00
Georgia	4.56	$1.70	$4.10
Idaho	0.06	$0.02	$0.05
Illinois	5.34	$1.90	$4.70
Indiana	2.64	$0.96	$2.40
Iowa	1.45	$0.52	$1.30
Kansas	1.43	$0.52	$1.30
Kentucky	1.97	$0.71	$1.80
Louisiana	2.67	$0.97	$2.40
Maine	0.19	$0.07	$0.17
Maryland	1.99	$0.72	$1.80
Massachusetts	1.23	$0.45	$1.10
Michigan	3.78	$1.40	$3.40
Minnesota	1.35	$0.49	$1.20
Mississippi	2.20	$0.80	$2.00
Missouri	3.78	$1.40	$3.40
Montana	0.07	$0.03	$0.06
Nebraska	0.67	$0.24	$0.60
Nevada	0.09	$0.03	$0.08
New Hampshire	0.23	$0.08	$0.21
New Jersey	2.96	$1.10	$2.60
New Mexico	0.22	$0.08	$0.20
New York	4.12	$1.50	$3.70
North Carolina	4.41	$1.60	$3.90
North Dakota	0.17	$0.06	$0.15
Ohio	5.17	$1.90	$4.60
Oklahoma	2.81	$1.00	$2.50
Oregon	0.11	$0.04	$0.10
Pennsylvania	4.87	$1.80	$4.40
Rhode Island	0.27	$0.10	$0.24
South Carolina	2.99	$1.10	$2.70
South Dakota	0.25	$0.09	$0.23
Tennessee	3.38	$1.20	$3.00
Texas	10.95	$4.00	$9.70
Utah	0.20	$0.07	$0.18
Vermont	0.09	$0.03	$0.08
Virginia	2.77	$1.00	$2.50
Washington	0.28	$0.10	$0.25

West Virginia	0.89	$0.32	$0.79
Wisconsin	2.06	$0.75	$1.80
Wyoming	0.05	$0.02	$0.05
National Total[c]		$36.00	$89.00

[a] State level benefits of the final rule are scaled by the distribution of mortality benefits simulated from the interim scenario described in Appendices 5A, 5B, and 5C.

[b] Estimates rounded to two significant figures; column values will not sum to total value. These estimates do not include confidence intervals.

[c] While climate benefits are included in the total co-benefits of this rule as described in Chapter 5, only health benefits (sum of mortality and morbidity endpoints) are included in the national total here.

APPENDIX 5E

TECHNICAL SUPPORT DOCUMENT:

SUMMARY OF EXPERT OPINIONS ON THE EXISTENCE OF A THRESHOLD IN THE CONCENTRATION-RESPONSE FUNCTION FOR $PM_{2.5}$-RELATED MORTALITY[28]

[28] U.S. Environmental Protection Agency. 2010. Technical Support Document: Summary of Expert Opinions on the Existence of a Threshold in the Concentration-Response Function for $PM_{2.5}$-related Mortality. Research Triangle Park, NC. June. Available on the Internet at: <www.epa.gov/ttn/ecas/regdata/Benefits/thresholdstsd.pdf>.

Summary of Expert Opinions on the Existence of a Threshold in the Concentration-Response Function for $PM_{2.5}$-related Mortality

Technical Support Document (TSD)

June 2010

Compiled by:
U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impact Division
Air Benefit-Cost Group
Research Triangle Park, North Carolina

Contents:

A. HES Comments on 812 Analysis (2010)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2010. Review of EPA's DRAFT Health Benefits of the Second Section 812 Prospective Study of the Clean Air Act. EPA-COUNCIL-10-001. June. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/0/72D4EFA39E48CDB28525774500738776/$File/EPA-COUNCIL-10-001-unsigned.pdf>.

Pg 2: "The HES generally agrees with other decisions made by the EPA project team with respect to PM, in particular, the PM mortality effect threshold model, the cessation lag model, the inclusion of infant mortality estimation, and differential toxicity of PM."

Pg 2: "Further, the HES fully supports EPA's use of a no-threshold model to estimate the mortality reductions associated with reduced PM exposure."

Pg 6: "The HES also supports the Agency's choice of a no-threshold model for PM-related effects."

Pg 13: "The HES fully supports EPA's decision to use a no-threshold model to estimate mortality reductions. This decision is supported by the data, which are quite consistent in showing effects down to the lowest measured levels. Analyses of cohorts using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality. Therefore, there is no evidence to support a truncation of the CRF."

HES Panel Members

Dr. John Bailar, Chair of the Health Effects Subcommittee, Scholar in Residence, The National Academies, Washington, DC

Dr. Michelle Bell, Associate Professor, School of Forestry and Environmental Studies, Yale University, New Haven, CT

Dr. James K. Hammitt, Professor, Department of Health Policy and Management, Harvard School of Public Health, Boston, MA

Dr. Jonathan Levy, Associate Professor, Department of Environmental Health, Harvard School of Public Health, Boston, MA

Dr. C. Arden Pope, III Professor, Department of Economics, Brigham Young University, Provo, UT

Mr. John Fintan Hurley, Research Director, Institute of Occupational Medicine (IOM), Edinburgh, United Kingdom, UK

Dr. Patrick Kinney, Professor, Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, New York, NY

Dr. Michael T. Kleinman, Professor, Department of Medicine, Division of Occupational and Environmental Medicine, University of California, Irvine, Irvine, CA

Dr. Bart Ostro, Chief, Air Pollution Epidemiology Unit, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland, CA

Dr. Rebecca Parkin, Professor and Associate Dean, Environmental and Occupational Health, School of Public Health and Health Services, The George Washington University Medical Center, Washington, DC

B. Scientific Statement from American Heart Association (2010)

Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC Jr, Whitsel L, Kaufman JD; on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism. (2010). "Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association." *Circulation*. 121: 2331-2378.

Pg 2338: "Finally, there appeared to be no lower-limit threshold below which PM_{10} was not associated with excess mortality across all regions."

Pg 2350: "There also appears to be a monotonic (eg, linear or log-linear) concentration-response relationship between $PM_{2.5}$ and mortality risk observed in cohort studies that extends below present-day regulations of 15 $\mu g/m^3$ for mean annual levels, without a discernable "safe" threshold." (cites Pope 2004, Krewski 2009, and Schwartz 2008)

Pg 2364: "The $PM_{2.5}$ concentration– cardiovascular risk relationships for both short- and long-term exposures appear to be monotonic, extending below 15 $\mu g/m^3$ (the 2006 annual NAAQS level) without a discernable "safe" threshold."

Pg 2365: "This updated review by the AHA writing group corroborates and strengthens the conclusions of the initial scientific statement. In this context, we agree with the concept and continue to support measures based on scientific evidence, such as the US EPA NAAQS, that seek to control PM levels to protect the public health. Because the evidence reviewed supports that there is no safe threshold, it appears that public health benefits would accrue from lowering $PM_{2.5}$ concentrations even below present-day annual (15 $\mu g/m^3$) and 24-hour (35 $\mu g/m^3$) NAAQS, if feasible, to optimally protect the most susceptible populations."

Pg 2366: "Although numerous insights have greatly enhanced our understanding of the PM-cardiovascular relationship since the first AHA statement was published, the following list represents broad strategic avenues for future investigation: ... Determine whether any "safe" PM threshold concentration exists that eliminates both acute and chronic cardiovascular effects in healthy and susceptible individuals and at a population level."

<u>Scientific Statement Authors</u>

Dr. Robert D. Brook, MD

Dr. Sanjay Rajagopalan, MD

Dr. C. Arden Pope, PhD

Dr. Jeffrey R. Brook, PhD

Dr. Aruni Bhatnagar, PhD, FAHA

Dr. Ana V. Diez-Roux, MD, PhD, MPH

Dr. Fernando Holguin, MD

Dr. Yuling Hong, MD, PhD, FAHA

Dr. Russell V. Luepker, MD, MS, FAHA

Dr. Murray A. Mittleman, MD, DrPH, FAHA

Dr. Annette Peters, PhD

Dr. David Siscovick, MD, MPH, FAHA

Dr. Sidney C. Smith, Jr, MD, FAHA

Dr. Laurie Whitsel, PhD

Dr. Joel D. Kaufman, MD, MPH

C. Integrated Science Assessment for Particulate Matter (2009)

U.S. Environmental Protection Agency (U.S. EPA). 2009. Integrated Science Assessment for Particulate Matter (Final Report). EPA-600-R-08-139F. National Center for Environmental Assessment – RTP Division. December. Available on the Internet at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546>.

Pg 1-22: "An important consideration in characterizing the public health impacts associated with exposure to a pollutant is whether the concentration-response relationship is linear across the full concentration range encountered, or if nonlinear relationships exist along any part of this range. Of particular interest is the shape of the concentration-response curve at and below the level of the current standards. The shape of the concentration-response curve varies, depending on the type of health outcome, underlying biological mechanisms and dose. At the human population level, however, various sources of variability and uncertainty tend to smooth and "linearize" the concentration-response function (such as the low data density in the lower concentration range, possible influence of measurement error, and individual differences in susceptibility to air pollution health effects). In addition, many chemicals and agents may act by perturbing naturally occurring background processes that lead to disease, which also linearizes population concentration-response relationships (Clewell and Crump, 2005, 156359; Crump et al., 1976, 003192; Hoel, 1980, 156555). These attributes of population dose-response may explain why the available human data at ambient concentrations for some environmental pollutants (e.g., PM, O_3, lead [Pb], ETS, radiation) do not exhibit evident thresholds for health effects, even though likely mechanisms include nonlinear processes for some key events. These attributes of human population dose-response relationships have been extensively discussed in the broader epidemiologic literature (Rothman and Greenland, 1998, 086599)."

Pg 2-16: "In addition, cardiovascular hospital admission and mortality studies that examined the PM_{10} concentration-response relationship found evidence of a log-linear no-threshold relationship between PM exposure and cardiovascular-related morbidity (Section 6.2) and mortality (Section 6.5)."

Pg 2-25: "2.4.3. PM Concentration-Response Relationship
An important consideration in characterizing the PM-morbidity and mortality association is whether the concentration-response relationship is linear across the full concentration range that is encountered or if there are concentration ranges where there are departures from linearity (i.e., nonlinearity). In this ISA studies have been identified that attempt to characterize the shape of the concentration-response curve along with possible PM "thresholds" (i.e., levels which PM concentrations must exceed in order to elicit a health response). The epidemiologic studies evaluated that examined the shape of the concentration-response curve and the potential presence of a threshold have focused on cardiovascular hospital admissions and ED visits and mortality associated with short-term exposure to PM_{10} and mortality associated with long-term exposure to $PM_{2.5}$.

"A limited number of studies have been identified that examined the shape of the PM cardiovascular hospital admission and ED visit concentration-response relationship. Of these studies, some conducted an exploratory analysis during model selection to determine if a linear

curve most adequately represented the concentration-response relationship; whereas, only one study conducted an extensive analysis to examine the shape of the concentration-response curve at different concentrations (Section 6.2.10.10). Overall, the limited evidence from the studies evaluated supports the use of a no-threshold, log-linear model, which is consistent with the observations made in studies that examined the PM-mortality relationship.

"Although multiple studies have previously examined the PM-mortality concentration-response relationship and whether a threshold exists, more complex statistical analyses continue to be developed to analyze this association. Using a variety of methods and models, most of the studies evaluated support the use of a no-threshold, log-linear model; however, one study did observe heterogeneity in the shape of the concentration-response curve across cities (Section 6.5). Overall, the studies evaluated further support the use of a no-threshold log-linear model, but additional issues such as the influence of heterogeneity in estimates between cities, and the effect of seasonal and regional differences in PM on the concentration-response relationship still require further investigation.

"In addition to examining the concentration-response relationship between short-term exposure to PM and mortality, Schwartz et al. (2008, 156963) conducted an analysis of the shape of the concentration-response relationship associated with long-term exposure to PM. Using a variety of statistical methods, the concentration-response curve was found to be indistinguishable from linear, and, therefore, little evidence was observed to suggest that a threshold exists in the association between long-term exposure to $PM_{2.5}$ and the risk of death (Section 7.6)."

Pg 6-75: "6.2.10.10. Concentration Response
The concentration-response relationship has been extensively analyzed primarily through studies that examined the relationship between PM and mortality. These studies, which have focused on short- and long-term exposures to PM have consistently found no evidence for deviations from linearity or a safe threshold (Daniels et al., 2004, 087343; Samoli et al., 2005, 087436; Schwartz, 2004, 078998; Schwartz et al., 2008, 156963) (Sections 6.5.2.7 and 7.1.4). Although on a more limited basis, studies that have examined PM effects on cardiovascular hospital admissions and ED visits have also analyzed the PM concentration-response relationship, and contributed to the overall body of evidence which suggests a log-linear, no-threshold PM concentration-response relationship.

"The results from the three multicity studies discussed above support no-threshold log-linear models, but issues such as the possible influence of exposure error and heterogeneity of shapes across cities remain to be resolved. Also, given the pattern of seasonal and regional differences in PM risk estimates depicted in recent multicity study results (e.g., Peng et al., 2005, 087463), the very concept of a concentration-response relationship estimated across cities and for all-year data may not be very informative."

Pg 6-197: "6.5.2.7. Investigation of Concentration-Response Relationship
The results from large multicity studies reviewed in the 2004 PM AQCD (U.S. EPA, 2004, 056905) suggested that strong evidence did not exist for a clear threshold for PM mortality effects. However, as discussed in the 2004 PM AQCD (U.S. EPA, 2004, 056905), there are several challenges in determining and interpreting the shape of PM-mortality concentration-

response functions and the presence of a threshold, including: (1) limited range of available concentration levels (i.e., sparse data at the low and high end); (2) heterogeneity of susceptible populations; and (3) investigate the PM-mortality concentration-response relationship.

"Daniels et al. (2004, 087343) evaluated three concentration-response models: (1) log-linear models (i.e., the most commonly used approach, from which the majority of risk estimates are derived); (2) spline models that allow data to fit possibly non-linear relationship; and (3) threshold models, using PM_{10} data in 20 cities from the 1987-1994 NMMAPS data. They reported that the spline model, combined across the cities, showed a linear relation without indicating a threshold for the relative risks of death for all-causes and for cardiovascular-respiratory causes in relation to PM_{10}, but "the other cause" deaths (i.e., all cause minus cardiovascular-respiratory) showed an apparent threshold at around 50 $\mu g/m^3$ PM^{10}, as shown in Figure 6-35. For all-cause and cardio-respiratory deaths, based on the Akaike's Information Criterion (AIC), a log-linear model without threshold was preferred to the threshold model and to the spline model.

"The HEI review committee commented that interpretation of these results required caution, because (1) the measurement error could obscure any threshold; (2) the city-specific concentration-response curves exhibited a variety of shapes; and (3) the use of AIC to choose among the models might not be appropriate due to the fact it was not designed to assess scientific theories of etiology. Note, however, that there has been no etiologically credible reason suggested thus far to choose one model over others for aggregate outcomes. Thus, at least statistically, the result of Daniels et al. (2004, 087343) suggests that the log-linear model is appropriate in describing the relationship between PM10 and mortality.

"The Schwartz (2004, 078998) analysis of PM_{10} and mortality in 14 U.S. cities, described in Section 6.5.2.1, also examined the shape of the concentration-response relationship by including indicator variables for days when concentrations were between 15 and 25 $\mu g/m^3$, between 25 and 34 $\mu g/m^3$, between 35 and 44 $\mu g/m^3$, and 45 $\mu g/m^3$ and above. In the model, days with concentrations below 15 $\mu g/m^3$ served as the reference level. This model was fit using the single stage method, combining strata across all cities in the case-crossover design. Figure 6-36 shows the resulting relationship, which does not provide sufficient evidence to suggest that a threshold exists. The authors did not examine city-to-city variation in the concentration-response relationship in this study.

"PM_{10} and mortality in 22 European cities (and BS in 15 of the cities) participating in the APHEA project. In nine of the 22 cities, PM10 levels were estimated using a regression model relating co-located PM10 to BS or TSP. They used regression spline models with two knots (30 and 50 $\mu g/m^3$) and then combined the individual city estimates of the splines across cities. The investigators concluded that the association between PM and mortality in these cities could be adequately estimated using the log-linear model. However, in an ancillary analysis of the concentration-response curves for the largest cities in each of the three distinct geographic areas (western, southern, and eastern European cities): London, England; Athens, Greece; and Cracow, Poland, Samoli et al. (2005, 087436) observed a difference in the shape of the concentration-response curve across cities. Thus, while the combined curves (Figure 6-37) appear to support

no-threshold relationships between PM_{10} and mortality, the heterogeneity of the shapes across cities makes it difficult to interpret the biological relevance of the shape of the combined curves.

"The results from the three multicity studies discussed above support no-threshold log-linear models, but issues such as the possible influence of exposure error and heterogeneity of shapes across cities remain to be resolved. Also, given the pattern of seasonal and regional differences in PM risk estimates depicted in recent multicity study results (e.g., Peng et al., 2005, 087463), the very concept of a concentration-response relationship estimated across cities and for all-year data may not be very informative."

Authors of ISA

Dr. Lindsay Wichers Stanek (PM Team Leader)—National Center for Environmental Assessment (NCEA), U.S. Environmental Protection Agency (U.S. EPA), Research Triangle Park, NC

Dr. Jeffrey Arnold—NCEA, U.S. EPA, Research Triangle Park, NC (now at Institute for Water Resources, U.S. Army Corps of Engineers, Washington, D.C)

Dr. Christal Bowman—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. James S. Brown—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Barbara Buckley—NCEA, U.S. EPA, Research Triangle Park, NC

Mr. Allen Davis—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Jean-Jacques Dubois—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Steven J. Dutton—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Tara Greaver—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Erin Hines—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Douglas Johns—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Ellen Kirrane—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Dennis Kotchmar—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Thomas Long—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Thomas Luben—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Qingyu Meng—Oak Ridge Institute for Science and Education, Postdoctoral Research Fellow to NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Kristopher Novak—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Joseph Pinto—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Jennifer Richmond-Bryant—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Mary Ross—NCEA, U.S. EPA, Research Triangle Park, NC

Mr. Jason Sacks—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Timothy J. Sullivan—E&S Environmental Chemistry, Inc., Corvallis, OR

Dr. David Svendsgaard—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Lisa Vinikoor—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. William Wilson—NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Lori White— NCEA, U.S. EPA, Research Triangle Park, NC (now at National Institute for Environmental Health Sciences, Research Triangle Park, NC)

Dr. Christy Avery—University of North Carolina, Chapel Hill, NC

Dr. Kathleen Belanger —Center for Perinatal, Pediatric and Environmental Epidemiology, Yale University, New Haven, CT

Dr. Michelle Bell—School of Forestry & Environmental Studies, Yale University, New Haven, CT

Dr. William D. Bennett—Center for Environmental Medicine, Asthma and Lung Biology, University of North Carolina, Chapel Hill, NC

Dr. Matthew J. Campen—Lovelace Respiratory Research Institute, Albuquerque, NM

Dr. Leland B. Deck— Stratus Consulting, Inc., Washington, DC

Dr. Janneane F. Gent—Center for Perinatal, Pediatric and Environmental Epidemiology, Yale University, New Haven, CT

Dr. Yuh-Chin Tony Huang—Department of Medicine, Division of Pulmonary Medicine, Duke University Medical Center, Durham, NC

Dr. Kazuhiko Ito—Nelson Institute of Environmental Medicine, NYU School of Medicine, Tuxedo, NY

Mr. Marc Jackson—Integrated Laboratory Systems, Inc., Research Triangle Park, NC

Dr. Michael Kleinman—Department of Community and Environmental Medicine, University of California, Irvine

Dr. Sergey Napelenok—National Exposure Research Laboratory, U.S. EPA, Research Triangle Park, NC

Dr. Marc Pitchford—National Oceanic and Atmospheric Administration, Las Vegas, NV

Dr. Les Recio—Genetic Toxicology Division, Integrated Laboratory Systems, Inc., Research Triangle Park, NC

Dr. David Quincy Rich—Department of Epidemiology, University of Medicine and Dentistry of New Jersey, Piscataway, NJ

Dr. Timothy Sullivan— E&S Environmental Chemistry, Inc., Corvallis, OR

Dr. George Thurston—Department of Environmental Medicine, NYU, Tuxedo, NY

Dr. Gregory Wellenius—Cardiovascular Epidemiology Research Unit, Beth Israel Deaconess Medical Center, Boston, MA

Dr. Eric Whitsel—Departments of Epidemiology and Medicine, University of North Carolina, Chapel Hill, NC

Peer Reviewers

Dr. Sara Dubowsky Adar, Department of Epidemiology, University of Washington, Seattle, WA

Mr. Chad Bailey, Office of Transportation and Air Quality, Ann Arbor, MI

Mr. Richard Baldauf, Office of Transportation and Air Quality, Ann Arbor, MI

Dr. Prakash Bhave, National Exposure Research Laboratory, U.S. EPA, Research Triangle Park, NC

Mr. George Bowker, Office of Atmospheric Programs, U.S. EPA, Washington, D.C.

Dr. Judith Chow, Division of Atmospheric Sciences, Desert Research Institute, Reno, NV

Dr. Dan Costa, U.S. EPA, Research Triangle Park, NC

Dr. Ila Cote, NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Robert Devlin, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC

Dr. David DeMarini, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC

Dr. Neil Donahue, Department of Chemical Engineering, Carnegie Mellon University, Pittsburgh, PA

Dr. Aimen Farraj, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC

Dr. Mark Frampton, Department of Environmental Medicine, University of Rochester Medical Center, Rochester, NY

Mr. Neil Frank, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Mr. Tyler Fox, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Jim Gauderman, Department of Environmental Medicine, Department of Preventive Medicine, University of Southern California, Los Angeles, CA

Dr. Barbara Glenn, National Center for Environmental Research, U.S. EPA, Washington, D.C.

Dr. Terry Gordon, School of Medicine, New York University, Tuxedo, NY

Mr. Tim Hanley, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Jack Harkema, Department of Pathobiology and Diagnostic Investigation, Michigan State University, East Lansing, MI

Ms. Beth Hassett-Sipple, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Amy Herring, Department of Biostatistics, University of North Carolina, Chapel Hill, NC

Dr. Israel Jirak, Department of Meteorology, Embry-Riddle Aeronautical University, Prescott, AZ

Dr. Mike Kleeman, Department of Civil and Environmental Engineering, University of California, Davis, CA

Dr. Petros Koutrakis, Exposure, Epidemiology and Risk Program, Harvard School of Public Health, Boston, MA

Dr. Sagar Krupa, Department of Plant Pathology, University of Minnesota, St. Paul, MN

Mr. John Langstaff, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Meredith Lassiter, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Mr. Phil Lorang, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Karen Martin, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Ms. Connie Meacham, NCEA, U.S. EPA, Research Triangle Park, NC

Mr. Tom Pace, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Jennifer Peel, Department of Environmental and Radiological Health Sciences, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO

Dr. Zackary Pekar, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Mr. Rob Pinder, National Exposure Research Laboratory, U.S. EPA, Research Triangle Park, NC

Mr. Norm Possiel, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Sanjay Rajagopalan, Division of Cardiovascular Medicine, Ohio State University, Columbus, OH

Dr. Pradeep Rajan, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Mr. Venkatesh Rao, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Ms. Joann Rice, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Mr. Harvey Richmond, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Ms. Victoria Sandiford, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Stefanie Sarnat, Department of Environmental and Occupational Health, Emory University, Atlanta, GA

Dr. Frances Silverman, Gage Occupational and Environmental Health, University of Toronto, Toronto, ON

Mr. Steven Silverman, Office of General Council, U.S. EPA, Washington, D.C.

Dr. Barbara Turpin, Department of Environmental Sciences, Rutgers University, New Brunswick, NJ

Dr. Robert Vanderpool, National Exposure Research Laboratory, U.S. EPA, Research Triangle Park, NC

Dr. John Vandenberg (Director)—NCEA-RTP Division, U.S. EPA, Research Triangle Park, NC

Dr. Alan Vette, National Exposure Research Laboratory, U.S. EPA, Research Triangle Park, NC

Ms. Debra Walsh (Deputy Director)—NCEA-RTP Division, U.S. EPA, Research Triangle Park, NC

Mr. Tim Watkins, National Exposure Research Laboratory, U.S. EPA, Research Triangle Park, NC

Dr. Christopher Weaver, NCEA, U.S. EPA, Research Triangle Park, NC

Mr. Lewis Weinstock, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Ms. Karen Wesson, Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC

Dr. Jason West, Department of Environmental Sciences and Engineering, University of North Carolina, Chapel Hill, NC

Mr. Ronald Williams, National Exposure Research Laboratory, U.S. EPA, Research Triangle Park, NC

Dr. George Woodall, NCEA, U.S. EPA, Research Triangle Park, NC

Dr. Antonella Zanobetti, Department of Environmental Health, Harvard School of Public Health, Boston, MA

D. CASAC comments on PM ISA and REA (2009)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2009. Review of EPA's Integrated Science Assessment for Particulate Matter (First External Review Draft, December 2008). EPA-COUNCIL-09-008. May. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/73ACCA834AB44A10852575BD0064346B/$File/EPA-CASAC-09-008-unsigned.pdf>.

Pg 9: "There is an appropriate discussion of the time-series studies, but this section needs to have an explicit finding that the evidence supports a relationship between PM and mortality that is seen in these studies. This conclusion should be followed by the discussion of statistical methodology and the identification of any threshold that may exist."

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2009. Consultation on EPA's Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment. EPA-COUNCIL-09-009. May. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/723FE644C5D758DF852575BD00763A32/$File/EPA-CASAC-09-009-unsigned.pdf>.

Pg 6: "On the issue of cut-points raised on 3-18, the authors should be prepared to offer a scientifically cogent reason for selection of a specific cut-point, and not simply try different cut-points to see what effect this has on the analysis. The draft ISA was clear that there is little evidence for a population threshold in the C-R function."

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2009. Review of *Integrated Science Assessment for Particulate Matter (Second External Review Draft, July 2009)*. EPA-CASAC-10-001. November. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/151B1F83B0231455852576780068 36B9/$File/EPA-CASAC-10-001-unsigned.pdf>.

Pg 2: "The paragraph on lines 22-30 of page 2-37 is not clearly written. Twice in succession it states that the use of a no-threshold log-linear model is supported, but then cites other studies that suggest otherwise. It would be good to revise this paragraph to more clearly state – well, I'm not sure what. Probably that more research is needed."

CASAC Panel Members

Dr. Jonathan M. Samet, Professor and Chair, Department of Preventive Medicine, University of Southern California, Los Angeles, CA

Dr. Joseph Brain, Philip Drinker Professor of Environmental Physiology, Department of Environmental Health, Harvard School of Public Health, Harvard University, Boston, MA

Dr. Ellis B. Cowling, University Distinguished Professor At-Large Emeritus, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James Crapo, Professor of Medicine, Department of Medicine, National Jewish Medical and Research Center, Denver, CO

Dr. H. Christopher Frey, Professor, Department of Civil, Construction and Environmental Engineering, College of Engineering, North Carolina State University, Raleigh, NC

Dr. Armistead (Ted) Russell, Professor, Department of Civil and Environmental Engineering, Georgia Institute of Technology, Atlanta, GA

Dr. Lowell Ashbaugh, Associate Research Ecologist, Crocker Nuclear Lab, University of California, Davis, Davis, CA

Prof. Ed Avol, Professor, Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA

Dr. Wayne Cascio, Professor, Medicine, Cardiology, Brody School of Medicine at East Carolina University, Greenville, NC

Dr. David Grantz, Director, Botany and Plant Sciences and Air Pollution Research Center, Riverside Campus and Kearney Agricultural Center, University of California, Parlier, CA

Dr. Joseph Helble, Dean and Professor, Thayer School of Engineering, Dartmouth College, Hanover, NH

Dr. Rogene Henderson, Senior Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

Dr. Philip Hopke, Bayard D. Clarkson Distinguished Professor, Department of Chemical Engineering, Clarkson University, Potsdam, NY

Dr. Morton Lippmann, Professor, Nelson Institute of Environmental Medicine, New York University School of Medicine, Tuxedo, NY

Dr. Helen Suh MacIntosh, Associate Professor, Environmental Health, School of Public Health, Harvard University, Boston, MA

Dr. William Malm, Research Physicist, National Park Service Air Resources Division, Cooperative Institute for Research in the Atmosphere, Colorado State University, Fort Collins, CO

Mr. Charles Thomas (Tom) Moore, Jr., Air Quality Program Manager, Western Governors' Association, Cooperative Institute for Research in the Atmosphere, Colorado State University, Fort Collins, CO

Dr. Robert F. Phalen, Professor, Department of Community & Environmental Medicine; Director, Air Pollution Health Effects Laboratory; Professor of Occupational & Environmental Health, Center for Occupation & Environment Health, College of Medicine, University of California Irvine, Irvine, CA

Dr. Kent Pinkerton, Professor, Regents of the University of California, Center for Health and the Environment, University of California, Davis, CA

Mr. Richard L. Poirot, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Frank Speizer, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

Dr. Sverre Vedal, Professor, Department of Environmental and Occupational Health Sciences, School of Public Health and Community Medicine, University of Washington, Seattle, WA

Dr. Donna Kenski, Data Analysis Director, Lake Michigan Air Directors Consortium, Rosemont, IL

Dr. Kathy Weathers, Senior Scientist, Cary Institute of Ecosystem Studies, Millbrook, NY

E. Krewski et al. (2009)

Krewski, Daniel, Michael Jerrett, Richard T. Burnett, Renjun Ma, Edward Hughes, Yuanli Shi, Michelle C. Turner, C. Arden Pope III, George Thurston, Eugenia E. Calle, and Michael J. Thun with Bernie Beckerman, Pat DeLuca, Norm Finkelstein, Kaz Ito, D.K. Moore, K. Bruce Newbold, Tim Ramsay, Zev Ross, Hwashin Shin, and Barbara Tempalski. (2009). Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. *HEI Research Repor*t, 140, Health Effects Institute, Boston, MA.

Pg 119: [About Pope et al. (2002)] "Each 10-μg/m^3 increase in longterm average ambient $PM_{2.5}$ concentrations was associated with approximately a 4%, 6%, or 8% increase in risk of death from all causes, cardiopulmonary disease, and lung cancer, respectively. There was no evidence of a threshold exposure level within the range of observed $PM_{2.5}$ concentrations. "

Krewski (2009). Letter from Dr. Daniel Krewski to HEI's Dr. Kate Adams (dated July July 7, 2009) regarding "EPA queries regarding HEI Report 140". Dr. Adams then forwarded the letter on July 10, 2009 to EPA's Beth Hassett-Sipple. (letter placed in docket #EPA-HQ-OAR-2007-0492).

Pg 4: "6. The Health Review Committee commented that the Updated Analysis completed by Pope et al. 2002 reported "no evidence of a threshold exposure level within the range of observed $PM_{2.5}$ concentrations" (p. 119). In the Extended Follow-Up study, did the analyses provide continued support for a no-threshold response or was there evidence of a threshold?

"Response: As noted above, the HEI Health Review Committee commented on the lack of evidence for a threshold exposure level in Pope et al. (2002) with follow-up through the year 1998. The present report, which included follow-up through the year 2000, also does not appear to demonstrate the existence of a threshold in the exposure-response function within the range of observed $PM_{2.5}$ concentrations."

<u>HEI Health Review Committee Members</u>

Dr. Homer A. Boushey, MD, Chair, Professor of Medicine, Department of Medicine, University of California–San Francisco

Dr. Ben Armstrong, Reader, in Epidemiological Statistics, Department of Public Health and Policy, London School of Hygiene and Tropical Medicine, United Kingdom

Dr. Michael Brauer, ScD, Professor, School of Environmental Health, University of British Columbia, Canada

Dr. Bert Brunekreef, PhD, Professor of Environmental Epidemiology, Institute of Risk Assessment Sciences, University of Utrecht, The Netherlands

Dr. Mark W. Frampton, MD, Professor of Medicine & Environmental Medicine, University of Rochester Medical Center, Rochester, NY

Dr. Stephanie London, MD, PhD, Senior Investigator, Epidemiology Branch, National Institute of Environmental Health Sciences

Dr. William N. Rom, MD, MPH, Sol and Judith Bergstein Professor of Medicine and Environmental Medicine and Director of Pulmonary and Critical Care Medicine, New York University Medical Center

Dr. Armistead Russell, Georgia Power Distinguished Professor of Environmental Engineering, School of Civil and Environmental Engineering, Georgia Institute of Technology

Dr. Lianne Sheppard, PhD, Professor, Department of Biostatistics, University of Washington

F. Schwartz et al. (2008)

Schwartz J, Coull B, Laden F. (2008). The Effect of Dose and Timing of Dose on the Association between Airborne Particles and Survival. *Environmental Health Perspectives*. 116: 64-69.

Pg 67: "A key finding of this study is that there is little evidence for a threshold in the association between exposure to fine particles and the risk of death on follow-up, which continues well below the U.S. EPA standard of 15 $\mu g/m^3$."

Pg 68: "In conclusion, penalized spline smoothing and model averaging represent reasonable, feasible approaches to addressing questions of the shape of the exposure–response curve, and can provide valuable information to decisionmakers. In this example, both approaches are consistent, and suggest that the association of particles with mortality has no threshold down to close to background levels."

G. Expert Elicitation on PM-Mortality (2006, 2008)

Industrial Economics, Inc., 2006. ***Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between $PM_{2.5}$ Exposure and Mortality*****. Prepared for the U.S.EPA, Office of Air Quality Planning and Standards, September. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/Uncertainty/pm_ee_report.pdf>.**

Pg v: "Each expert was given the option to integrate their judgments about the likelihood of a causal relationship and/or threshold in the C-R function into his distribution or to provide a distribution "conditional on" one or both of these factors."

Pg vii: "Only one of 12 experts explicitly incorporated a threshold into his C-R function.[3] The rest believed there was a lack of empirical and/or theoretical support for a population threshold. However, three other experts gave differing effect estimate distributions above and below some cut-off concentration. The adjustments these experts made to median estimates and/or uncertainty at lower $PM^{2.5}$ concentrations were modest."

> "[3] Expert K indicated that he was 50 percent sure that a threshold existed. If there were a threshold, he thought that there was an 80 percent chance that it would be less than or equal to 5 $\mu g/m^3$, and a 20 percent chance that it would fall between 5 and 10 $\mu g/m^3$."

Pg ix: "Compared to the pilot study, experts in this study were in general more confident in a causal relationship, less likely to incorporate thresholds, and reported higher mortality effect estimates. The differences in results compared with the pilot appear to reflect the influence of new research on the interpretation of the key epidemiological studies that were the focus of both elicitation studies, more than the influence of changes to the structure of the protocol."

Pg 3-25: "3.1.8 THRESHOLDS
The protocol asked experts for their judgments regarding whether a threshold exists in the $PM_{2.5}$ mortality C-R function. The protocol focused on assessing expert judgments regarding theory and evidential support for a population threshold (i.e., the concentration below which no member of the study population would experience an increased risk of death).[32] If an expert wished to incorporate a threshold in his characterization of the concentration-response relationship, the team then asked the expert to specify the threshold $PM_{2.5}$ concentration probabilistically, incorporating his uncertainty about the true threshold level.

"From a theoretical and conceptual standpoint, all experts generally believed that individuals exhibit thresholds for PM-related mortality. However, 11 of them discounted the idea of a population threshold in the C-R function on a theoretical and/or empirical basis. Seven of these experts noted that theoretically one would be unlikely to observe a population threshold due to the variation in susceptibility at any given time in the study population resulting from combinations of genetic, environmental, and socioeconomic factors.[33] All 11 thought that there was insufficient empirical support for a population threshold in the C-R function. In addition, two experts (E and L) cited analyses of the ACS cohort data in Pope et al. (2002) and another (J) cited Krewski et al. (2000a & b) as supportive of a linear relationship in the study range.

"Seven of the experts favored epidemiological studies as ideally the best means of addressing the population threshold issue, because they are best able to evaluate the full range of susceptible individuals at environmentally relevant exposure levels. However, those who favored epidemiologic studies generally acknowledged that definitive studies addressing thresholds would be difficult or impossible to conduct, because they would need to include a very large and diverse population with wide variation in exposure and a long follow-up period. Furthermore, two experts (B and I) cited studies documenting difficulties in detecting a threshold using epidemiological studies (Cakmak et al. 1999, and Brauer et al., 2002, respectively). The experts generally thought that clinical and toxicological studies are best suited for researching mechanisms and for addressing thresholds in very narrowly defined groups. One expert, B, thought that a better understanding of the detailed biological mechanism is critical to addressing the question of a threshold.

"One expert, K, believed it was possible to make a conceptual argument for a population threshold. He drew an analogy with smoking, indicating that among heavy smokers, only a proportion of them gets lung cancer or demonstrates an accelerated decline in lung function. He thought that the idea that there is no level that is biologically safe is fundamentally at odds with toxicological theory. He did not think that a population threshold was detectable in the currently available epidemiologic studies. He indicated that some of the cohort studies showed greater uncertainty in the shape of the C-R function at lower levels, which could be indicative of a threshold.

"Expert K chose to incorporate a threshold into his C-R function. He indicated that he was 50 percent sure that a threshold existed. If there were a threshold, he thought that there was an 80 percent chance that it would be less than or equal to 5 $\mu g/m^3$, and a 20 percent chance that it would fall between 5 and 10 $\mu g/m^3$."

Roman, Henry A., Katherine D. Walker, Tyra L. Walsh, Lisa Conner, Harvey M. Richmond, Bryan J. Hubbell, and Patrick L. Kinney. (2008). "Expert Judgment Assessment of the Mortality Impact of Changes in Ambient Fine Particulate Matter in the U.S." *Environ. Sci. Technol.*, 42(7):2268-2274.

Pg 2271: "Eight experts thought the true C-R function relating mortality to changes in annual average $PM_{2.5}$ was log-linear across the entire study range (ln(mortality)) $\beta \times$ PM). Four experts (B, F, K, and L) specified a "piecewise" log-linear function, with different β coefficients for PM concentrations above and below an expert-specified break point. This approach allowed them to express increased uncertainty in mortality effects seen at lower concentrations in major epidemiological studies. Expert K thought the relationship would be log-linear above a threshold."

Pg 2271: "Expert K also applied a threshold, T, to his function, which he described probabilistically. He specified $P(T > 0) = 0.5$. Given $T > 0$, he indicated $P(T \leq 5\ \mu g/m^3) = 0.8$ and $P(5\ \mu g/m^3 < T \leq 10\ \mu g/m^3) = 0.2$. Figure 3 does not include the impact of applying expert K's threshold, as the size of the reduction in benefits will depend on the distribution of baseline PM levels in a benefits analysis."

Experts:

Dr. Doug W. Dockery, Harvard School of Public Health

Dr. Kazuhiko Ito, Nelson Institute of Environmental Medicine, NYU School of Medicine, Tuxedo, NY

Dr. Dan Krewski, University of Ottawa

Dr. Nino Künzli, University of Southern California Keck School of Medicine

Dr. Morton Lippmann, Professor, Nelson Institute of Environmental Medicine, New York University School of Medicine, Tuxedo, NY

Dr. Joe Mauderly, Lovelace Respiratory Research Institute

Dr. Bart Ostro, Chief, Air Pollution Epidemiology Unit, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland, CA

Dr. Arden Pope, Professor, Department of Economics, Brigham Young University, Provo, UT

Dr. Richard Schlesinger, Pace University

Dr. Joel Schwartz, Harvard School of Public Health

Dr. George Thurston—Department of Environmental Medicine, NYU, Tuxedo, NY

Dr. Mark Utell, University of Rochester School of Medicine and Dentistry

H. CASAC comments on PM Staff Paper (2005)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2005. EPA's Review of the National Ambient Air Quality Standards for Particulate Matter (Second Draft PM Staff Paper, January 2005). EPA-SAB-CASAC-05-007. June. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/E523DD36175EB5AD8525701B007332AE/$File/SAB-CASAC-05-007_unsigned.pdf>.

Pg 6: "A second concern is with methodological issues. The issue of the selection of concentration-response (C-R) relationships based on locally-derived coefficients needs more discussion. The Panel did not agree with EPA staff in calculating the burden of associated incidence in their risk assessment using either the predicted background or the lowest measured level (LML) in the utilized epidemiological analysis. The available epidemiological database on daily mortality and morbidity does not establish either the presence or absence of threshold concentrations for adverse health effects. Thus, in order to avoid emphasizing an approach that assumes effects that extend to either predicted background concentrations or LML, and to standardize the approach across cities, for the purpose of estimating public health impacts, the Panel favored the primary use of an assumed threshold of 10 $\mu g/m^3$. The original approach of using background or LML, as well as the other postulated thresholds, could still be used in a sensitivity analysis of threshold assumptions.

"The analyses in this chapter highlight the impact of assumptions regarding thresholds, or lack of threshold, on the estimates of risk. The uncertainty associated with threshold or nonlinear models needs more thorough discussion. A major research need is for more work to determine the existence and level of any thresholds that may exist or the shape of nonlinear concentration-response curves at low levels of exposure that may exist, and to reduce uncertainty in estimated risks at the lowest PM concentrations."

CASAC Panel Members

Dr. Rogene Henderson, Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

Dr. Ellis Cowling, University Distinguished Professor-at-Large, North Carolina State University, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James D. Crapo, Professor, Department of Medicine, Biomedical Research and PatientCare, National Jewish Medical and Research Center, Denver, CO

Dr. Philip Hopke, Bayard D. Clarkson Distinguished Professor, Department of Chemical Engineering, Clarkson University, Potsdam, NY

Dr. Jane Q. Koenig, Professor, Department of Environmental Health, School of Public Health and Community Medicine, University of Washington, Seattle, WA

Dr. Petros Koutrakis, Professor of Environmental Science, Environmental Health , School of Public Health, Harvard University (HSPH), Boston, MA

Dr. Allan Legge, President, Biosphere Solutions, Calgary, Alberta

Dr. Paul J. Lioy, Associate Director and Professor, Environmental and Occupational Health Sciences Institute, UMDNJ - Robert Wood Johnson Medical School, NJ

Dr. Morton Lippmann, Professor, Nelson Institute of Environmental Medicine, New York University School of Medicine, Tuxedo, NY

Dr. Joe Mauderly, Vice President, Senior Scientist, and Director, National Environmental Respiratory Center, Lovelace Respiratory Research Institute, Albuquerque, NM

Dr. Roger O. McClellan, Consultant, Albuquerque, NM

Dr. Frederick J. Miller, Consultant, Cary, NC

Dr. Gunter Oberdorster, Professor of Toxicology, Department of Environmental Medicine, School of Medicine and Dentistry, University of Rochester, Rochester, NY

Mr. Richard L. Poirot, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Robert D. Rowe, President, Stratus Consulting, Inc., Boulder, CO

Dr. Jonathan M. Samet, Professor and Chair, Department of Epidemiology, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD

Dr. Frank Speizer, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

Dr. Sverre Vedal, Professor of Medicine, School of Public Health and Community Medicine University of Washington, Seattle, WA

Mr. Ronald White, Research Scientist, Epidemiology, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD

Dr. Warren H. White, Visiting Professor, Crocker Nuclear Laboratory, University of California -Davis, Davis, CA

Dr. George T. Wolff, Principal Scientist, General Motors Corporation, Detroit, MI

Dr. Barbara Zielinska, Research Professor, Division of Atmospheric Science, Desert Research Institute, Reno, NV

I. HES Comments on 812 Analysis (2004)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2004. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis – Benefits and Costs of the Clean Air Act, 1990-2020. Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis. EPA-SAB-COUNCIL-ADV-04-002. March. Available on the Internet at <http://yosemite.epa.gov/sab%5CSABPRODUCT.NSF/08E1155AD24F871C85256E5400433D5D/$File/council_adv_04002.pdf>.

Pg 20: "The Subcommittee agrees that the whole range of uncertainties, such as the questions of causality, shape of C-R functions and thresholds, relative toxicity, years of life lost, cessation lag structure, cause of death, biologic pathways, or susceptibilities may be viewed differently for acute effects versus long-term effects.

"For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful work on this issue. They report that the associations between $PM_{2.5}$ and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold. Graphical analyses of these studies (Dockery et al., 1993, Figure 3 and Krewski et al., 2000, page 162) also suggest a continuum of effects down to lower levels. Therefore, it is reasonable for EPA to assume a no threshold model down to, at least, the low end of the concentrations reported in the studies."

HES Panel Members

Dr. Bart Ostro, California Office of Environmental Health Hazard Assessment (OEHHA), Oakland, CA

Mr. John Fintan Hurley, Institute of Occupational Medicine (IOM), Edinburgh, Scotland

Dr. Patrick Kinney, Columbia University, New York, NY

Dr. Michael Kleinman, University of California, Irvine, CA

Dr. Nino Künzli, University of Southern California, Los Angeles, CA

Dr. Morton Lippmann, New York University School of Medicine, Tuxedo, NY Dr. Rebecca Parkin, The George Washington University, Washington, DC

Dr. Trudy Cameron, University of Oregon, Eugene, OR

Dr. David T. Allen, University of Texas, Austin, TX

Ms. Lauraine Chestnut, Stratus Consulting Inc., Boulder, CO

Dr. Lawrence Goulder, Stanford University, Stanford, CA

Dr. James Hammitt, Harvard University, Boston, MA

Dr. F. Reed Johnson, Research Triangle Institute, Research Triangle Park, NC

Dr. Charles Kolstad, University of California, Santa Barbara, CA

Dr. Lester B. Lave, Carnegie Mellon University, Pittsburgh, PA

Dr. Virginia McConnell, Resources for the Future, Washington, DC

Dr. V. Kerry Smith, North Carolina State University, Raleigh, NC

Other Panel Members

Dr. John Evans, Harvard University, Portsmouth, NH Dr. Dale Hattis, Clark University, Worcester, MA Dr. D. Warner North, NorthWorks Inc., Belmont, CA Dr. Thomas S. Wallsten, University of Maryland, College Park, MD

J. NRC – Committee on Estimating the Health Risk Reduction Benefits of Proposed Air Pollution Regulations (2002)

National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. Washington, DC: The National Academies Press.

Pg 109: "**Linearity and Thresholds**

"The shape of the concentration-response functions may influence the overall estimate of benefits. The shape is particularly important for lower ambient air pollution concentrations to which a large portion of the population is exposed. For this reason, the impact of the existence of a threshold may be considerable.

"In epidemiological studies, air pollution concentrations are usually measured and modeled as continuous variables. Thus, it may be feasible to test linearity and the existence of thresholds, depending on the study design. In time-series studies with the large number of repeated measurements, linearity and thresholds have been formally addressed with reasonable statistical power. For pollutants such as PM_{10} and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold. For example, examination of the mortality effects of short-term exposure to PM_{10} in 88 cities indicates that the concentration-response functions are not due to the high concentrations and that the slopes of these functions do not appear to increase at higher concentrations (Samet et al. 2000). Many other mortality studies have examined the shape of the concentration-response function and indicated that a linear (nonthreshold) model fit the data well (Pope 2000). Furthermore, studies conducted in cities with very low ambient pollution concentrations have similar effects per unit change in concentration as those studies conducted in cities with higher concentrations. Again, this finding suggests a fairly linear concentration-response function over the observed range of exposures.

"Regarding the studies of long-term exposure, Krewski et al. (2000) found that the assumption of a linear concentration-response function for mortality outcomes was not unreasonable. However, the statistical power to assess the shape of these functions is weakest at the upper and lower end of the observed exposure ranges. Most of the studies examining the effects of long-term exposure on morbidity compare subjects living in a small number of communities (Dockery et al. 1996; Ackermmann-Liebrich 1997; Braun-Fahrländer et al. 1997). Because the number of long-term effects studies are few and the number of communities studied is relatively small (8 to 24), the ability to test formally the absence or existence of a no-effect threshold is not feasible. However, even if thresholds exist, they may not be at the same concentration for all health outcomes.

"A review of the time-series and cohort studies may lead to the conclusion that although a threshold is not apparent at commonly observed concentrations, one may exist at lower levels. An important point to acknowledge regarding thresholds is that for health benefits analysis a key threshold is the population threshold (the lowest of the individual thresholds). However, the population threshold would be very difficult to observe empirically through epidemiology, because epidemiology integrates information from very large groups of people (thousands). Air pollution regulations affect even larger groups of people (millions). It is reasonable to assume that among such large groups susceptibility to air pollution health effects varies considerably across individuals and depends on a large set of underlying factors, including

genetic makeup, age, exposure measurement error, preexisting disease, and simultaneous exposures from smoking and occupational hazards. This variation in individual susceptibilities and the resulting distribution of individual thresholds underlies the concentration-response function observed in epidemiology. Thus, until biologically based models of the distribution of individual thresholds are developed, it may be productive to assume that the population concentration-response function is continuous and to focus on finding evidence of changes in its slope as one approaches lower concentrations.

EPA's Use of Thresholds

"In EPA's benefits analyses, threshold issues were discussed and interpreted. For the PM and ozone National Ambient Air Quality Standards (NAAQS), EPA investigated the effects of a potential threshold or reference value below which health consequences were assumed to be zero (EPA 1997). Specifically, the high-end benefits estimate assumed a 12-microgram per cubic meter ($\mu g/m^3$) mean threshold for mortality associated with long-term exposure to $PM_{2.5}$. The low-end benefits estimate assumed a 15-$\mu g/m^3$ threshold for all PM-related health effects. The studies, however, included concentrations as low as 7.5 $\mu g/m^3$. For the Tier 2 rule and the HD engine and diesel-fuel rule, no threshold was assumed (EPA 1999, 2000). EPA in these analyses acknowledged that there was no evidence for a threshold for PM.

"Several points should be noted regarding the threshold assumptions. If a threshold is assumed where one was not apparent in the original study, then the data should be refit and a new curve generated with the assumption of a zero slope over a segment of the concentration-response function that was originally found to be positively sloped. The assumption of a zero slope over a portion of the curve will force the slope in the remaining segment of the positively sloped concentration-response function to be greater than was indicated in the original study. A new concentration-response function was not generated for EPA's benefits analysis for the PM and ozone NAAQS for which threshold assumptions were made. The generation of the steeper slope in the remaining portion of the concentration-response function may fully offset the effect of assuming a threshold. These aspects of assuming a threshold in a benefits analysis where one was not indicated in the original study should be conveyed to the reader. The committee notes that the treatment of thresholds should be evaluated in a consistent and transparent framework by using different explicit assumptions in the formal uncertainty analyses (see Chapter 5)."

Pg 117: "Although the assumption of no thresholds in the most recent EPA benefits analyses was appropriate, EPA should evaluate threshold assumptions in a consistent and transparent framework using several alternative assumptions in the formal uncertainty analysis."

Pg 136: "Two additional illustrative examples are thresholds for adverse effects and lag structures.[2] EPA considers implausible any threshold for mortality in the particulate matter (PM) exposure ranges under consideration (EPA 1999a, p. 3-8). Although the agency conducts sensitivity analyses incorporating thresholds, it provides no judgment as to their relative plausibility. In a probabilistic uncertainty analysis, EPA could assign appropriate weights to various threshold models. For PM-related mortality in the Tier 2 analysis, the committee expects that this approach would have resulted in only a slight widening of the probability distribution for avoided mortality and a slight reduction in the mean of that distribution, thus reflecting EPA's views about the implausibility of thresholds. The committee finds that such formal incorporation of EPA's expert judgments about the plausibility of thresholds into its primary analysis would have been an improvement.

"Uncertainty about thresholds is a special aspect of uncertainty about the shape of concentration-response functions. Typically, EPA and authors of epidemiological studies assume that these functions are linear on some scale. Often, the scale is a logarithmic transformation of the risk or rate of the health outcome,

but when a rate or risk is low, a linear function on the logarithmic scale is approximately linear on the scale of the rate or risk itself. Increasingly, epidemiological investigators are employing analytic methods that permit the estimation of nonlinear shapes for concentration-response functions (Greenland et al. 1999). As a consequence, EPA will need to be prepared to incorporate nonlinear concentration-response functions from epidemiological studies into the agency's health benefits analyses. Any source of error or bias that can distort an epidemiological association can also distort the shape of an estimated concentration -response function, as can variation in individual susceptibility (Hattis and Burmaster 1994; Hattis et al. 2001)."

Pg 137: "In principle, many components of the health benefits model need realistic probabilistic models (see Table 5-1 for a listing of such components), in addition to concentration-response thresholds and time lags between exposure and response. For example, additional features of the concentration-response function—such as projection of the results from the study population to the target populations (which may have etiologically relevant characteristics outside the range seen in the study population) and the projection of baseline frequencies of morbidity and mortality into the future—must be characterized probabilistically. Other uncertainties that might affect the probability distributions are the estimations of population exposure (or even concentration) from emissions, estimates of emissions themselves, and the relative toxicity of various classes of particles. Similarly, many aspects of the analysis of the impact of regulation on ambient concentrations and on population exposure involve considerable uncertainty and, therefore, may be beneficially modeled in this way. Depending on the analytic approach used, joint probability distributions will have to be specified to incorporate correlations between model components that are structurally dependent upon each other, or the analysis will have to be conducted in a sequential fashion that follows the model for the data-generating process.

"EPA should explore alternative options for incorporating expert judgment into its probabilistic uncertainty analyses. The agency possesses considerable internal expertise, which should be employed as fully as possible. Outside experts should also be consulted as needed, individually or in panels. In all cases, when expert judgment is used in the construction of a model component, the experts should be identified and the rationales and empirical bases for their judgments should be made available."

NRC members

Dr. JOHN C. BAILAR, III (Chair), (emeritus) University of Chicago, Chicago, Illinois

Dr. HUGH ROSS ANDERSON, University of London, London, England

Dr. MAUREEN L. CROPPER, University of Maryland, College Park

Dr. JOHN S. EVANS, Harvard University, Boston, Massachusetts

Dr. DALE B. HATTIS, Clark University, Worcester, Massachusetts

Dr. ROGENE F. HENDERSON, Lovelace Respiratory Research Institute, Albuquerque, New Mexico

Dr. PATRICK L. KINNEY, Columbia University, New York, New York

Dr. NINO KÜNZLI, University of Basel, Basel, Switzerland; as of September 2002, University of Southern California, Los Angeles

Dr. BART D. OSTRO, California Environmental Protection Agency, Oakland

Dr. CHARLES POOLE, University of North Carolina, Chapel Hill

Dr. KIRK R. SMITH, University of California, Berkeley

Dr. PETER A. VALBERG, Gradient Corporation, Cambridge, Massachusetts

Dr. SCOTT L. ZEGER, Johns Hopkins University, Baltimore, Maryland

CHAPTER 6
EMPLOYMENT AND ECONOMIC IMPACT ANALYSIS

In this chapter, we estimate select employment effects of the rule, for both the regulated industry (electric power industry) and the environmental control industry.

6.1 Employment Impacts for the MATS

In addition to addressing the costs and benefits of the MATS, EPA has estimated certain impacts of this on employment, which are presented in this section.[1] While a standalone analysis of employment impacts is not included in a standard cost-benefit analysis, such an analysis is of particular concern in the current economic climate of sustained unemployment. Executive Order 13563, states, "Our regulatory system must protect public health, welfare, safety and our environment while promoting economic growth, innovation, competiveness, and job creation " (emphasis added). Therefore, and consistent with recent efforts to characterize the employment effects of economically significant rules, the Agency has provided this analysis to inform the discussion of labor demand and employment impacts.

The analysis includes two sets of estimates. The first involves the employment impacts on the regulated industry over time. The second involves certain short-term and on-going employment impacts (increase in labor demand) associated with the construction of needed pollution control equipment, and other activities, to comply with the regulation. EPA estimates that the net employment effect on the regulated industry will range from −15,000 to +30,000 jobs, with a central estimate of +8,000. This aggregate figure includes potential job losses from increased costs as well as potential job increases as a result of additional hiring for compliance. In the pollution control sector, EPA estimates an increase of 46,000 job-years. EPA also provides a qualitative discussion of other potential employment effects, including both increases and decreases. Because of the uncertainties involved, these sets of estimates should not be added in an attempt to characterize the overall employment effect.

The Agency has not quantified the rule's effects on all labor in other sectors not regulated by the MATS, or the effects induced by changes in workers' incomes. What follows is an overview of the various ways that environmental regulation can affect employment, followed by a discussion of the estimated impacts of this rule. EPA continues to explore the relevant theoretical and empirical literature, which continues to evolve, and to seek public

[1] See the employment impacts appendix included in this RIA.

comments in order to ensure that such estimates are as accurate, useful, and informative as possible.

From an economic perspective, labor is an input into producing goods and services. If regulation leads to more labor being used to produce a given amount of output, the additional labor is reflected by an increase in the cost of production.[2] When an increase in employment occurs as a result of a regulation, it is a cost to firms. Moreover, when the economy is at full employment, we would not expect an environmental regulation to have an impact on overall employment because labor is being shifted from one sector to another. On the other hand, in periods of high unemployment, employment effects (both positive and negative) are possible. For example, an increase in labor demand due to regulation may result in a short-term net increase in overall employment due to the potential hiring of previously unemployed workers by the regulated sector to help meet new requirements (e.g., to install new equipment) or by the pollution control sector to produce new abatement capital. When significant numbers of workers are unemployed, the opportunity costs associated with displacing jobs in other sectors are likely to be smaller. And, in general, if a regulation imposes high costs and does not increase the demand for labor, it may lead to a decrease in employment.

To provide a partial picture of the employment consequences of this rule, EPA investigates the expected consequences for the regulated sector and for the pollution control sector. First, the analysis uses the results of Morgenstern, Pizer, and Shih (2002) to estimate the effects of the regulation on the regulated industry, the electric power industry in this case. This approach has been used by EPA previously in recent Regulatory Impact Analyses. Second, EPA uses information derived from engineering studies and projections of pollution controls from the power sector modeling to generate estimates of employment impacts to the pollution control sector.

Section 6.2 discusses the estimates of the employment consequences in the electricity sectors, using the Morgenstern, et al. approach. Section 6.3 estimates the employment consequences in the pollution control sector.

[2] It should be noted that if more labor must be used to produce a given amount of output, then this implies a decrease in labor productivity. A decrease in labor productivity will cause a short-run aggregate supply curve to shift to the left, and businesses will produce less, all other things being equal.

6.2 Employment Impacts Primarily on the Regulated Industry: Morgenstern, Pizer, and Shih (2002)

EPA examined possible employment effects within the electric utility sector using a peer-reviewed, published study that explores historical relationships between industrial employment and environmental regulations (Morgenstern, Pizer, and Shih, 2002). For context, in 2007, the electric power generation, transmission and distribution sector (NAICS 2211) had approximately 510,000 paid employees (according to the 2007 Economic Census). Estimates from Morgenstern et. al. study have been applied in recent RIAs to derive the employment effects of new regulations within the regulated industry. (See, for example, the Regulatory Impact Analyses for the proposed MATS and final CSAPR regulations). With certain qualifications, we believe that this study is relevant to this employment analysis, as it was for the MATS proposal, since the pollution control strategies or measures that form the basis of the cost inputs in the Morgenstern et al. analysis are primarily add-on or end-of-line pollution controls, in general covering more than 70% of the abatement expenditures in most years and industries analyzed as shown in Table 6-1. The analysis of control strategies presented in Chapter 3 of this RIA are composed entirely of add-on or end-of-line pollution controls. Thus, the cost inputs in the Morgenstern et al. analysis are consistent with the cost inputs that enter into this analysis of employment impacts within the regulated industry for MATS. It should be noted that the electric utility sector is less labor-intensive than the industries examined by Morgenstern et al. (2002). To this extent, it is possible that the positive employment impact estimates are high.

Table 6-1. Percent of Abatement Expenditures in Different PACE Studies from Add-On or End-of-Line Control Measures[3]

	Percent			
Industry	**1979**	**1983**	**1988**	**1991**
Pulp and Paper	84	80	61	47
Plastics	85	88	75	67
Petroleum Refining	72	57	63	61
Iron and Steel	96	93	94	92

[3] U.S. Bureau of the Census. *Pollution Abatement Costs and Expenditures*. Washington, DC: U.S. Government Printing Office, various years. The pulp and paper industry is defined by SIC 2611 & 2621, plastics by SICE 282, petroleum refining by SIC 2911, and iron and steel by SIC 332. For pulp and paper and iron and steel industries in 1983 the data is partially estimated based on non reported data due to disclosure reasons. The 1984 (1989) data for plastics (pulp and paper) is used instead of 1983 (1988) due to a lack of reported data in the original year.

Determining the direction of employment effects in the regulated industry is challenging due to competing effects. A regulation that imposes costs may, for that reason, have an adverse effect on employment, but if a regulation leads to the hiring of additional workers, it may, for that reason, have a positive effect on employment. The fundamental insight of Morgenstern, et al. is that environmental regulations can be understood as requiring regulated firms to add a new output (environmental quality) to their product mixes. Although legally compelled to satisfy this new demand, regulated firms have to finance this additional production with the proceeds of sales of their other (market) products. Satisfying this new demand requires additional inputs, including labor, and may alter the relative proportions of labor and capital used by regulated firms in their production processes. Thus, Morgenstern et al. decompose the overall effect of a regulation on employment into the following three subcomponents:

- The "Demand Effect": higher production costs raise market prices, reducing consumption (and production), thereby reducing demand for labor within the regulated industry (an unambiguously negative effect);
- The "Cost Effect": Assuming that the capital/labor ratio in the production process is held fixed, as production costs increase, plants use more of all inputs, including labor, to maintain a given level of output. For example, in order to reduce pollutant emissions while holding output levels constant, regulated firms may require additional labor (an unambiguously positive effect) ;
- The "Factor-Shift Effect": Regulated firms' production technologies may be more or less labor intensive after complying with a regulation (i.e., more/less labor is required per dollar of output). "Environmental activities may be more labor intensive than conventional production," meaning that "the amount of labor per dollar of output will rise." However, activities may, instead, be less labor intensive because "cleaner operations could involve automation and less employment, for example." (p. 416) (ambiguous effect)

Decomposing the overall employment impact of environmental regulation into three subcomponents clarifies the conceptual relationship between environmental regulation and employment in regulated sectors, and permitted Morgenstern, et al. to provide an empirical estimate of the net impact. For present purposes, the net effect is of particular interest, and is the focus of our analysis.

The demand effect is expected to have an unambiguously negative effect on employment, the cost effect to have an unambiguously positive effect on employment, and the factor-shift effect to have an ambiguous effect on employment. Without more information with

respect to the magnitudes of these three competing effects, it is not possible to predict the net employment effect in the regulated sector.

Using plant-level Census information between the years 1979 and 1991, Morgenstern et al. estimate the size of each effect for four highly polluting and regulated industries (petroleum, plastic material, pulp and paper, and steel). On average across the four industries, each additional $1 million ($1987) spending on pollution abatement results in a (statistically insignificant) net increase of 1.55 (+/- 2.24) jobs. As a result, the authors conclude that increases in pollution abatement expenditures can have positive effects on employment and do not necessarily cause economically significant employment changes. The conclusion is similar to Berman and Bui (2001), who found that increased air quality regulation in Los Angeles did not cause in large employment changes.

Ideally, the EPA would first apply the methodology of Morgenstern et al. to current pollution expenditure and market data for the regulated firms to identify the relationship between abatement costs and employment, then use this relationship to extrapolate the effect of new projected abatement costs on these firms. Unfortunately, current firm-level abatement cost and market characteristics are not available. Therefore, the EPA has used the estimated relationship from the Morgenstern et al. data to extrapolate the employment impact of the new projected abatement costs without accounting for the industry and firm differences.

Since the Morgenstern, et al. parameter estimates are expressed in jobs per million ($1987)[4] of environmental compliance expenditures, their study offers a transparent and simple way to transfer estimates for other employment analysis. For each of the three job effects outlined above, EPA used the Morgenstern et. al. four industry average parameters and standard errors along with the estimated private compliance costs to provide a range (based on the 95th percentile of results) of employment effects in the electricity sector associated with the rule. By applying these estimates to annualized cost for the final rule for the electric power sector as shown in Chapter 3 of this RIA ($9.60 billion in 2007$) , the Agency estimated each effect. The results are:

- Demand effect: −39,000 to +2,000 jobs in the directly affected sector with a central estimate of −18,000;

[4] The Morgenstern et al. analysis uses “production worker” as defined in the US Census Bureau’s Annual Survey of Manufactures (ASM) in order to define a job. This definition can be found on the Internet at http://www.census.gov/manufacturing/asm/definitions/index.html

- Cost effect: +4,000 to +21,000 jobs in the directly affected sector with a central estimate of +12,000; and
- Factor-shift effect: +200 to +27,000 jobs in the directly affected sector with a central estimate of +14,000.

EPA estimates the net employment effect to range from −15,000 to +30,000 jobs in the directly affected sector with a central estimate of +8,000.[5,6] EPA recognizes there will be other employment effects that are not considered in the Morgenstern et al. study. For example, employment in pollution control industries may increase as firms purchase more pollution control equipment and services to meet the rule's requirements. EPA does provide such an estimate of employment change later in this section in a separate analysis.

A defensible methodology for evaluating the employment impacts beyond the pollution control and regulated sectors is not yet available, though as noted before, net effects on employment are expected to be at or very close to zero for the economy overall under full employment. Attempts to estimate such effects usually rely on input-output methodologies that hold technologies and the proportion of various inputs constant over time, making them inappropriate for estimating long run impacts of regulation.

6.2.1 Limitations

The Morgenstern et al. approach to employment analysis has the advantage of carefully controlling for many possibly confounding effects in order to separate the effect of changes in regulatory costs on employment. Although the Morgenstern et al. paper provides information about the potential job effects of environmental protection programs, however, there are several caveats associated with using those estimates to analyze the final rule. First, the Morgenstern et al. estimates presented in Table 6-2 and used in EPA's analysis represent the weighted average parameter estimates for a set of manufacturing industries (pulp and paper, plastics, petroleum, and steel). Unfortunately this set of industries does not overlap directly with the electric utility sector. Second, relying on Morgenstern et al. implicitly assumes that the employment estimates derived from 1979–1991 data are still applicable. Third, the methodology used in Morgenstern et al. assumes that regulations affect plants in proportion to

[5] Since Morgenstern's analysis reports environmental expenditures in $1987, we make an inflation adjustment the IPM costs using the ratio of the annual consumer price index, U.S. city, all items reported by the U.S. Bureau of Labor Statistics: CPI_{1987}/ CPI_{2007} = (113.6/207.3) = 0.55.

[6] Net employment effect = 1.55× $9,600 million × 0.55. Given the 95% confidence interval for this effect, this estimated net result is not statistically different from zero.

their total costs. In other words, each additional dollar of regulatory burden affects a plant by an amount equal to that plant's total costs relative to the aggregate industry costs. By transferring the estimates, EPA assumes a similar distribution of regulatory costs by plant size and that the regulatory burden does not disproportionately fall on smaller or larger plants. EPA also assumes that the net employment impact can be linearly extrapolated from the abatement cost (i.e., that every million 1987 dollars generates a central estimate of 1.55 jobs). Fourth, the Morgenstern et al. analysis makes particular assumptions about the role of imports and the effect of previous regulation on plant closures. While imports are not an issue for MATS, the stringency of the current regulation is expected to result in a number of power plant closures due to early retirement of coal-fired EGU capacity in 2015, as indicated in Chapter 3 of this RIA.

Finally, the Morgenstern et.al. methodology does not examine the effects of regulation on employment in sectors related to, but outside of the regulated sector. However, it does suggest that the relationship between the employment impact in any sector and increased costs due to regulation is ambiguous.

Table 6-2. Employment Impacts Within the Regulated Industry Using Peer-Reviewed Study Estimates using Morgenstern et al. (2002)

	Estimates Using Morgenstern, et. al (2002)			
	Demand Effect	**Cost Effect**	**Factor Shift Effect**	**Net Effect**
Change in Full-Time Jobs per Million Dollars of Environmental Expenditure[a]	−3.56	2.42	2.68	1.55
Standard Error	2.03	0.83	1.35	2.24
EPA Estimate for Rule[b]	−39,000 to +2,000	+4,000 to +21,000	+200 to +27,000	−15,000 to +30,000[c]

[a] Expressed in 1987 dollars. Adjustment of dollars from 2007 to 1987 is accomplished through use of the annual Consumer Price Index – All Urban Consumers, found on the Internet at ftp://ftp.bls.gov/pub/special.requests/cpi/cpiai.txt. U.S. Department of Labor, Bureau of Labor Statistics. Washington, D.C.

[b] According to the 2007 Economic Census, the electric power generation, transmission and distribution sector (NAICS 2211) had approximately 510,000 paid employees. Both the midpoint and range for each effect are reported in the last row of the table.

[c] EPA has used this study to estimate the mean net employment impact of this rule, and provided the 95% confidence interval results to reflect the high degree of uncertainty regarding the effect on employment within the regulated industry. The confidence interval includes zero indicating we are uncertain as to the sign of the effect, but the interval itself does reveal information on the magnitude of the effect.

6.3 Employment Impacts of the MATS-Pollution Control Sector Approach by 2015[7]

Regulations set in motion new orders for pollution control equipment and services. New categories of employment have been created in the process of implementing environmental regulations. When a regulation is promulgated, one typical response of industry is to order pollution control equipment and services in order to comply with the regulation when it becomes effective, while closure of plants that choose not to comply occurs after the compliance date. With such a response by industry as a basis, this section presents estimates for short term employment needed to design, construct, and install the control equipment in the three or four year period leading up to the compliance date. Environmental regulation may increase revenue and employment in the environmental technology industry. While these increases represent gains for that industry, they are costs to the regulated industries required to install the equipment. As with any pool of labor, the gross size of the labor pool does not reflect the net impact on overall employment after adjusting for shifts in other sectors.

Regulated firms hire workers to design and build pollution controls. Once the equipment is installed, regulated firms hire workers to operate and maintain the pollution control equipment – much like they hire workers to produce more output. Of course, these firms may also reassign existing employees to do these activities. Environmental regulations also support employment in many basic industries. In addition to the increase in employment in the pollution control industry (to fill increased orders for pollution control equipment placed by the regulated sector), environmental regulations also support employment in industries that provide intermediate goods to the pollution control industry. For example, an investment in capital expenditures to reduce air pollution involves the purchase of abatement equipment. The equipment manufacturers, in turn, order steel, tanks, vessels, blowers, pumps, and chemicals to manufacture and install the equipment. A study by Bezdek, Wendling, and DiPernab (2008) found that "investments in environmental protection create jobs and displace jobs, but the net effect on employment is positive."[8] The majority of the jobs associated with added pollution controls (e.g., boilermakers, general construction workers, etc.) will provide domestic employment opportunities, but some goods and services demanded and/or provided to the pollution control industry (e.g., steel, cement, etc.) are internationally traded goods.

[7] EPA expects that the installation of retrofit control equipment in response to the requirements of this proposal will primarily take place within 3 years of the effective date of the final rule, but there may be a possibility that some installations may occur within 4 years of the effective date.

[8] Environmental protection, the economy, and jobs: National and regional analyses, Roger H. Bezdek, Robert M. Wendling and Paula DiPerna, Journal of Environmental Management Volume 86, Issue 1, January 2008, Pages 63-79.

The focus of this part of the employment analysis is on short-term employment related to the compliance actions of the affected entities. This analysis estimates the employment impacts due to the increased demand for pollution control equipment in response to MATS.[9] Results indicate that the MATS has the potential to result in a net increase of labor in these industries, driven by the high demand for new pollution controls. Overall, the results of the pollution control sector approach indicate that the MATS could support an increase of about 46,000 job-years[10] by 2015.

6.3.1 Overall Approach and Methodology for Pollution Control Sector Approach

EPA developed estimates of the potential employment changes for the Pollution Control Sector using a bottom-up engineering based methodology combined with macroeconomic data on industrial output and productivity, to estimate employment impacts. The approach relies heavily on the projections and costing analysis from the IPM model, which uses industry specific data and assumptions to derive compliance costs and energy impacts (See Chapter 3). Central to the approach are prior EPA studies on similar issues, and in particular, data and information from extensive engineering studies that the Agency has commissioned.[11] The analysis develops employment estimates by relying on IPM projections from the MATS analysis for the specific types of pollution control technologies expected to be installed to comply with the rule.[12] More specifically, the analysis includes estimates for the labor needed to design, manufacture and install the needed pollution control equipment over the 3 to 4 years leading up to compliance in 2015.

For construction labor, the labor needs are derived from an update to a 2002 EPA resource analysis for building various pollution controls (FGD – Flue Gas Desulfurization or scrubbers, SCR- selective catalytic reduction, ACI – activated carbon injection, DSI - dry sorbent injection, and FF - Fabric Filters) and are further classified into different labor categories. These categories include boilermakers, engineers and a catch-all "other" installation labor. For the inputs needed (e.g., steel), the updated 2002 resource study was used to determine the steel

[9] For more detail on methodology, approach, and assumptions, see Appendix 6A.

[10] Numbers of job years are not the same as numbers of individual jobs, but represents the amount of work that can be performed by the equivalent of one full-time individual for a year (or FTE). For example, 25 job years may be equivalent to five full-time workers for five years, twenty-five full-time workers for one year, or one full-time worker for twenty-five years.

[11] Engineering and Economic Factors Affecting the Installation of Control Technologies for Multipollutant Strategies EPA-600/R-02/073 (2002) and Engineering and Economic Factors Affecting the Installation of Control Technologies – An Update (2011).

[12] Detailed results from IPM for the MATS can be found in Chapter 3 of the RIA.

demand for each MW of additional pollution control, combined with labor productivity data from the Economic Census and BLS for relevant industries. More detail on methodology, assumptions, and data sources can be found in Appendix 6A for this RIA. Projections from IPM were used to estimate the incremental retrofit capacities projected in response to the final rule. These additional pollution controls are shown in Table 6-3, and reflect the added pollution controls needed to meet the requirements of the rule. Additional information on the power sector impacts can be found in Chapter 3 of the RIA.

Table 6-3. Increased Pollution Control Installations due to MATS, by 2015 (GW)

Retrofit Type	IPM Projected Additional Pollution Control
FGD	17
ACI	99
DSI	44
FF	102

6.3.2 Summary of Employment Estimates from Pollution Control Sector Approach

Table 6-4 shows the results of employment impacts resulting from the additional demand for the aforementioned pollution controls. The results indicate that MATS could support or create roughly 46,000 one-time job-years of increased cost of direct labor, driven by the need to design and build the pollution control retrofits.

Table 6-4. Employment Effects Using the Pollution Control Sector Approach for the MATS (in Job-Years)[13]

Employment	Incremental Employment
One-Time Employment Changes for Construction	
1. Boilermakers	20,000
2. Engineers	5,000
3. General Construction	21,000
Total	**46,000**

[13] Numbers are rounded to nearest thousand. MATS is not anticipated to result in any notable new capacity in response to the rule, and thus is not considered as part of this analysis.

6.3.3 Other Employment Impacts of MATS

In addition to the employment impacts estimated for the regulated sector and pollution control sectors, there are likely to be other employment impacts associated with MATS. These include changes resulting from labor needed to operate the needed pollution controls, increased demand for materials used in pollution control operation, shifts in demand for fuel in response to the rule, changes in employment resulting from additional coal retirements, and changes in other industries due to changes in the price of electricity and natural gas.[14] The EPA has provided estimates of some of these effects below, which are discussed in more detail in Appendix 6A. The most notable of those that the Agency is unable to estimate are the impacts on employment as a result of the increase in electricity and other energy prices in the economy. Nor is the Agency able to quantify all the employment changes in industries that support and supply the pollution control industry. Because of this inability to estimate all the important employment impacts, EPA neither sums the impacts that the Agency is able to estimate for these other employment impacts or make any inferences of whether there is a net gain or loss of employment across these categories. A summary of the other employment impacts can be found in Table 6-5, with additional detail provided in Appendix 6A.

Table 6-5. Other Employment Impacts of MATS (in Job-Years)

Employment Impacts from Increased Demand for Pollution Control Operating Inputs	
Lime (FGD)	280
Activated Carbon (ACI)	460
Trona (DSI)	3,130
Baghouse material (FF)	20
Employment Impacts from Pollution Control Operation	4,320
Employment Impacts from Retirements of Existing Coal Capacity	(2,500)
Employment Impacts from Changes in Coal Demand	(430)
Employment Impacts from Changes in Natural Gas Demand	670

Note: See Appendix 6A for more detail.

6.4 Summary of Employment Impacts

The employment approaches used by EPA rely on different analytical techniques and are applied to different industries during different time periods, and they use different units of

[14] The employment approaches used by EPA rely on different analytical techniques and are applied to different industries during different time periods, and they use different units of analysis. These estimates should not be summed because of the different metrics, length and methods of analysis. The Morgenstern estimates are used for the ongoing employment impacts for the regulated entities (the electric power sector).

analysis. These estimates should not be summed because of the different metrics, length and methods of analysis. The Morgenstern estimates are used for the ongoing employment impacts for the regulated entities (the electric power sector). The short term estimates for employment needed to design, construct, and install the control equipment in the three or four year period leading up to the compliance date are also provided. Finally some of the other types of employment impacts that will be ongoing are estimated.

In Table 6-6, we show the employment impacts of the MATS as estimated by the pollution control sector approach and by the Morgenstern approach.

Table 6-6. Estimated Employment Impact Table for the MATS

	Annual (Reoccurring)	One Time (Construction During Compliance Period)
Pollution Control Sector approach[a]	Not Applicable	46,000
Net Effect on Electric Utility Sector Employment from Morgenstern et al. approach[c]	8,000[b] -15, 000 to +30,000[d]	Not Applicable

[a]These one-time impacts on employment are estimated in terms of job-years. These employment estimates should not be summed because of the different metrics, length and methods of analysis.

[b]This estimate is not statistically different from zero.

[c]These annual or recurring employment impacts are estimated in terms of production workers as defined by the US Census Bureau's Annual Survey of Manufacturers (ASM).

[d]95% confidence interval

6.5 Potential Effect of Electricity Price Increase on Economy-Wide Production Costs

As with any input into production, the new price of electricity, reflecting the costs of MATS, will be absorbed in some fraction by industries that use electricity in their operations. Firms can respond to price changes by making changes to their processes, raising their prices, reducing production, etc. However, electricity expenditures are only a modest component of overall economic activity.

On an expenditures-weighted basis, electricity comprises only 0.75% of total production expenditures across all sectors in 2002 (BEA, 2007b, 2007c).[15] As reported in Chapter 3, the Retail Electricity Price Model forecasts a 3.1% increase in the contiguous U.S. electricity price in 2015 (see Table 3-12) as a result of MATS. Therefore, the upper estimate of the initial increase

[15] The BEA's benchmark I/O summary-level data includes information on the share of expenditures by industry spent on 135 commodity categories for 133 different sectors. These data provide a "comprehensive picture of inner workings of the economy" (Stewart et al., 2007). For more detail, see BEA 2007a and 2009.

in production costs across all sectors from direct electricity expenses is 0.023% (=[0.031 * 0.0075]*100%).[16] This 0.023% increase in average production expenditures represents a credible upper estimate of the average direct effect of higher electricity expenses because it assumes that production, consumption, and input levels do not change in the economy.[17] In reality, we know that producers and consumers can often use less electricity-intensive substitute goods and services to avoid a significant portion of these costs even in the short-run, which would mitigate these production cost increases. We also know that producers of intermediate goods and services that adjust to higher electricity prices can also make changes that lead to price adjustments for final goods and services sectors (as discussed below, indirect electricity price effects are not included in this illustrative estimate). Taking into account the fact that these numbers represent an upper estimate of initial production costs from the direct increase in electricity expenditures, EPA does not expect that increases in average production expenses from direct electricity price changes in this range are sufficient to cause significant shifts in overall economic activity outside the electricity sector and its major input markets. Note that this per unit percent cost increase does not reflect other potential economic effects of this rule. For example, the increased expenditures on pollution abatement equipment could create more demand for labor in those industries. Alternatively, as producers switch away from electricity-intensive inputs, the demand for other inputs may increase, changing the cost of production for those factors of production.

This estimate has a number of limitations. First, as mentioned above, it reflects an upper estimate on the initial change to the average production cost of goods and services from direct electricity expenses because the calculation used to estimate these changes assumes that production, consumption and input use will not change in response to higher electricity prices. Second, as mentioned above, this analysis also does not account for the effect that higher electricity prices have on the factors used by other sectors (e.g. the cost of components and other inputs). For sectors that use both electricity and other energy-intensive inputs, the effect of higher electricity prices on input prices can be important but applying the BEA data to account for the indirect effect of electricity price pass-through on factors relies even more

[16] Note that we are only performing simple calculations for upper estimate increases in per unit production costs as a direct consequence of higher electricity prices. A modeling approach would require assumptions about behavioral response to price changes, and we are assuming for this analysis that there is no behavioral response to higher electricity prices.

[17] This means that all other inputs, including capital, labor, and materials are assumed to be fixed when generating an upper estimate per unit production expense from direct electricity prices for the industries included in this analysis.

heavily on assumptions about the inability of sectors to change their factor mix in response to relative factor prices changes. Third, important differences across sectors, regions and consumer classes may be masked by the nationwide estimated average expenditure changes. However, because the Retail Electricity Price Model does not estimate price changes for different customer classes, and because the BEA data does not provide a regional decomposition of the economic accounts suitable to calculating regional upper estimates on per unit production expenses, regional and consumer class price differences cannot be calculated. Similarly, there are sectors that will have a meaningfully higher or lower maximum increase in average production expenditures within the context of the national average. Fourth, the share of electricity used may have changed since 2002. In general, electricity consumption per dollar of gross domestic product fell from 2002 through 2009, but electricity expenditures relative to gross domestic product rose slightly over this time (EIA, 2011; BEA 2011). Not accounting for this change over time in expenditures on electricity may lead to a slight underestimate of the increase in average production expenditures, averaged across the entire economy, as a result of this rule.

While there are several caveats to this approach, this calculation suggests that electricity prices under MATS are not expected to have a large impact on production costs for the economy as a whole. Initial production cost impacts of less than 0.023% from direct electricity expenditures are unlikely to lead to significant impacts on the overall economy and would fall within the normal variability range of input price variation observed by producers in the past. This is consistent with the overall history of the implementation of the Clean Air Act (Jaffe et al., 1995).

This upper estimate of average initial production cost increases from direct electricity expenditures cannot be used to estimate changes in employment as a result of the regulation, either nationally or for individual sectors. First, as noted above, these calculations do not account for the ability of the real economy to adjust to changes in price through input substitution, technological innovation, or other means. It is necessary to account for changes in production, consumption, and input use to estimate the change in total employment. Second, this approach does not account for changes in consumer and producer behavior as they adjust the quantity of goods and services supplied or demanded in all of the markets affected by the regulation. Changes in employment (both increases and decreases) in downstream sectors will reflect the balance of all of these interactions.

An evaluation of the employment impacts beyond the pollution control and regulated sectors is not yet available, though as noted before, net effects on employment are expected to

be at or very close to zero for the economy overall under full employment. In the case of this rule, labor may be a complement or a substitute to electricity in production, depending on the sector. It is also the case that environmental regulation may increase labor productivity by improving health, which may increase employment (via an increase in overall economic productivity, see the discussion in Chapter 5). Attempts to estimate such economy-wide effects by holding technologies and the proportion of various inputs constant over time are inappropriate for estimating long run impacts of regulation and an inaccurate representation of the behavior of real-world firms.

6.6 Estimating Social Cost and Economic Impacts

In the Transport Rule proposed in the summer of 2010 and in other rulemakings, EPA used a different model to estimate the social cost and economic impacts of the regulatory approach than the model applied in this RIA. That model, EPA's EMPAX, is a CGE model that dynamically cascades the cost of a regulation through the entire economy. Since that rule was proposed, a different version of EMPAX was used to estimate the social cost of the Clean Air Act in a new EPA report entitled "The Benefits and Costs of the Clean Air Act from 1990 to 2020" (EPA, 2010, herein referred to as the Section 812 report). This version of EMPAX accounts for the benefits of reducing pollution on labor productivity and on the demand for health care, which significantly influenced the model's estimates of the social cost and economic impacts of the Clean Air Act relative to an analysis using EMPAX in which these benefits-related effects were not accounted for. In December 2010, in its review of the 812 Report EPA's Science Advisory Board (SAB) found that "The inclusion of benefit-side effects (reductions in mortality, morbidity, and health-care expenditures) in a computable general equilibrium (CGE) model represents a significant step forward in benefit-cost analysis" (SAB, 2010). A description of the changes to the model and implications are described in detail in chapter 8 of the Section 812 report. EPA has determined that it needs to update the EMPAX model version used for RIAs to account for these beneficial effects of reducing pollution prior to its use in any additional regulatory analysis. The EMPAX model version used for the Section 812 report cannot be used for this rulemaking because it contains energy and economic data that are consistent with the multi-year timeframes and energy scenarios of the 812 study but not with the single target year, analysis timeframe, and energy scenario most appropriate for this current rulemaking analysis. For example, much of the energy data in the EMPAX model employed in the Section 812 report is from the Energy Information Administration's Annual Energy Outlook 2005. With these impacts of reducing pollution on labor productivity and the demand for health care now in the process of being incorporated into the model, the SAB's perspective on the desirability of accounting for these effects in the CGE analysis for the 812 study, and the typical practice by

EPA's Office of Air and Radiation of having analyses within RIAs to be consistent in design with those included in the most recent available Section 812 report, EPA will not use EMPAX for this RIA.

6.7 References

Berman, E., and L. T. M. Bui. 2001. Environmental Regulation and Labor Demand: Evidence from the South Coast Air Basin.‖ Journal of Public Economics 79(2):265-295.

Bezdek, Roger H. , Robert M. Wendling and Paula DiPerna, Environmental protection, the economy, and jobs: National and regional analyses. Journal of Environmental Management Volume 86, Issue 1**,** January 2008, Pages 63-79.

Environmental Business International (EBI), Inc., San Diego, CA. Environmental Business Journal, monthly (copyright). http://www.ebiusa.com/

Jaffe, Adam B., Steven R. Peterson, Paul R. Portney, Robert N. Stavins. (1995). "Environmental Regulation and the Competitiveness of U.S. Manufacturing: What Does the Evidence Tell Us?" *Journal of Economic Literature*, 33(1), pp. 132-163

Morgenstern, R. D., W. A. Pizer, and J. S. Shih. 2002. Jobs versus the Environment: An Industry-Level Perspective.‖ Journal of Environmental Economics and Management 43(3):412-436.

Stewart, Ricky L., Jessica Brede Stone and Mary L. Streitweisser. 2007. "U.S. Benchmark Input-Output Accounts, 2002." *Survey of Current Business.* October, 2007.

US Bureau of Economic Analysis (BEA). 2007a. *Appendix B.--Classification of Value Added and Final Uses in the 2002 Benchmark Input-Output Accounts*. Available in: *2002 Summary Tables, 2002 Benchmark Input-Output Data.* Retrieved from http://www.bea.gov/industry/io_benchmark.htm#2002data.

US Bureau of Economic Analysis (BEA). 2007b. Commodity-by-Industry Direct Requirements after Redefinitions, 2002. Available in: *2002 Summary Tables, 2002 Benchmark Input-Output Data.* Retrieved from http://www.bea.gov/industry/io_benchmark.htm#2002data.

US Bureau of Economic Analysis (BEA). 2007c. "The Use of Commodities by Industries after Redefinitions, 2002." Available in: *2002 Summary Tables, 2002 Benchmark Input-Output Data.* Retrieved from http://www.bea.gov/industry/io_benchmark.htm#2002data.

US Bureau of Economic Analysis (BEA). 2009. *Concepts and Methods of the U.S. Input-Output Accounts.* Retrieved from: http://www.bea.gov/papers/pdf/IOmanual_092906.pdf.

US Bureau of Economic Analysis (BEA). 2011. Gross Domestic Product. In National Income and Product Account Tables (Table 1.1.5). Retrieved From http://www.bea.gov//national/nipaweb/DownSS2.asp.

US Bureau of the Census. *2007 Economic Census,* Washington, DC: U.S. Government Printing Office, http://www.census.gov/econ/census07/.

US Bureau of the Census. *Pollution Abatement Costs and Expenditures* Washington, DC: U.S. Government Printing Office, various years.

US Bureau of Labor Statistics, United States Department of Labor. —Industry Labor Productivity and Cost Data Tables, Annual Percent Changes. (2010).

US Energy Information Administration (EIA), Annual Energy Review 2010. —Coal Mining Productivity By State and Mine Type.‖

US Energy Information Agency (EIA). 2011. Electricity End Use, 1949-2010. In Annual Energy Review 2010 (Table 8.9). Retrieved From http://www.eia.gov/totalenergy/data/annual/index.cfm#electricity

US Environmental Protection Agency. Engineering and Economic Factors Affecting the Installation of Control Technologies for Multipollutant Strategies, EPA-600/R-02/073 (2002).

APPENDIX 6A
EMPLOYMENT ESTIMATES OF DIRECT LABOR IN RESPONSE TO THE MERCURY AND AIR TOXICS STANDARDS IN 2015

This appendix presents the short-term employment estimates of the Mercury and Air Toxics Standards (MATS), henceforth referred to as the final MATS. The focus of the employment analysis in this study is only on the first order employment impacts related to the compliance actions of the affected coal-fired entities within the power sector.[18] It does not include the ripple effects of those impacts on the broader economy (i.e., the "multiplier" effect), nor does it include the wider economy-wide effects of the changes to energy markets (such as higher electricity prices).[19] Moreover, this study provides only a static snapshot of the impacts for 2015 and does not account for the dynamic adjustments of the affected entities as they adapt to the final MATS, such as those arising from technological innovation and learning-by-doing. This analysis is also independent of other techniques used by the U.S. EPA to estimate certain employment effects of particular regulations.

The estimates of the employment impacts are divided into several categories: job gains due to the increased demand for pollution control equipment; job losses due to retirements of coal capacity; and job shifts due to changes in demand for fuels. The various employment metrics can also be distinguished by one-time employment changes (e.g., pollution control construction), and ongoing employment changes (e.g., fuel use changes and pollution control operation or coal retirements). Results indicate that the final MATS has the potential to provide significant short-term employment opportunities, primarily driven by the high demand for new pollution control equipment. The employment gains related to the new pollution controls are likely to be tempered by some losses due to certain coal retirements, although, as discussed below, some of these workers who lose their jobs due to plant retirements could find replacement employment operating the new pollution controls at nearby units. Finally, job losses due to reduced coal demand are expected to be offset by job gains due to increased natural gas demand, resulting in very small positive (i.e., less than three hundred) net change in employment due to fuel demand changes. Overall, the preliminary results indicate that the final MATS could support a net of slightly over 46,000 one-time job-years and a net of about 6,000 ongoing job-years in 2015. These results are summarized in Table 6A-1 below.

[18] This analysis does not include potential employment effects resulting from projected impacts on oil/gas-fired units.

[19] For more detail on the economic impacts of the proposed rule, see Chapter 3 of the Regulatory Impact Analysis accompanying final MATS.

Table 6A-1: Net Employment Changes for 2015 (job-years)[a,b]

	One Time	Ongoing
New Pollution Control Equipment	46,120	8,210
Retirements of Generating Units	-	(2,500)
Changes in Fuel Use	-	240
Net Effect	**46,120**	**5,950**

[a] Total job years of labor for controls projected to be installed by 2015. MATS is not anticipated to result in any notable new capacity in response to the rule, and thus is not considered as part of this analysis.

[b] Totals may not add due to rounding.

The job-years estimated here is a snapshot of the first order employment effect of the final MATS in 2015. While there is no temporal dimension to this study, some of these jobs are likely to be spread over several years, and some will last longer. Most of the construction related labor demand, for example, is expected to provide a short-term, temporary boost to employment that could last two or three years, along with any "multiplier effects" (i.e., secondary employment supported in upstream sectors) that are not included in these job-year estimates. Most of the operational labor needs and labor shifts resulting from fuel changes are likely to be longer term. Thus, in terms of the impacts of the final MATS on economy-wide employment over time, this analysis shows there could be a significant temporary increase to employment levels starting well before 2015, which would likely recede thereafter as the construction phases for the needed pollution controls wind down. Over time, the operational jobs will continue to provide a small boost to employment over "business as usual" baseline employment levels. Note that this synopsis does not account for other employment impacts of the final MATS, such as those resulting from higher energy prices.

6A.1 Overall Approach

The estimates for the near-term employment effects of the final MATS utilize studies conducted by EPA on engineering and resource requirements for various compliance activities, such as installing pollution control equipment, switching fuels, or ceasing plant operations as they become uneconomic. Some of the information used here was obtained from a 2002 EPA engineering study for multi-pollutant control strategies.[20] This study was also the basis for the employment analysis for the proposed MATS. For the final MATS, EPA has undertaken a separate study to update the 2002 analysis in order to refine and update the Agency's understanding of the resource requirements (labor and materials) of various compliance

[20] *Engineering and Economic Factors Affecting the Installation of Control Technologies for Multi-pollutant Strategies*. EPA-600/R-02/073 (October, 2002).

activities, including estimates for newer pollution control equipment that were not included in the 2002 analysis.[21] For example, the 2002 study focused on pollution control technologies that directly address SO_2 and NO_x emissions, while the updated study also includes pollution control technologies that reduce mercury emissions and hazardous air pollutants as well. Collectively, these studies are referred to as EPA *pollution control studies* in this appendix. This employment analysis is based on information from the updated study where available, as well as data from the original 2002 study, where updated information was unavailable.

The basic approach involved using power sector projections and various energy market implications under the final MATS from modeling using EPA's data and assumptions with the Integrated Planning Model (IPM), along with data from secondary sources, to estimate the first order employment impacts for 2015. Throughout this analysis, incremental employment is measured in job-years, since there is no temporal dimension to this analysis.[22] Also, this appendix does not include estimates of total employment impacts *over time*, though there is a distinction between short-term construction related labor needs and more long-term operational labor needs for new pollution controls (though these operational labor requirements are also measured in 2015 job-years only).

6A.1.2 Employment Changes due to New Pollution Control Equipment

EPA's IPM projections for the final MATS policy case were used to estimate the incremental pollution control demand. These are shown in Table 6A-2 below:[23] Note that the capacity estimates shown in Table 6A-2 do not include EPA's projections for ESP and FGD upgrades on existing units. Because the engineering studies used in this employment analysis do not include resource estimates for these technologies, EPA chose not to analyze the employment impacts for these technologies. This exclusion is likely to understate the total employment impacts for the final MATS.

[21] *Engineering and Economic Factors Affecting the Installation of Control Technologies – An Update*. Andover Technology Partners and ICF International. October 20, 2011.

[22] A job-year is defined as the amount of work that can be performed by the equivalent of one full-time individual for one year (or FTE).

[23] According to IPM, there is some overlap between the different types of pollution control equipment demand at individual facilities. To the extent that there could be some efficiency gains at plants installing multiple controls due to economies of scale, the job estimates presented here could overstate the impacts.

Table 6A-2: Increased Pollution Control Demand due to the final MATS, 2015 (GW)

Pollution Control Type	IPM Projected Additional Pollution Control
Scrubbers (FGD) [24]	17
Activated Carbon Injection (ACI)	99
Dry Sorbent Injection (DSI)	44
Fabric Filter (FF) [25]	102

The employment impacts due to increased pollution control demand are divided into three categories, one of which is associated with the construction and installation labor requirements, while the remaining two are associated with the resources required for the ongoing operation of the pollution control equipment. The labor needed for constructing and installing these controls are for construction-related sectors, such as boilermakers, engineers, and other installation labor. The two categories of labor needs for ongoing resource requirements include employment in sectors that supply resources needed to run these pollution controls (such as reagents); and utility sector jobs to operate the control equipment. The following sections discuss the approach for each:

- For the construction labor estimates, per-unit labor needs were taken from the pollution control studies, which included man-hours required per MW for each of the control technologies listed above. The total installation labor was then sub-divided into different labor categories, such as boilermakers, engineers and a catch-all "other installation labor", using estimated shares of the different labor types in the EPA pollution control studies.

- For the longer term labor associated with operating the pollution controls, per-unit estimates of the main resources needed for the particular types of equipment (see Table 6A-3 below for a list of the resources) are also taken from EPA's pollution control

[24] In addition to the scrubber capacity shown in Table 2, EPA also projects an additional 2.6 GW of dry scrubbers on units burning waste coal and pet. coke and have existing baghouses. This capacity is not included in the table above, however, employment impacts associated with these controls are included in this appendix as discussed below.

[25] This number includes the total incremental fabric filters, as reported in Chapter 3. For the purpose of estimating the construction jobs from fabric filters this appendix uses an incremental capacity of 84 GW (i.e., incremental fabric filters that are standalone, or installed with DSI or ACI+Toxecon). To avoid double counting, the remaining 18 GW of fabric filters that represent those installed on units with dry scrubbers are excluded from the employment analysis, under the assumption that the labor estimates for dry scrubbers include resources required for both the scrubber as well as the fabric filter that goes with it.

studies. Resources needed for FF (such as the filter bags) were estimated from the incremental Variable Operation and Maintenance (VOM) costs from EPA's IPM modeling results.[26] These were then multiplied by the incremental GW for each pollution control to obtain the total (physical) quantity of resources needed. Total tonnage for each resource was then converted to dollars of increased economic output for these resources using price estimates developed by Sargent & Lundy for EPA's IPM Base Case v4.10 modeling assumptions (see notes at the end of Table 6A-3 below). Finally, the labor productivity for each particular sector was used to estimate the number of job-years these could create in 2015. Labor productivities for each sector were adjusted to account for increased worker productivity in 2015. Data for baseline worker productivity and corresponding growth rates to account for future productivities came from the Economic Census and the Bureau of Labor Statistics (BLS) estimates.[27]

- The final employment vector estimated was for the utility sector labor needed to operate these pollution control equipment. This estimate was based on the incremental Fixed Operation and Maintenance (FOM) costs from EPA's IPM modeling results, excluding costs due to retirements. Thus, this study assumes that the FOM costs are a reasonable proxy for the payroll costs that are part of the FOM costs in EPA's modeling (FOM costs are defined as the operating and maintenance costs incurred by the utility, such as those for payroll, irrespective of whether the equipment is operated). The FOM costs were then translated into employment based on estimates of payroll per worker for the power sector taken from the 2007 Economic Census and BLS estimates.[28]

[26] Because FF requirements are not endogenously determined in IPM, it required a different approach than the other controls.

[27] Total value of shipments in 2007 and total employees were taken from 2007 Economic Census, Statistics by Industry for Mining and Manufacturing sectors. The average annual growth rate of labor productivity was taken from the Bureau of Labor Statistics. Average growth rate calculated for years 1992-2007, applied to 2007 productivity to determine 2015 estimates of productivity. See the Detailed Methodology section at the end for more details about the data used for these calculations.

[28] Same sources as other productivity estimates (2007 Economic Census and BLS), however, uses employees and total payroll rather than revenue or value of shipments.

Table 6A-3: Estimated Pollution Control Resource Needs (Quantity and Prices Used)

	Amount of Resource Used	Price Used ($/unit)
Lime, FGD (tons)	1,490,391	$95[29]
Activated Carbon, ACI (tons)	184,771	$1,500
Trona, DSI (tons)	10,667,613	$150

Price Sources:

- Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies Mercury Control Cost Development Methodology FINAL", March 2011, Project 12301-009, Perrin Quarles Associates, Inc.
- Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies Dry Sorbent Injection Cost Development Methodology FINAL", August 2010, Project 12301-007, Perrin Quarles Associates, Inc.
- Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies SDA FGD Cost Development Methodology FINAL", August 2010, Project 12301-007, Perrin Quarles Associates, Inc.

6A.1.3 Results

Table 6A-4 presents the estimated employment impacts in 2015 resulting from the additional pollution controls needed to meet the final MATS requirements. According to this analysis, these investments could provide the opportunity to support about 54,500 job-years to design, construct, and operate the needed pollution control equipment in 2015. Note, some of these jobs are expected to start before, and continue beyond 2015 (such as the resource related job-years), but this analysis only provides a snapshot for 2015.

[29] For FGD this study uses the price for Lime (Dry FGD) which is significantly greater than the Limestone (Wet FGD) price. This price was used because EPA's modeling indicates most of the incremental FGD units are likely to be dry scrubbers.

Table 6A-4: Jobs Due to Pollution Control Equipment under the final MATS (Job-years in 2015)

Jobs for Construction		Incremental Employment
1. Boilermakers		20,190
2. Engineers		5,060
3. General Construction		20,870
	Sub-Total:	**46,120**
Jobs for Operation		
Jobs from Increased Operating Resource Use		
1. Lime (FGD)		280
2. Activated Carbon (ACI)		460
3. Trona (DSI)		3,130
4. Baghouse material (FF)		20
	Sub-Total:	**3,890**
Jobs for Pollution Control Operation		**4,320**
Total Labor:		**54,330**

Note: Totals may not add due to rounding

The number of job-years estimated for pollution control installation (i.e., "Jobs for Construction" in Table 6A-4 above) is driven in large part by the demand for new FFs used in EPA's modeling. As shown in Table 6A-2, up to 84 GW of new FF capacity is projected to come online in 2015 due to the final MATS that are relevant for this employment analysis. The demand for new FFs is estimated to contribute nearly 70 percent of the new employment resulting from the installation of pollution controls. Moreover, of the labor needed due to increased resource use, the Trona required for DSI is estimated to support higher number of jobs than the other resources. This is because the DSI technology requires significantly higher quantities of reagents than the other pollution controls, based on EPA's engineering estimates and pollution control studies. The second highest resource-related employment gains would likely come from the activated carbon needed in response to the final MATS.

Of the roughly 54,500 job-years estimated in Table 6A-4, about 4,300 job-years, or about 8 percent, are estimated to occur within the utility sector for labor needed to operate the pollution controls (referred to as "Jobs for Pollution Control Operation" in Table 6A-4). The rest of the labor demand will benefit the pollution control industry and other economic sectors. The increased demand for resources and chemicals needed to operate the pollution controls will result in increased employment in sectors such as mining, chemicals, and other

manufacturing sectors. The majority of these first order employment effects, however, are likely to benefit construction-related sectors, such as construction, boilermaker, heavy engineering, and other heavy construction sectors, resulting from the construction and installation of the new pollution controls at affected sources throughout the country.

6A.1.3.1 Employment Changes due to Coal Retirements

Employment changes due to incremental coal plant retirements were estimated by first identifying the retiring coal units[30] from EPA's modeling results (for the base and the final MATS policy cases). EPA projects roughly 4.7 GW of additional coal retirements by 2015 with the final MATS in place.[31]

In order to convert the retired coal capacity into potential employment losses, it was assumed that changes in the operating costs for the retired coal units can be used as a proxy for payroll expenditures and the lost economic output due to coal retirements. Thus, the changes in the FOM costs for these particular retiring units were derived using EPA's IPM modeling results, and converted to lost jobs using data from the Economic Census and BLS output/worker estimates for the utility sector.[32] Employment losses due to plant retirements will not only affect those that are directly working at the plant (i.e., plant operators), but would also affect administrative and other "back-office" workers for those utilities and their support organizations. This appendix assumes that the FOM costs related to retiring plants are a good proxy for these types of job losses.

Table 6A-5: Annual Job Losses due to Coal Capacity Retirements for 2015

FOM Decrease from Retirements (million)	$288
Workers Per Million$ in payroll	8.7
Workers lost due to retirements (job-year):	**2,500**

[30] Oil and gas steam unit emissions requirements, and potential retirements, were not directly included in EPA's IPM modeling under the MATS policy scenario. An analysis of these units was conducted separately, and to the extent that there may be some retirements of oil and gas units, then the estimates of potential job losses due to retirements provided here will understate the employment losses.

[31] Retirement estimates are based on IPM System Summary Reports from EPA's modeling runs. Where applicable, data from IPM parsed outputs were adjusted to account for partial retirements reported in the parsed outputs.

[32] The same specific sources as cited before, however, used workers and total payroll.

Results indicate there could potentially be about 2,500 job losses (measured in job-years for 2015, but any *net* job losses under this category are likely to be permanent), due to coal retirements. However, two mitigating factors could reduce the negative employment impacts due to retirements. First, many of the retiring units are at plants that are likely to have other units operating under the policy scenario. In such cases, some of the excess labor pool at the retiring units could well be absorbed at other units within the same firm.

Second, as Table 6A-4 indicates, utilities are expected to have the need to fill about 4,300 additional job slots to operate the pollution controls needed to meet the requirements of the final MATS. If workers with experience at existing coal facilities become available through plant retirements, some of these workers could be absorbed in operating these new pollution controls.

6A.1.3.2 Employment Changes due to Changes in Fuel Use

Employment impacts due to projected fuel use changes (coal and natural gas production shifts) were estimated using EPA's modeling results. First, employment losses due to reductions in coal demand were estimated using an approach similar to EPA's coal employment analyses under Title IV of the Clean Air Act Amendments.[33] Using this approach, EPA's projected coal demand changes (in short tons) for various coal supplying regions were converted to job-years using EIA data on regional coal mining productivity (in short tons per employee hour), using 2008 labor productivity estimates.[34,35]

Results of the coal employment impacts of the final MATS are presented in Table 6A-6 below.

[33] Impacts of the Acid Rain Program on Coal Industry Employment. EPA 430-R-01-002 March 2001.

[34] From US Energy Information Administration (EIA) Annual Energy Review, Coal Mining Productivity Data. Used 2008.

[35] Unlike the labor productivity estimates for various equipment resources which were forecasted to 2015 using BLS average growth rates, we used the most recent historical productivity estimates for fuel sectors. In general, labor productivity for the fuel sectors (both coal and natural gas) showed a significantly higher degree of variability in recent years than the manufacturing sectors, which would have introduced a high degree of uncertainty in forecasting productivity growth rates for future years.

Table 6A-6: Annual Employment Impacts Due To Changes in Coal Use for 2015

Coal by Region	Change in Coal Demand (MM Tons)	Labor Productivity	Job-year Change
Appalachia	(11.8)	2.91	(1,950)
Interior	19.9	4.81	1,990
West	(17.3)	19.91	(420)
Waste Coal	(0.7)	5.96	(60)
Net Total	**(9.9)**	--	**(430)**

Notes: Used US national coal productivity for waste coal
Totals may not add due to rounding

For natural gas production, labor productivity per unit of natural gas was unavailable, unlike coal labor productivities used above. Most secondary data sources (such as Census and EIA) provide estimates for the combined oil and gas extraction sector. This appendix thus uses an adjusted labor productivity estimate for the combined oil and gas sector that accounts for the relative contributions of oil and natural gas in the total sector output (in terms of the value of energy output in MMBtu). This estimate of labor productivity is then used with the incremental natural gas demand for the final MATS to estimate the job-years for 2015.

Table 6A-7: Annual Employment Impact due to Changes in Fuel Use (2015)

Fuel Type	Employment
Coal Job Years Lost	**(430)**
Natural Gas	
Incremental Natural Gas Use (MMBtu)	175,786,505
Labor Productivity (MMBtu/job-year)	261,840
Job-years gained	**670**
Net Employment Effects of Fuel use changes	**240**

Note: Totals may not add due to rounding

Thus, about 430 job losses in the coal mining sector are likely to be offset by about 670 job gains in the natural gas production related sectors, for a net effect of about 240 job-year gains due to the changes in fuel use. The changes in coal mining employment is driven by a significant increase in demand for Interior coal which leads to about the same amount of job gains as is lost due to the decreased demand for Appalachian coal (see Table 6A-6 above). This,

coupled with the fact that there is likely to be some job gains due to increased demand for natural gas, results in a small net job gain due to fuel use changes for the final MATS.

6A.2 Results Summary

Overall, the final MATS is expected to provide an increase to short-term employment resulting from substantial investments in new pollution control equipment. For 2015, the results indicate the final MATS could support or create around 46,000 job-years driven by the need to design and construct the needed equipment. While there could be some employment losses due to coal retirements that will likely have a negative effect on some utilities and the coal mining sector, employment gains in pollution control operation activities and the natural gas sector are likely to offset some of those losses. As previously discussed, this assessment does not account for the long-run economy-wide effects of the final MATS.

6A.3 Detailed Methodology

This section provides more details on the data and approaches used to estimate the employment impacts discussed above. The section also details the sources for individual data elements.

6A.3.1 Pollution Control Equipment Labor

6A.3.1.1 Installation Labor

Table 6A-8: Installation Labor Requirement[36]

Pollution Control Type	Incremental GW Installed	Man-hours/MW	Boilermakers (%)	Engineers (%)	Others (%)
FGD [37]	17	1730	40	20	40
ACI	99	10	50	17	33
DSI	44	55	50	17	33

[36] See Chapter 3 for more detail.

[37] EPA also projects 2.6 GW of dry scrubbers on waste coal and pet. coke units with existing baghouses, which are not shown in this table. Employment impacts from these units, however, are included in the pollution control construction figures, calculated using the capital cost for these controls ($220.7 MM) and estimated manhours/$ capital cost (0.00598) developed from the same example dry scrubbers used to find the manhours/MW of capacity.

FF [38]	102	780	45	7	48

Source: Engineering and Economic Factors affecting the Installation of Control Technologies – An Update, Andover Technology Partners, 2011

Installation labor is estimated by using the incremental GW installed for each pollution control type from EPA's modeling using IPM. This was then converted into total man-hours needed for installation using estimates of man-hours/MW primarily from EPA's 2011 update on pollution control technology,. Total man-hours for each pollution control type were then converted into man-years assuming 2,080 working hours per year.

Total man-years for each pollution control type were then broken down into various sectors using the percentages, shown in Table 6A-8. These percentages were estimated from the 2002 study, updated from the 2011 study, where applicable.

6A.3.1.2 Operating Resource Labor

Table 6A-9: Resources Needed for Operation

Pollution Control Type	Incremental Total GW	Resource (Units in parenthesis)	Usage Estimates	Price ($/unit)	Industry Assumed for Productivity Calculations	Productivity*
FGD	17	Lime (Tons/MWh)	0.013	95	Lime Manufacturing	2.0
ACI	99	Activated Carbon (Tons/MWh)	0.00025	1,500	Other Chemical Product Manufacturing	1.6
DSI	44	Trona (Tons/MWh)	0.033	150	Potash Soda and Borate Mineral Mining	2.0
FF	102	Bag-house Resources	*Resource Labor determined Using VOM cost for FFs		Plastics Material and Resin Manufacturing	0.6

**Workers/$Million in Output, Forecasted to 2015*

Sources: Usage:

- Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies Mercury Control Cost Development Methodology FINAL", March 2011, Project 12301-009, Perrin Quarles Associates, Inc.
- Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies Dry Sorbent Injection Cost Development Methodology FINAL", August 2010, Project 12301-007, Perrin Quarles Associates, Inc.
- Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies SDA FGD Cost

[38] This number includes the total incremental fabric filters. For the purpose of estimating the construction jobs from fabric filters, this analysis uses an incremental capacity of 84 GW. The remaining 18 GW represent fabric filters installed with dry FGD units, which are excluded because the labor estimates for dry scrubbers includes the labor for fabric filters that is installed in conjunction.

Labor related to resources used in operating pollution control equipment was estimated using the total incremental GW of pollution control capacity which was first converted to total MWh of incremental capacity assuming 85 percent capacity factor. For each pollution control type, the next step involved choosing the primary operating resource. This approach is consistent with prior EPA's analyses on similar topics. The next step involved estimating the resource needs by each control type, generally in tons of material using the resource usage estimates as shown in Table 6A-9. Using the total usage for each pollution control input (in tons) and associated average prices, total expenditure by each resource type was then calculated. This total expenditure was then converted to labor using workers per $Million in total output for the industry associated with producing each respective input material.[39]

6A.3.1.3 Operating Labor

Table 6A-10: Operating Labor Assumptions

Incremental FOM from IPM Parsed ($ Billion)	2.20
FF and other Capital Costs Included in FOM ($ Billion)	1.70
Remaining FOM used to find O&M Labor ($ Million)	496.5
Productivity*	8.7

**Workers per $Million in Payroll for Electricity Generating Sector, Forecast to 2015*
Sources: Productivity from 2007 Economic Census and Growth Rate from BLS.

Labor requirement to operate the controls is estimated for all equipment types combined, using the incremental FOM costs from IPM. The IPM incremental FOM cost estimate included capital costs for fabric filters and scrubber improvement costs, which were first subtracted to obtain the true FOM costs ($496.5 million). Resulting FOM cost estimate was then converted to labor needs using the workers/$ Million in total payroll for the Electric Generating Sector.

6A.3.2 Retirement Labor

Table 6A-11: Inputs to Labor from Retirements

[39] Fabric filters follow a different pattern. Instead of a resource usage estimate, we used the VOM cost for FFs and converted this to jobs using the workers per million dollars output for the relevant manufacturing industry sector.

Capacity of Incremental Retirements in SSR (MW)	4.7
O&M Decrease scaled to SSR Retirements ($MM) (To account for Partial Retirements)	288.2
Workers Per $Million in payroll, forecast to 2015	8.7

Sources: Productivity from 2007 Economic Census and Growth Rate from BLS.

Retirement labor was estimated by first identifying the retiring units from EPA's modeling using IPM parsed outputs (using incremental retirements in the policy case). The next step involved estimating the capacity of incremental retirements as well as the change in the FOM costs due to these retirements. Because of the discrepancies between partial retirements in EPA's parsed outputs and System Summary Reports (SSR), FOM costs were scaled proportionately to reflect the lower SSR-based estimates, as shown in Table 6A-11 above. FOM cost decreases were then converted to job-years lost due to retirements using workers per $Million in payroll.

6A.3.3 Fuel Use Labor

Table 6A-12: Inputs to Labor for Fuel Use

Coal by Region	2015 Incremental Fuel USE (Tons)	2008 Short Tons/Employee hour
Appalachia	-11,770,000	2.9
Interior	19,870,000	4.8
West	-17,260,000	19.9
Waste Coal	-700,000	6.0
Natural Gas		
EIA Total Natural Gas Production 2007 (TCF)		24.664
EIA Total Crude Oil Production 2007 (Barrels)		1,848,450,000
EIA Natural Gas Heat Content 2007 (Btu/cf)		1,027
EIA Petroleum Heat Content (MMBtu/Barrel)		6.151
Total Crude Oil and Natural Gas Production (MMBtu)		36,699,744,000
Economic Census 2007 Oil and Gas Extraction Employees		140,160
MMBtu per Man-year for Oil and Gas Extraction		261,842

Incremental Natural Gas from IPM (TCF)	0.171
Incremental Natural Gas from IPM (Converted to MMBtu)	175,786,505

**Workers per $Million in Payroll for Electricity Generating Sector, Forecast to 2015*
Note: Heat Contents from EIA are assumed to be for fuels used in Electric Power Sector
Sources: Short Tons per hour from US EIA, Coal Industry Annual. Total Production from 2009 EIA Annual Energy Review. Heat Contents from EIA, Heat Content of Natural Gas Consumed and 2009 Annual Energy Review. Employment Data from 2007 Economic Census.

Fuel use related employment impacts were estimated by using IPM results for incremental changes in coal and natural gas use (policy case over the base case). For coal, estimates of coal use in tons by region from IPM were used in conjunction with labor productivity estimates from the EIA for each region (in short tons/ employee hour), to calculate the change in job-hours needed. These were then converted to job-years, assuming 2,080 working hours per year. As discussed above, because of the high variability in coal mining labor productivity in recent years, no attempt was made to forecast coal (and natural gas, for consistency) productivities, instead the most recent historical estimates were used in this appendix (which was the 2008 labor productivity for coal).

For natural gas, the first step was estimating labor productivity since such information was not available directly from any reliable source. EIA production data from the Annual Energy Review for natural gas and crude oil (in TCF and barrels, respectively), along with EIA heat content estimates were used to find total crude oil and natural gas production in MMBtu for 2007. Labor productivity in MMBtu per job-year for the Oil and Gas Extraction sector was then estimated using data from the Census on oil and gas extraction employment. Then, the incremental natural gas demand from EPA's IPM modeling results (in TCF) was converted to MMBtu of natural gas demand using EIA data on natural gas heat content. This was then used with the labor productivity estimated above to calculate the total job-years needed for increased natural gas demand for the final MATS.

6A.4 References

Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies Mercury Control Cost Development Methodology FINAL", March 2011, Project 12301-009, Perrin Quarles Associates, Inc.

Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies Dry Sorbent Injection Cost Development Methodology FINAL", August 2010, Project 12301-007, Perrin Quarles Associates, Inc.

Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies SDA FGD Cost Development Methodology FINAL", August 2010, Project 12301-007, Perrin Quarles Associates, Inc.

United States Department of Labor. Bureau of Labor Statistics. Industry Labor Productivity and Cost Data Tables, Annual Percent Changes. 2010.

US Census Bureau. 2007 and 2002 Economic Census, 2000 Annual Survey of Manufacturers, Manufacturing and Mining: Detailed Statistics by Industry for the US and Utilities: Summary Statistics for the US.

US Energy Information Administration (EIA). Annual Energy Review 2009, Assorted Data for Coal, Natural Gas, and Petroleum. 2009.

US Energy Information Administration (EIA). Natural Gas 2009, Heat Content of Natural Gas Consumed 2009.

US EPA. Clear Skies Act, Technical Package, Section D. "Projected Impacts on Generation and Fuel Use". 2003.

US EPA. Clean Air Interstate Rule, Technical Support Document. "Boilermaker Labor Analysis and Installation Timing." OAR-2003-0053 (March, 2005).

US EPA. "Impacts of the Acid Rain Program on Coal Industry Employment." EPA 430-R-01-002 (March, 2001).

US EPA, Office of Research and Development. "Engineering and Economic Factors Affecting the Installation of Control Technologies for Multi-pollutant Strategies." EPA-600/R-02/073 (October, 2002).

US EPA, Engineering and Economic Factors Affecting the Installation of Control Technologies: An Update. Prepared by Andover Technology Partners and ICF International. October, 2011.

CHAPTER 7
STATUTORY AND EXECUTIVE ORDER ANALYSES

7.1 Introduction

This chapter presents discussion and analyses relating to Executive Orders and statutory requirements relevant for the final Mercury and Air Toxics Standards (MATS). We discuss the analysis conducted to comply with Executive Order (EO) 12866 and the Paperwork Reduction Act (PRA) as well as potential impacts to affected small entities required by the Regulatory Flexibility Act (RFA), as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA). We also describe the analysis conducted to meet the requirements of the Unfunded Mandates Reform Act of 1995 (UMRA) assessing the impact of the final rule on state, local and tribal governments and the private sector. In addition, we address the requirements of EO 13132: Federalism; EO 13175: Consultation and Coordination with Indian Tribal Governments; EO 13045: Protection of Children from Environmental Health and Safety Risks; EO 13211: Actions that Significantly Affect Energy Supply, Distribution, or Use; the National Technology Transfer and Advancement Act; EO 12898: Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations; and the Congressional Review Act.

7.2 Executive Order 12866: Regulatory Planning and Review and Executive Order 13563, Improving Regulation and Regulatory Review

Under Executive Order (EO) 12866 (58 FR 51,735, October 4, 1993), this action is an "economically significant regulatory action" because it is likely to have an annual effect on the economy of $100 million or more or adversely affect in a material way the economy, a sector of the economy, productivity, competition, jobs, the environment, public health or safety, or state, local, or tribal governments or communities. Accordingly, the EPA submitted this action to the Office of Management and Budget (OMB) for review under Executive Orders 12866 and 13563 any changes in response to OMB recommendations have been documented in the docket for this action. In addition, EPA prepared this Regulatory Impact Analysis (RIA) of the potential costs and benefits associated with this action.

When estimating the human health benefits and compliance costs detailed in this RIA, the EPA applied methods and assumptions consistent with the state-of-the-science for human health impact assessment, economics and air quality analysis. The EPA applied its best professional judgment in performing this analysis and believes that these estimates provide a reasonable indication of the expected benefits and costs to the nation of this rulemaking. This RIA describes in detail the empirical basis for the EPA's assumptions and characterizes the

various sources of uncertainties affecting the estimates below. In doing what is laid out above in this paragraph, the EPA adheres to EO 13563, "Improving Regulation and Regulatory Review," (76 FR 3821; January 18, 2011), which is a supplement to EO 12866.

In addition to estimating costs and benefits, EO 13563 focuses on the importance of a "regulatory system [that]...promote[s] predictability and reduce[s] uncertainty" and that "identify[ies] and use[s] the best, most innovative, and least burdensome tools for achieving regulatory ends." In addition, EO 13563 states that "[i]n developing regulatory actions and identifying appropriate approaches, each agency shall attempt to promote such coordination, simplification, and harmonization. Each agency shall also seek to identify, as appropriate, means to achieve regulatory goals that are designed to promote innovation." We recognize that the utility sector faces a variety of requirements, including ones under CAA section 110(a)(2)(D) dealing with the interstate transport of emissions contributing to ozone and PM air quality problems, with coal combustion wastes, and with the implementation of CWA section 316(b). In developing today's final rule, the EPA recognizes that it needs to approach these rulemakings in ways that allow the industry to make practical investment decisions that minimize costs in complying with all of the final rules, while still achieving the fundamentally important environmental and public health benefits that underlie the rulemakings.

A summary of the monetized costs, benefits, and net benefits for the final rule at discount rates of 3 percent and 7 percent is the Executive Summary and Chapter 8 of this RIA.

7.3 Paperwork Reduction Act

The information collection requirements in this rule have been submitted for approval to the Office of Management and Budget (OMB) under the Paperwork Reduction Act, 44 U.S.C. 3501 et seq. The information collection requirements are not enforceable until OMB approves them.

The information requirements are based on notification, recordkeeping, and reporting requirements in the NESHAP General Provisions (40 CFR part 63, subpart A), which are mandatory for all owners and operators subject to national emission standards. These recordkeeping and reporting requirements are specifically authorized by CAA section 114 (42 U.S.C. 7414). All information submitted to the EPA pursuant to the recordkeeping and reporting requirements for which a claim of confidentiality is made is safeguarded according to Agency policies set forth in 40 CFR Part 2, subpart B. This final rule requires maintenance inspections of the control devices but would not require any notifications or reports beyond those required by

the General Provisions. The recordkeeping provisions require only the specific information needed to determine compliance.

The annual monitoring, reporting, and recordkeeping burden for this collection (averaged over the first 3 years after the effective date of the standards) is estimated to be $158 million. This includes 698,907 labor hours per year at a total labor cost of $49 million per year, and total non-labor capital costs of $108 million per year. This estimate includes initial and annual performance tests, semiannual excess emission reports, developing a monitoring plan, notifications, and recordkeeping. Initial capital expenses to purchase monitoring equipment for affected units are estimated at a cost of $231 million. This includes 504,629 labor hours at a total labor cost of $35 million for planning, selection, purchase, installation, configuration, and certification of the new systems and total non-labor capital costs of $196 million. All burden estimates are in 2007 dollars and represent the most cost effective monitoring approach for affected facilities.

An Agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for the EPA's regulations are listed in 40 CFR Part 9. When this ICR is approved by OMB, the Agency will publish a technical amendment to 40 CFR Part 9 in the Federal Register to display the OMB control number for the approved information collection requirements contained in this final rule.

7.4 Final Regulatory Flexibility Analysis

The Regulatory Flexibility Act (RFA) generally requires an agency to prepare a regulatory flexibility analysis of any rule subject to notice and comment rulemaking requirements under the Administrative Procedure Act or any other statute unless the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities. Small entities include small businesses, small organizations, and small governmental jurisdictions.

For purposes of assessing the impacts of today's rule on small entities, small entity is defined as: (1) a small business that is an electric utility producing 4 billion kilowatt-hours or less as defined by NAICS codes 221122 (fossil fuel-fired electric utility steam generating units) and 921150 (fossil fuel-fired electric utility steam generating units in Indian country); (2) a small governmental jurisdiction that is a government of a city, county, town, school district or special district with a population of less than 50,000; and (3) a small organization that is any not-for-profit enterprise which is independently owned and operated and is not dominant in its field.

Pursuant to section 603 of the RFA, the EPA prepared an initial regulatory flexibility analysis (IRFA) for the proposed rule and convened a Small Business Advocacy Review Panel to obtain advice and recommendations of representatives of the regulated small entities. A detailed discussion of the Panel's advice and recommendations is found in the Panel Report (EPA-HQ-OAR-2009-0234-2921). A summary of the Panel's recommendations is presented at 76 FR 24975.

As required by section 604 of the RFA, we also prepared a final regulatory flexibility analysis (FRFA) for today's final rule. The FRFA addresses the issues raised by public comments on the IRFA, which was part of the proposal of this rule. The FRFA is summarized below and in the preamble.

7.4.1 Reasons Why Action Is Being Taken

In 2000, the EPA made a finding that it was appropriate and necessary to regulate coal- and oil-fired electric utility steam generating units (EGUs) under Clean Air Act (CAA) section 112 and listed EGUs pursuant to CAA section 112(c). On March 29, 2005 (70 FR 15,994), the EPA published a final rule (2005 Action) that removed EGUs from the list of sources for which regulation under CAA section 112 was required. That rule was published in conjunction with a rule requiring reductions in emissions of mercury from EGUs pursuant to CAA section 111, i.e., CAMR, May 18, 2005, 70 FR 28606). The Section 112(n) Revision Rule was vacated on February 8, 2008, by the U.S. Court of Appeals for the District of Columbia Circuit. As a result of that vacatur, CAMR was also vacated and EGUs remain on the list of sources that must be regulated under CAA section 112. This action provides the EPA's final NESHAP for EGUs.

7.4.2 Statement of Objectives and Legal Basis for Final Rules

MATS will protect air quality and promote public health by reducing emissions of HAP. In the December 2000 regulatory determination, the EPA made a finding that it was appropriate and necessary to regulate EGUs under CAA section 112. The February 2008 vacatur of the 2005 Action reverted the status the rule to the December 2000 regulatory determination. CAA section 112(n)(1)(A) and the 2000 determination do not differentiate between EGUs located at major versus area sources of HAP. Thus, the NESHAP for EGUs will regulate units at both major and area sources. Major sources of HAP are those that have the potential to emit at least 10 tons per year (tpy) of any one HAP or at least 25 tpy of any combination of HAP. Area sources are any stationary sources of HAP that are not major sources.

7.4.3 Summary of Issues Raised during the Public Comment Process on the IRFA

The EPA received a number of comments related to the Regulatory Flexibility Act during the public comment process. A consolidated version of the comments received is reproduced below. These comments can also be found in their entirety in the response to comment document in the docket.

Comment: Several commenters expressed concern with the SBAR panel. Some believe Small Entity Representatives (SERs) were not provided with regulatory alternatives including descriptions of significant regulatory options, differing timetables, or simplifications of compliance and reporting requirements, and subsequently were not presented with an opportunity to respond. One commenter believes the EPA's formal SBAR Panel notification and subsequent information provided by the EPA to the Panel did not include information on the potential impacts of the rule as required by section 609(b)(1). Additional commenters suggested that the EPA's rulemaking schedule put pressure on the SBAR Panel through the abbreviated preparation for the Panel. Commenters also expressed concerns that the EPA did not provide participants more than cursory background information on which to base their comments. One commenter stated that the EPA did not provide deliberative materials, including draft proposed rules or discussions of regulatory alternatives, to the SBAR Panel members. One commenter stated the SBAR Panel Report does not meet the statutory obligation to recommend less burdensome alternatives. The commenter suggested the EPA panel members declined to make recommendations that went further than consideration or investigation of broad regulatory alternatives, with the exception of those recommendations in which the EPA rejected alternative interpretations of the CAA section 112 and relevant court cases. Two stated that the EPA did not respond to the concerns of the small business community, the SBA, or OMB, ignoring concerns expressed by the SER panelists. One commenter believes the EPA failed to convene required meetings and hearings with affected parties as required by law for small business entities. One commenter stated that the SERs' input is very important because more than 90 percent of public power utility systems meet the definition and qualify as small businesses under the SBREFA.

Response: The RFA requires that SBAR Panels collect advice and recommendations from SERs on the issues related to:

- the number and description of the small entities to which the proposed rule will apply;

- the projected reporting, record keeping and other compliance requirements of the proposed rule;
- duplication, overlap or conflict between the proposed rule and other federal rules; and
- alternatives to the proposed rule that accomplish the stated statutory objectives and minimize any significant economic impact on small entities.

The RFA does not require a covered agency to create or assemble information for SERs or for the government panel members. While section 609(b)(4) requires that the government Panel members review any material the covered agency has prepared in connection with the RFA, the law does not prescribe the materials to be reviewed. The EPA's policy, as reflected in its RFA guidance, is to provide as much information as possible, given time and resource constraints, to enable an informed Panel discussion. In this rulemaking, because of a court-ordered deadline, the EPA was unable to hold a pre-panel meeting but still provided SERs with the information available at the time, held a standard Panel Outreach meeting to collect verbal advice and recommendations from SERs, and provided the standard 14-day written comment period to SERs. The EPA received substantial input from the SERs, and the Panel report describes recommendations made by the Panel on measures the Administrator should consider that would minimize the economic impact of the proposed rule on small entities. The EPA complied with the RFA.

Comment: One commenter requested that the EPA work with utilities such that new regulations are as flexible and cost efficient as possible.

Response: In developing the final rule, the EPA has considered all information provided prior to, as well as in response to, the proposed rule. The EPA has endeavored to make the final regulations flexible and cost efficient while adhering to the requirements of the CAA.

Comment: One commenter was concerned about the ability of small entities or nonprofit utilities such as those owned and/or operated by rural electric co-op utilities, and municipal utilities to comply with the proposed standards within three years. The commenter believes that the EPA disregarded the SER panelists who explained that under these current economic conditions they have constraints on their ability to raise capital for the construction of control projects and to acquire the necessary resources in order to meet a three-year compliance deadline. Two commenters expressed concern that smaller utilities and those in rural areas will be unable to get vendors to respond to their requests for proposals, because they will be able to make more money serving larger utilities.

Response: The preamble to the proposed rule (76FR 25054, May 3, 2011) provides a detailed discussion of how the EPA determined compliance times for the proposed (and final) rule. The EPA has provided pursuant to section 112(i)(3)(A) the maximum three-year period for sources to come into compliance. Sources may also seek a one-year extension of the compliance period from their title V permitting authority if the source needs that time to install controls. CAA section 112(i)(3)(B). If the situation described by commenters (i.e., where small entities or nonprofit utilities constraints on ability to raise capital for construction of control projects and to acquire necessary resources) results in the source needing additional time to install controls, they would be in a position to request the one-year extension. The EPA discusses in more detail in section VII of this preamble how the agency plans to address those units that are still unable to comply within the statutorily mandated period.

Comment: Several commenters believe the EPA did not adequately consider the disproportionately large impact on smaller generating units. The commenters note the diseconomies in scale for pollution controls for such units. One commenter noted the rule will create a more serious compliance hurdle for small communities that depend on coal-fired generation to meet their base load demand. The commenter notes that by not subcategorizing units, the EPA is dictating a fuel switch due to the disproportionately high cost on small communities. The other commenter believes the MACT and NSPS standards are unachievable by going too far without really considering the impacts on small municipal units, as public powers is critical to communities, jobs, economic viability and electric reliability. A generating and transmissions electric cooperative which qualifies as a small entity believes the rule will ultimately result in increased electricity costs to its members and will negatively impact the economies of the primarily rural areas that they serve. Another commenter believes there is no legal or factual basis for creating subcategories or weaker standards for state, tribal, or municipal governments or small entities that are operating obsolete units, particularly given the current market situation and applicable equitable factors. The commenter suggests both the EPA's and SBA's analyses focus exclusively on the effects on entities causing HAP emissions and primarily on those operating obsolete EGUs, and fail to consider either impacts on downwind businesses and governments or the positive impacts on small entities and governments owning and operating competing, clean and modern EGUs.

Response: The EPA disagrees with the commenters' belief that the impacts on smaller generating units were not adequately considered when developing the rule. The EPA determined the number of potentially impacted small entities and assessed the potential impact of the proposed action on small entities, including municipal units. A similar assessment

was conducted in support of the final action. Specifically, the EPA estimated the incremental net annualized compliance cost, which is a function of the change in capital and operating costs, fuel costs, and change in revenue. The projected compliance cost was considered relative to the projected revenue from generation. Thus, the EPA's analysis accounts not only for the additional costs these entities face resulting from compliance, but also the impact of higher electricity prices. The EPA evaluated suggestions from SERs, including subcategorization recommendations. In the preamble to the proposed rule, the EPA explains that, normally, any basis for subcategorizing must be related to an effect on emissions, rather than some difference which does not affect emissions performance. The EPA does not see a distinction between emissions from smaller generating units versus larger units. The EPA acknowledges the comment that there is no legal or factual basis for creating subcategories or weaker standards for state, tribal, or municipal governments or small entities that are operating obsolete units.

Comment: One commenter notes that the EPA recognizes LEEs in the rule such that they should receive less onerous monitoring requirements; however, the EPA does not recognize that small and LEEs also need and merit more flexible and achievable pollution control requirements. The commenter notes that the capital costs for emissions control at small utility units is disproportionately high due to inefficiencies in Hg removal, space constraints for control technology retrofits, and the fact that small units have fewer rate base customers across which to spread these costs. The commenter cites the Michigan Department of Environmental Quality report titled “Michigan’s Mercury Electric Utility Workgroup, Final Report on Mercury Emissions from Coal-Fired Power Plants,” (June 2005). The commenter notes that the EPA has addressed such concerns previously, citing the RIA for the 1997 8-hour ozone standard. The commenter also suggests smaller utility systems generally have less capital to invest in pollution control than larger, investor-owned systems, due to statutory inability to borrow from the private capital markets, statutory debt ceilings, limited bonding capacity, borrowing limitations related to fiscal strain posed by other, non-environmental factors, and other limitations.

Response: The EPA acknowledges that the rule contains reduced monitoring requirements for existing units that qualify as LEEs. Although the EPA does not believe that reduced pollution control requirements are warranted for LEEs, including small entity LEEs, we believe that flexible and achievable pollution control requirements are promoted through alternative standards, alternative compliance options, and emissions averaging as a means of demonstrating compliance with the standards for existing EGUs.

Comment: One commenter believes that the EPA should develop more limited monitoring requirements for small EGUs. The commenter notes small entities do not possess the monetary resources, manpower, or technical expertise needed to operate cutting-edge monitoring techniques such as Hg CEMS and PM CEMs. The commenter notes the EPA could have identified monitoring alternatives to the SER panel for consideration.

Response: The EPA provided monitoring alternatives to using PM CEMS, HCl CEMS, and Hg CEMS in its proposed standards and in this final rule. The continuous compliance alternatives are available to all affected sources, including small entities. As alternatives to the use of PM CEMS and HCl CEMS, sources are allowed to conduct additional performance testing. Sorbent trap monitoring is allowed in lieu of Hg CEMS.

Comment: Several commenters believe the EPA has not sufficiently complied with the requirements of the RFA or adequately considered the impact this rulemaking would have on small entities. One commenter believes the EPA has not engaged in meaningful outreach and consultation with small entities and therefore recommends that the EPA seek to revise the court-ordered deadlines to which this rulemaking is subject, re-convene the SBAR panel, prepare a new initial regulatory flexibility analysis (IRFA), and issue it for additional public comment prior to final rulemaking. The commenter believes the IRFA does not sufficiently consider impacts on small entities as identified in the SBAR Panel Report. The commenter believes it is not apparent that the EPA considered the recommendations of the Panel. The commenter believes the description of significant alternatives in the IRFA is almost entirely quoted from the SBAR Panel Report, which the commenter does not believe is an adequate substitute for the EPA's own analysis of alternatives. The commenter also notes the EPA does not discuss the potential impacts of its decisions on small entities or the impacts of possible flexibilities. Where the EPA does consider regulatory alternatives in principle, the commenter believes it does not provide sufficient support for its decisions to understand on what basis the EPA rejected alternatives that may or may not have reduced burden on small entities while meeting the stated objectives of the rule. Additionally, the commenter notes that the EPA did not evaluate the economic or environmental impacts of significant alternatives to the proposed rule. One commenter believes that the EPA's stated reasons for declining to specify or analyze an area source standard are inadequate under the RFA. The commenter believes the EPA must give serious consideration to regulatory alternatives that accomplish the stated objectives of the CAA while minimizing any significant economic impacts on small entities and that the EPA has a duty to specify and analyze this option or to more clearly state its policy reasons for excluding serious consideration of a separate standard for area sources. A commenter believes

the EPA did not fully consider the subcategorization of sources such as boilers designed to burn lignite coals versus other fossil fuels, especially in regard to non-Hg metal and acid gas emissions. The commenter references the SBAR Panel Report suggestion provided in the preamble of the proposed rule that the EPA consider developing an area source vs. major source distinction for the source category and the EPA's response. Another commenter is concerned that the recommendations made by the SER participants were ignored and not discussed in the rulemaking. Specifically, the commenter notes the EPA did not discuss subcategorizing by age, type of plant, fuel, physical space constraints or useful anticipated life of the plant. Nor did the EPA establish GACT for smaller emitters to alleviate regulatory costs and operational difficulties. A commenter believes it is likely that different numerical or work practice standards are appropriate for area sources of HAP.

Response: The EPA disagrees with one commenter's assertion that the agency has not complied with the requirements of the RFA. The EPA complied with both the letter and spirit of the RFA, notwithstanding the constraints of the court-ordered deadline. For example, the EPA notified the Chief Counsel for Advocacy of the SBA of its intent to convene a Panel; compiled a list of SERs for the Panel to consult with; and convened the Panel. The Panel met with SERs to collect their advice and recommendations; reviewed the EPA materials; and drafted a report of Panel findings. The EPA further disagrees with the commenter's assertion that the EPA's IRFA does not sufficiently consider impacts on small entities. The EPA's IRFA, which is included in chapter 10 of the RIA for the proposed rule, addresses the statutorily required elements of an IRFA such as, the economic impact of the proposed rule on small entities and the Panel's findings.

The EPA disagrees with the comment that recommendations made by the SERs and not considered or discussed in the proposed rulemaking such as recommendations regarding subcategorization and separate GACT standards for area sources. The preamble to the proposed standards includes a detailed discussion of how the EPA determined which subcategories and sources would be regulated (76 FR 25036-25037, May 3, 2011). In that discussion, the EPA explains the rationale for its proposed subcategories based on five unit design types. In addition, the EPA acknowledges the subcategorization suggestions from the SERs and explains its reasons for not subcategorizing on those bases. The preamble to the proposed standards also includes a discussion of the SERs' suggestion that area source EGUs be distinguished from major-source EGUs and the EPA's reasons for not making that distinction (76 FR 25020-25021, May 3, 2011).

The EPA also disagrees with the suggestion that the Agency pursue an extension of the timeline for final rulemaking such that the SBAR Panel can be reconvened and a new IRFA can be

prepared and released for public comment prior to the final rulemaking. The EPA entered into a Consent Decree to resolve litigation alleging that the EPA failed to perform a non-discretionary duty to promulgate CAA section 112(d) standards for EGUs. *American Nurses Ass'n v. EPA*, 08-2198 (D.D.C.). That Decree required the EPA to sign the final MATS rule by November 16, 2011, unless the agency sought to extend the deadline consistent with the requirements of the modification provision of the Consent Decree. If plaintiffs in the *American Nurses* litigation objected to an extension request, which the EPA believes would have been likely based on their comments on the proposed rule, the Agency would have had to file a motion with the Court seeking an extension of the deadline. Consistent with governing case law, the Agency would have been required to demonstrate in its motion for extension that it was impossible to finalize the rule by the deadline provided in the Consent Decree. *See Sierra Club v. Jackson*, Civil Action No. 01-1537 (D.D.C.) (Opinion of the Court denying EPA's motion to extend a consent decree deadline). The EPA negotiated a 30-day extension and was able to complete the rule by December 16, 2011; accordingly, the Agency had no basis for seeking a further extension of time.

A detailed description of the changes made to the rule since proposal, including those made as a result of feedback received during the public comment process can be found in sections VI (NESHAP) and X (NSPS) in the preamble. Changes explained in the identified sections include those related to applicability; subcategorization; work practices; periods of startup, shutdown, and malfunction; initial testing and compliance; continuous compliance; and notification, recordkeeping, and reporting.

7.4.4 Description and Estimate of the Affected Small Entities

For the purposes of assessing the impacts of MATS on small entities, a small entity is defined as:

> (1) A small business according to the Small Business Administration size standards by the North American Industry Classification System (NAICS) category of the owning entity. The range of small business size standards for electric utilities is 4 billion kilowatt hours (kWh) of production or less;
>
> (2) A small government jurisdiction that is a government of a city, county, town, district, or special district with a population of less than 50,000; and
>
> (3) A small organization that is any not for profit enterprise that is independently owned and operated and is not dominant in its field.

The EPA examined the potential economic impacts to small entities associated with this rulemaking based on assumptions of how the affected entities will install control technologies in compliance with MATS. The SBREFA analysis does not examine potential indirect economic impacts associated with this rule, such as employment effects in industries providing fuel and pollution control equipment, or the potential effects of electricity price increases on industries and households.

The EPA used Velocity Suite's Ventyx data as a basis for identifying plant ownership and compiling the list of potentially affected small entities. The Ventyx dataset contains detailed ownership and corporate affiliation information. The analysis focused only on those EGUs affected by the rule, which includes units burning coal, oil, petroleum coke, or coal refuse as the primary fuel, and excludes any combustion turbine units or EGUs burning natural gas. Also, because the rule does not affect combustion units with an equivalent electricity generating capacity up to 25 megawatts (MW), small entities that do not own at least one combustion unit with a capacity greater than 25 MW were removed from the dataset. For the affected units remaining, boiler and generator capacity, heat input, generation, and emissions data were aggregated by owner and then by parent company. Entities with more than 4 billion kWh of annual electricity generation were removed from the list, as were municipal owned entities serving a population greater than 50,000. For cooperatives, investor-owned utilities, and subdivisions that generate less than 4 billion kWh of electricity annually but which may be part of a large entity, additional research on power sales, operating revenues, and other business activities was performed to make a final determination regarding size. Finally, small entities for which the EPA's modeling with the Integrated Planning Model (IPM) does not project generation in 2015 in the base case were omitted from the analysis because they are not projected to be operating and, thus, are not projected to face the costs of compliance with the rule. After omitting entities for the reasons above, the EPA identified a total of 82 potentially affected small entities that are affiliated with 102 electric generating units.

7.4.5 Compliance Cost Impacts

This section presents the methodology and results for estimating the impact of MATS on small entities in 2015 based on the following endpoints:

- annual economic impacts of MATS on small entities and

- ratio of small entity compliance cost impacts to revenues from electricity generation.[1]

7.4.5.1 Methodology for Estimating Impacts of MATS on Small Entities

EPA estimated compliance costs of MATS as follows:

$$C_{Compliance} = \Delta C_{Operating+Capital} + \Delta C_{Fuel} - \Delta R$$

where C represents a component of cost as labeled, and Δ R represents the value of change in electricity generation, calculated as the difference in revenues between the base case and MATS.

Based on this formula, compliance costs for a given small entity could either be positive or negative (i.e., cost savings) based on their compliance choices and market conditions. Under MATS, some units will forgo some level of electricity generation (and, thus, revenues) to comply and this impact will be lessened on those entities by the projected increase in electricity prices under the MATS scenario (which raises their revenues from the remainder of their sales). On the other hand, some units may increase electricity generation, and coupled with the increase in electricity prices, will see an increase in electricity revenues resulting in lower net compliance costs. If entities are able to increase revenue more than an increase in retrofit and fuel costs, ultimately they will have negative net compliance costs (or savings). Because this analysis evaluates the total costs as a sum of the costs associated with compliance choices as well as changes in electricity revenues, it captures savings or gains such as those described. As a result, what EPA describes as a cost is really more of a measure of the net economic impact of the rule on small entities.

For this analysis, EPA used unit-level IPM parsed outputs – from modeling runs conducted with EPA's base case v4.10_MATS assumptions – to estimate costs based on the parameters above. These impacts were then summed for each small entity, adjusting for ownership share.[2] Net impact estimates were based on the following: changes in operating and capital costs, driven mainly by retrofit installations or upgrades, change in fuel costs, and

[1] This methodology for estimating small entity impacts has been used in recent EPA rulemakings such as the CSAPR promulgated by EPA in July, 2011.

[2] Unit-level cost impacts are adjusted for ownership shares for individual small entities, so as not to overestimate burden on each company. If an individual unit is owned by multiple small entities, total costs for that unit to meet MATS obligations are distributed across all owners based on the percentage of the unit owned by each company. Ownership percentage was estimated based on the Ventyx database.

change in electricity generation revenues under MATS relative to the base case. These individual components of compliance cost were estimated as follows:

(1) **Operating and capital costs:** Using the IPM parsed outputs for the base case and MATS policy case, EPA identified units that installed one or more pollution control technologies under the rule. The equations for calculating operating and capital costs were adopted from technology assumptions used in EPA's version of IPM (version 4.10). The model calculates the capital cost (in $/MW); the fixed operation and maintenance (O&M) cost (in $/MW-year); and the variable O&M cost (in $/MWh).

(2) **Fuel costs:** Fuel costs were estimated by multiplying fuel input (in million British thermal units, MMBtu) by region and fuel prices ($/MMBtu) from EPA's modeling with IPM. The incremental fuel expenditures under MATS were then estimated by taking the difference in fuel costs between MATS and the base case.

(3) **Value of electricity generated:** EPA estimated the value of electricity generated by multiplying the electricity generation from EPA's IPM modeling results with the regionally-adjusted retail electricity price ($/MWh), for all entities except those categorized as "Private" in Ventyx. For private entities, EPA used wholesale electricity price instead of retail electricity price because most of the private entities are independent power producers (IPP). IPPs sell their electricity to wholesale purchasers and do not own transmission facilities and, thus, their revenue was estimated based on wholesale electricity prices.

7.4.5.2 Results

The number of potentially affected small entities by ownership type and potential impacts of MATS are summarized in Table 7-1. All costs are presented in 2007 dollars. EPA estimated the annualized net compliance cost to small entities to be approximately $106 million in 2015.

Table 7-1. Projected Impact of MATS on Small Entities in 2015

EGU Ownership Type	Number of Potentially Affected Entities	Number of Entities Projected to Withdraw all Affected Units as Uneconomic	Total Net Compliance Costs (2007$ millions)	Number of Small Entities with Compliance Cost > 1% of Generation Revenues	Number of Small Entities with Compliance Cost > 3% of Generation Revenues
Co-Op	19	0	-29.7	9	8
IOU	8	0	33.0	7	5
Municipal	42	0	49.7	16	15
Sub-division	9	0	44.8	4	3
Private	4	3	8.4	4	4
Total	**82**	**3**	**106**	**40**	**35**

Notes: The total number of entities with costs greater than 1 percent or 3 percent of revenues includes only entities experiencing positive costs. About 23 of the 82 total potentially affected small entities are estimated to have cost savings under MATS (see text above for an explanation).

Definitions of ownership types are based on those provided by Ventyx's Energy Velocity.

Co-op (Cooperative): non-profit, customer-owned electric companies that generate and/or distribute electric power.

IOU (Investor-Owned Utility): Includes Investor Owned assets (e.g., a marketer, independent power producer, financial entity) and electric companies owned by stockholders, etc.

Municipal: A municipal utility, responsible for power supply and distribution in a small region, such as a city.

Sub-division: Political Subdivision Utility is a county, municipality, school district, hospital district, or any other political subdivision that is not classified as a municipality under state law.

Private: Similar to investor-owned, but ownership shares are not openly traded on the stock markets.

Source: ICF International analysis based on IPM modeling results

EPA assessed the economic and financial impacts of the final rule using the ratio of compliance costs to the value of revenues from electricity generation, and our results focus on those entities for which this measure could be greater than 1 percent or 3 percent. Of the 82 small entities identified, EPA's analysis shows 40 entities may experience compliance costs greater than 1 percent of base generation revenues in 2015, and 35 may experience compliance costs greater than 3 percent of base revenues.[3] Also, all generating capacity at 3 small entities is projected to be uneconomic to maintain. In this analysis, the cost of withdrawing a unit as uneconomic is estimated as the base case profit that is forgone by not operating under the policy case. Because 35 of the 82 total entities, or more than 40 percent, are estimated to incur compliance cost greater than 3 percent of base revenues, EPA has

[3] One percent and three percent of generation revenue criteria based on: "EPA's *Action Development Process*: Final Guidance for EPA Rulewriters: Regulatory Flexibility Act as amended by the Small Business Regulatory Enforcement Fairness Act." OPEI Regulatory Development Series. November 2006. This can be found on the Internet at http://www.epa.gov/sbrefa/documents/rfaguidance11-00-06.pdf.

concluded that it cannot certify that there will be no SISNOSE for this rule. Results for small entities discussed here, however, do not account for the reality that electricity markets are regulated in parts of the country. Entities operating in regulated or cost-of-service markets should be able to recover all of their costs of compliance through rate adjustments.

Note that the estimated costs for small entities are significantly lower than those estimated by EPA for the MATS proposal (which were $379 million). This is driven by a small group of units (less than 6 percent) which were projected to be uneconomic to operate under the proposal (and hence incurred lost profits due to lost electricity revenues), but are now projected to continue their operations under MATS. In addition, EPA's modeling indicates one unit that would have operated at a low capacity factor under the base case would find it economical to increase its generation significantly under MATS to meet electricity demand in its region. Excluding this unit, the total cost impacts across all entities would be roughly $175 million. Changes in compliance behavior for this small group of units, in particular the one unit which operates at a higher capacity factor, has a substantial impact on total costs for the entire group as their increased generation revenues offsets a large portion of the compliance costs.

The separate components of annualized costs to small entities under MATS are summarized in Table 7-2. The most significant components of incremental costs to these entities are increased capital and operating costs for retrofits, followed by changes in electricity revenues (i.e., cost savings).

Table 7-2. Incremental Annualized Costs under MATS Summarized by Ownership Group and Cost Category in 2015 (2007$ millions)

EGU Ownership Type	Capital+ Operating Costs ($MM)	Fuel Costs ($MM)	Change in Electricity Revenue ($MM)	Total
	A	B	C	**=A+B-C**
Co-Op	161.5	86.4	277.5	-29.7
IOU	39.3	0	6.3	33.0
Municipal	76.4	1.9	28.7	49.7
Sub-division	73.9	2.2	31.3	44.8
Private	5.5	0	-2.9	8.4
Total	**356**	**91**	**341**	**106**

Note: Totals may not add due to rounding.
Source: ICF International analysis based on IPM modeling results

Capital and operating costs increase across all ownership types, but the direction of changes in electricity revenues vary among ownership types. All ownership types, with the exception of private entities, experience a net gain in electricity revenues under the MATS, unlike projections from EPA's modeling during the proposal, where only municipals benefitted from higher electricity revenues. The change in electricity revenue takes into account both the profit lost from units that do not operate under the policy case and the difference in revenue for operating units under the policy case. According to EPA's modeling, an estimated 274 MW of capacity owned by small entities is considered uneconomic to operate under the policy case, resulting in a net loss of $13 million (in 2007$) in profits. On the other hand, many operating units actually increase their electricity revenue due to higher electricity prices under MATS. In addition, as mentioned above, EPA's modeling indicates one unit finds it economical to increase its capacity factor significantly under the policy case which results in significantly higher revenues offsetting the costs.

7.4.6 Description of Steps to Minimize Impacts on Small Entities

Consistent with the requirements of the RFA and SBREFA, the EPA has taken steps to minimize the significant economic impact on small entities. Because this rule does not affect units with a generating capacity of less than 25 MW, small entities that do not own at least one generating unit with a capacity greater than 25 MW are not subject to the rule. According to the EPA's analysis, among the coal- and oil-fired EGUs (i.e., excluding combined cycle gas turbines and gas combustion turbines) about 26 potentially small entities only own EGUs with a

capacity less than or equal to 25 MW, and none of those entities are subject to the final rule based on the statutory definition of potentially regulated units.

For units affected by the proposed rule, the EPA considered a number of comments received, both during the Small Business Advocacy Review (SBAR) Panel and the public comment period. While none of the alternatives adopted are specifically applied to small entities, the EPA believes these modifications will make compliance less onerous for all regulated units, including those owned by small entities.

7.4.6.1 Work Practice Standards

Consistent with Sierra Club v. EPA, the EPA proposed numerical emission standards that would apply at all times, including during periods of startup and shutdown. After reviewing comments and other data regarding the nature of these periods of operation, the EPA is finalizing a work practice standard for periods of start up and shut down. The EPA is also finalizing work practice standards for organic HAP from all subcategories of EGUs. The EPA has chosen to finalize work practice standards because the significant majority of data for measured organic HAP emissions from EGUs are below the detection levels of the EPA test methods, and, as such, the Agency considers it impracticable to reliably measure emissions from these units. Descriptions of the work practice requirements for startup and shutdown, as well as organic HAP, can be found in Section VI.D-E. of the preamble.

7.4.6.2 Continuous Compliance and Notification, Record-keeping, and Reporting

The final rule greatly simplifies the continuous compliance requirements and provides two basic approaches for most situations: use of continuous monitoring and periodic testing. The frequency of periodic testing has been decreased from monthly in the proposal to quarterly in the final rule. In addition to simplifying compliance, the EPA believes these changes considerably reduce the overall burden associated with recordkeeping and reporting. These changes to the final rule are described in more detail in Section VI.G-H. of the preamble.

7.4.6.3 Subcategorization

The Small Entity Representatives on the SBAR Panel were generally supportive of subcategorization and suggested a number of additional subcategories the EPA should consider when developing the final rule. While it was not practicable to adopt the proposed subcategories, the EPA maintained the existing subcategories and split the “liquid oil-fired units” subcategory into two individual subcategories – continental and non-continental units.

7.4.6.4 MACT Floor Calculations

As recommended by the EPA SBAR Panel representative, the EPA established the MACT floors using all the available ICR data that was received to the maximum extent possible consistent with the CAA requirements. The Agency believes this approach reasonably ensures that the emission limits selected as the MACT floors adequately represent the level of emissions actually achieved by the average of the units in the top 12 percent, considering operational variability of those units. Additionally, following proposal, the EPA reviewed and revised the procedure intended to account for the contribution of measurement imprecision to data variability in establishing effective emissions limits.

7.4.6.5 Alternatives Not Adopted

The EPA chose not to adopt several of the suggestions posed either during the SBAR Panel or public comment period. The EPA did not propose a percent reduction standard as an alternative to the concentration-based MACT floor. The percent reduction format for Hg and other HAP emissions would not have addressed the EPA's desire to promote, and give credit for, coal preparation practices that remove Hg and other HAP before firing. Also, to account for the coal preparation practices, sources would be required to track the HAP concentrations in coal from the mine to the stack, and not just before and after the control device(s), and such an approach would be difficult to implement and enforce. Furthermore, the EPA does not believe the percent reduction standard is in line with the Court's interpretation of the Clean Air Act section 112 requirements. Even if we believed it was appropriate to establish a percent reduction standard, we do not have the data necessary to establish percent reduction standards for HAP, as explained further in the response to comments document.

The EPA chose not to establish GACT standards for area sources for a number of reasons. The data show that similar HAP emissions and control technologies are found on both major and area sources greater than 25 MWe, and some large units are synthetic area sources. In fact, because of the significant number of well-controlled EGUs of all sizes, we believe it would be difficult to make a distinction between MACT and GACT. Moreover, the EPA believes the standards for area source EGUs should reflect MACT, rather than GACT, because there is no essential difference between area source and major source EGUs with respect to emissions of HAP.

The EPA chose not to exercise its discretionary authority to establish health-based emission standards for HCL and other HAP acid gases. Given the limitations of the currently available information (e.g., the HAP mix where EGUs are located, and the cumulative impacts of

respiratory irritants from nearby sources), the environmental effects of HCl and the other acid gas HAP, and the significant co-benefits from reductions in criteria pollutants the EPA determined that setting a conventional MACT standard for HCl and the other acid gas HAP was the appropriate course of action.

As required by section 212 of SBREFA, the EPA also is preparing a Small Entity Compliance Guide to help small entities comply with this rule. Small entities will be able to obtain a copy of the Small Entity Compliance guide at the following Web site: http://www.epa.gov/airquality/powerplanttoxics/actions.html.

7.5 Unfunded Mandates Reform Act (UMRA) Analysis

Title II of the UMRA of 1995 (Public Law 104-4)(UMRA) establishes requirements for federal agencies to assess the effects of their regulatory actions on state, local, and tribal governments and the private sector. Under Section 202 of the UMRA, 2 U.S.C. 1532, EPA generally must prepare a written statement, including a cost-benefit analysis, for any proposed or final rule that "includes any Federal mandate that may result in the expenditure by State, local, and tribal governments, in the aggregate, or by the private sector, of $100,000,000 or more ... in any one year." A "Federal mandate" is defined under Section 421(6), 2 U.S.C. 658(6), to include a "Federal intergovernmental mandate" and a "Federal private sector mandate." A "Federal intergovernmental mandate," in turn, is defined to include a regulation that "would impose an enforceable duty upon State, Local, or tribal governments," Section 421(5)(A)(i), 2 U.S.C. 658(5)(A)(i), except for, among other things, a duty that is "a condition of Federal assistance," Section 421(5)(A)(i)(I). A "Federal private sector mandate" includes a regulation that "would impose an enforceable duty upon the private sector," with certain exceptions, Section 421(7)(A), 2 U.S.C. 658(7)(A).

Before promulgating an EPA rule for which a written statement is needed under Section 202 of the UMRA, Section 205, 2 U.S.C. 1535, of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective, or least burdensome alternative that achieves the objectives of the rule. Moreover, section 205 allows EPA to adopt an alternative other than the least costly, most cost-effective or least burdensome alternative if the Administrator publishes an explanation why that alternative was not adopted.

In a manner consistent with the intergovernmental consultation provisions of Section 204 of the UMRA, EPA carried out consultations with the governmental entities affected by this rule. EPA held meetings with states and tribal representatives in which the Agency presented its

plan to develop a proposal and provided opportunities for participants to provide input as part of the rulemaking process. EPA has also analyzed the economic impacts of MATS on government entities and this section presents the results of that analysis. The UMRA analysis does not examine potential indirect economic impacts associated with the rule, such as employment effects in industries providing fuel and pollution control equipment, or the potential effects of electricity price increases on industries and households.

7.5.1 Identification of Affected Government Entities

Using Ventyx data, EPA identified state- and municipality-owned utilities and subdivisions that would be affected by this rule. EPA then used IPM parsed outputs (based on EPA modeling assumptions) to associate these entities with individual generating units. The analysis focused only on EGUs affected by MATS, which includes units burning coal, oil, petroleum coke, or waste coal as the primary fuel, and excludes any combustion turbine units. Entities that did not own at least one unit with a generating capacity of greater than 25 MW were also removed from the dataset because of their exemption from the rule. Finally, government entities for which EPA's modeling does not project generation in 2015 under the base case were also exempted from this analysis, because they are not projected to operate and are thus not projected to face compliance costs with this rule. Based on this, EPA identified 96 state, municipal, and sub-divisions affiliated with 172 electric generating units that are potentially affected by MATS.

7.5.2 Compliance Cost Impacts

After identifying the potentially affected government entities, EPA estimated the impact of MATS in 2015 based on the following:

- total impacts of compliance on government entities and
- ratio of government entity impacts to revenues from electricity generation.

7.5.2.1 Methodology for Estimating Impacts MATS on Government Entities

EPA estimated compliance costs of MATS as follows:

$$C_{Compliance} = \Delta C_{Operating+Capital} + \Delta C_{Fuel} - \Delta R$$

where C represents a component of cost as labeled, and Δ R represents the retail value of change in electricity generation, calculated as the difference in projected revenues between the base case and MATS.

Based on this formula, compliance costs for a given government entity could either be positive or negative (i.e., cost savings) based on their compliance choices and market conditions. Under MATS, some units will forgo some level of electricity generation (and thus revenues) to comply and this impact will be lessened on those entities by the projected increase in electricity prices under MATS. On the other hand, some units may increase electricity generation, and coupled with the increase in electricity prices, will see an increase in electricity revenues resulting in lower net compliance costs. If entities are able to increase revenue more than an increase in retrofit and fuel costs, ultimately they will have negative net compliance costs (or savings). Because this analysis evaluates the total costs as a sum of the costs associated with compliance choices as well as changes in electricity revenues, it captures savings or gains such as those described. As a result, what EPA describes as a cost is really more of a measure of the net economic impact of the rule on government entities.

For this analysis, EPA used unit-level data from IPM runs conducted with EPA's modeling assumptions to estimate costs based on the parameters above. These impacts were then aggregated for each government entity, adjusting for ownership share. Compliance cost estimates were based on the following: changes in capital and operating costs, change in fuel costs, and change in electricity generation revenues under MATS relative to the base case. These components of compliance cost were estimated as follows:

(1) Capital and operating costs: Using EPA's modeling results for the base case and the MATS policy case, EPA identified units that install control technology under this rule and the technologies installed. The equations for calculating operating and capital costs were adopted from EPA's version of IPM (version 4.10_MATS). The model calculates the capital cost (in $/MW); the fixed operation and maintenance (O&M) cost (in $/MW-year); and the variable O&M cost (in $/MWh)

(2) Fuel costs: Fuel costs were estimated by multiplying fuel input (MMBtu) by region and fuel prices ($/MMBtu) from EPA's modeling. The change in fuel expenditures under MATS was then estimated by taking the difference in fuel costs between MATS and the base case.

(3) Value of electricity generated: EPA estimated the value of electricity generated by multiplying the estimated electricity generation from EPA's IPM modeling results with the regional-adjusted retail electricity prices ($/MWh).

7.5.2.2 Results

As was done for the small entities analysis, EPA assessed the economic and financial impacts of the rule using the ratio of compliance costs to the value of revenues from electricity generation, and our results focus on those entities for which this measure could be greater than 1 percent or 3 percent of base revenues. EPA projects that 42 government entities will have compliance costs greater than 1 percent of base generation revenue in 2015 and 32 may experience compliance costs greater than 3 percent of base revenues. Overall, 6 units owned by government entities are projected to be uneconomic to maintain.

The separate components of the annualized costs to government entities under MATS are summarized in Table 7-3 below. The most significant components of incremental costs to these entities are the increased capital and operating costs, followed by increases in electricity revenues (i.e., a cost saving).

Table 7-3. Incremental Annualized Costs under MATS Summarized by Ownership Group and Cost Category (2007$ millions) in 2015

EGU Ownership Type	Capital Costs + Operating Costs($MM)	Fuel Costs ($ MM)	Change in Revenue ($ MM)	Total
	A	B	C	=A+B-C
Sub-Division	128.0	50.7	106.4	72.3
State	65.9	1.2	32.7	34.4
Municipal	516.3	45.4	374.3	187.4
Total	**710**	**97**	**513**	**294**

Note: Totals may not add due to rounding.
Definitions of ownership types are based on those provided by Ventyx's Energy Velocity.
Municipal: A municipal utility, responsible for power supply and distribution in a small region, such as a city.
Sub-division: Political Subdivision Utility is a county, municipality, school district, hospital district, or any other political subdivision that is not classified as a municipality under state law.
Source: ICF International analysis based on IPM modeling results

The number of potentially affected government entities by ownership type and potential impacts of MATS are summarized in Table 7-4. All costs are reported in 2007$ millions. EPA estimated the annualized net compliance cost to government entities to be approximately $294 million in 2015.

Table 7-4. Summary of Potential Impacts on Government Entities under MATS in 2015

EGU Ownership Type	Number of Potentially Affected Entities	Number of Entities Withdrawing all Affected units	Total Net Costs of MACT compliance ($ MM)	Number of Government Entities with Compliance Cost > 1% of Generation Revenues	Number of Government Entities with Compliance Cost > 3% of Generation Revenues
Sub-Division	11	0	72.3	5	4
State	5	0	34.4	4	3
Municipal	80	0	187.4	33	25
Total	**96**	**0**	**294**	**42**	**32**

Note: The total number of entities with costs greater than 1 percent or 3 percent of revenues includes only entities experiencing positive costs. About 30 of the 96 total potentially affected government entities are estimated to have cost savings under the MACT policy case (see text above for an explanation).

Source: ICF International analysis based on IPM modeling results

Capital and operating costs increase over all ownership types. All ownership types, however, also experience a net gain in electricity revenue, mainly due to higher electricity prices under the policy case. As described in the small entity analysis, the change in electricity revenue takes into account both the profit lost from units that do not operate under the policy case and the difference in revenue for operating units under the policy case. According to EPA's modeling, an estimated 757 MW of electricity generation is estimated to be uneconomic to operate under the policy case, accounting for about $20 million in lost profits. On the other hand, many operating units actually increase their electricity revenue due to higher electricity prices under the MATS policy scenario.

7.6 Executive Order 13132, Federalism

Under EO 13132, the EPA may not issue an action that has federalism implications, that imposes substantial direct compliance costs, and that is not required by statute, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by state and local governments, or the EPA consults with state and local officials early in the process of developing the final action.

The EPA has concluded that this action may have federalism implications, because it may impose substantial direct compliance costs on state or local governments, and the Federal government will not provide the funds necessary to pay those costs. Accordingly, the EPA provides the following federalism summary impact statement as required by section 6(b) of EO 13132.

Based on estimates in the RIA, provided in the docket, the final rule may have federalism implications because the rule may impose approximately $294 million in annual direct compliance costs on an estimated 96 state or local governments. Specifically, we estimate that there are 80 municipalities, 5 states, and 11 political subdivisions (i.e., a public district with territorial boundaries embracing an area wider than a single municipality and frequently covering more than one county for the purpose of generating, transmitting and distributing electric energy) that may be directly impacted by this final rule. Responses to the EPA's 2010 ICR were used to estimate the nationwide number of potentially impacted state or local governments. As previously explained, this 2010 survey was submitted to all coal- and oil-fired EGUs listed in the 2007 version of DOE/EIA's "Annual Electric Generator Report," and "Power Plant Operations Report."

The EPA consulted with state and local officials in the process of developing the rule to permit them to have meaningful and timely input into its development. The EPA met with 10 national organizations representing state and local elected officials to provide general background on the rule, answer questions, and solicit input.

7.7 Executive Order 13175, Consultation and Coordination with Indian Tribal Governments

EPA has concluded that this action may have tribal implications. The EPA offered consultation with tribal officials early in the regulation development process to permit them an opportunity to have meaningful and timely input. Consultation letters were sent to 584 tribal leaders and provided information regarding the EPA's development of this rule and offered consultation. Three consultation meetings were held: December 7, 2010, with the Upper Sioux Community of Minnesota; December 13, 2010, with the Moapa Band of Paiutes, Forest County Potawatomi, Standing Rock Sioux Tribal Council, and Fond du Lac Band of Chippewa; January 5, 2011, with the Forest County Potawatomi and a representative from the National Tribal Air Association. In these meetings, the EPA presented the authority under the CAA used to develop these rules and an overview of the industry and the industrial processes that have the potential for regulation. Tribes expressed concerns about the impact of EGUs on Indian country. Specifically, they were concerned about potential Hg deposition and the impact on the water resources of the Tribes, with particular concern about the impact on subsistence lifestyles for fishing communities, the cultural impact of impaired water quality for ceremonial purposes, and the economic impact on tourism. In light of these concerns, the Tribes expressed interest in an expedited implementation of the rule. Other concerns expressed by Tribes related to how the Agency would consider variability in setting the standards and the use of tribal-specific fish

consumption data from the Tribes in our assessments. They were not supportive of using work practice standards as part of the rule and asked the Agency to consider going beyond the MACT floor to offer more protection for the tribal communities.

In addition to these consultations, the EPA also conducted outreach on this rule through presentations at the National Tribal Forum in Milwaukee, WI; phone calls with the National Tribal Air Association; and a webinar for Tribes on the proposed rule. The EPA specifically requested tribal data that could support the appropriate and necessary analyses and the RIA for this rule. In addition, the EPA held individual consultations with the Navajo Nation on October 12, 2011; as well as the Gila River Indian Community, Ak-Chin Indian Community, and the Hopi Nation on October 14, 2011. These Tribes expressed concerns about the impact of the rule on the Navajo Generating Station (NGS), the impact on the cost of the water allotted to the Tribes from the Central Arizona Project (CAP), the impact on tribal revenues from the coal mining operations (i.e., assumptions about reduced mining if NGS were to retire one or more units), and the impacts on employment of tribal members at both the NGS and the mine. More specific comments can be found in the docket.

7.8 Protection of Children from Environmental Health and Safety Risks

This final rule is subject to EO 13045 (62 FR 19885, April 23, 1997) because it is an economically significant regulatory action as defined by EO 12866, and the EPA believes that the environmental health or safety risk addressed by this action may have a disproportionate effect on children. Accordingly, we have evaluated the environmental health or safety effects of the standards on children.

Although this final rule is based on technology performance, the standards are designed to protect against hazards to public health with an adequate margin of safety as described in the preamble. The protection offered by this rule may be particularly important for children, especially the developing fetus. As referenced in Chapter 4 of this RIA, "Mercury and Other HAP Benefits Analysis," children are more vulnerable than adults to many HAP emitted by EGUs due to differential behavior patterns and physiology. These unique susceptibilities were carefully considered in a number of different ways in the analyses associated with this rulemaking, and are summarized in the RIA.

7.9 Statement of Energy Effects

Our analysis to comply with EO 13211 (Statement of Energy Effects) can be found in Section 3.16 of this RIA.

7.10 National Technology Transfer and Advancement Act

Section 12(d) of the National Technology Transfer and Advancement Act (NTTAA) of 1995 (Public Law No. 104-113; 15 U.S.C. 272 note) directs the EPA to use voluntary consensus standards in its regulatory activities unless to do so would be inconsistent with applicable law or otherwise impractical. Voluntary consensus standards are technical standards (e.g., materials specifications, test methods, sampling procedures, business practices) that are developed or adopted by voluntary consensus standards bodies. The NTTAA directs the EPA to provide Congress, through OMB, explanations when the Agency decides not to use available and applicable voluntary consensus standards.

This rulemaking involves technical standards. The EPA cites the following standards in the final rule: EPA Methods 1, 2, 2A, 2C, 2F, 2G, 3A, 3B, 4, 5, 5D, 17, 19, 23, 26, 26A, 29, 30B of 40 CFR Part 60 and Method 320 of 40 CFR Part 63. Consistent with the NTTAA, the EPA conducted searches to identify voluntary consensus standards in addition to these EPA methods. No applicable voluntary consensus standards were identified for EPA Methods 2F, 2G, 5D, and 19. The search and review results have been documented and are placed in the docket for the proposed rule.

The three voluntary consensus standards described below were identified as acceptable alternatives to EPA test methods for the purposes of the final rule.

The voluntary consensus standard American National Standards Institute (ANSI) / American Society of Mechanical Engineers (ASME) PTC 19-10-1981, “Flue and Exhaust Gas Analyses [Part 10, Instruments and Apparatus]” is cited in the final rule for its manual method for measuring the O2, CO2, and CO content of exhaust gas. This part of ANSI/ASME PTC 19-10-1981 is an acceptable alternative to Method 3B.

The voluntary consensus standard ASTM D6348-03 (Reapproved 2010), “Standards Test Method for Determination of Gaseous Compounds by Extractive Direct Interface Fourier Transform (FTIR) Spectroscopy” is acceptable as an alternative to Method 320 and is cited in the final rule, but with several conditions: (1) The test plan preparation and implementation in the Annexes to ASTM D-6348-03, Sections A1 through A8 are mandatory; and (2) In ASTM D6348-03 Annex A5 (Analyte Spiking Technique), the percent (%) R must be determined for each target analyte (Equation A5.5). In order for the test data to be acceptable for a compound, %R must be 70 % ≥ R ≤ 130%. If the %R value does not meet this criterion for a target compound, the test data are not acceptable for that compound and the test must be repeated for that analyte (i.e., the sampling and/or analytical procedure should be adjusted before a

retest). The %R value for each compound must be reported in the test report, and all field measurements must be corrected with the calculated %R value for that compound by using the following equation: Reported Result = (Measured Concentration in the Stack x 100) / % R.

The voluntary consensus standard ASTM D6784-02, "Standard Test Method for Elemental, Oxidized, Particle-Bound and Total Mercury in Flue Gas Generated from Coal-Fired Stationary Sources (Ontario Hydro Method)," is an acceptable alternative to use of EPA Method 29 for Hg only or Method 30B for the purpose of conducting relative accuracy tests of mercury continuous monitoring systems under this final rule. Because of the limitations of this method in terms of total sampling volume, it is not appropriate for use in performance testing under this rule. In addition to the voluntary consensus standards the EPA used in the final rule, the search for emissions measurement procedures identified 16 other voluntary consensus standards. The EPA determined that 14 of these 16 standards identified for measuring emissions of the HAP or surrogates subject to emission standards in the final rule were impractical alternatives to EPA test methods for the purposes of this final rule. Therefore, the EPA does not intend to adopt these standards for this purpose. The reasons for this determination for the 14 methods are discussed below, and the remaining 2 methods are discussed later in this section.

The voluntary consensus standard ASTM D3154-00, "Standard Method for Average Velocity in a Duct (Pitot Tube Method)," is impractical as an alternative to EPA Methods 1, 2, 3B, and 4 for the purposes of this rulemaking because the standard appears to lack in quality control and quality assurance requirements. Specifically, ASTM D3154-00 does not include the following: (1) proof that openings of standard pitot tube have not plugged during the test; (2) if differential pressure gauges other than inclined manometers (e.g., magnehelic gauges) are used, their calibration must be checked after each test series; and (3) the frequency and validity range for calibration of the temperature sensors.

The voluntary consensus standard ASTM D3464-96 (Reapproved 2001), "Standard Test Method Average Velocity in a Duct Using a Thermal Anemometer," is impractical as an alternative to EPA Method 2 for the purposes of this rule primarily because applicability specifications are not clearly defined, e.g., range of gas composition, temperature limits. Also, the lack of supporting quality assurance data for the calibration procedures and specifications, and certain variability issues that are not adequately addressed by the standard limit the EPA's ability to make a definitive comparison of the method in these areas.

The voluntary consensus standard ISO 10780:1994, "Stationary Source Emissions–Measurement of Velocity and Volume Flowrate of Gas Streams in Ducts," is impractical as an alternative to EPA Method 2 in this rule. The standard recommends the use of an L-shaped pitot, which historically has not been recommended by the EPA. The EPA specifies the S-type design which has large openings that are less likely to plug up with dust.

The voluntary consensus standard, CAN/CSA Z223.2-M86 (1999), "Method for the Continuous Measurement of Oxygen, Carbon Dioxide, Carbon Monoxide, Sulphur Dioxide, and Oxides of Nitrogen in Enclosed Combustion Flue Gas Streams," is unacceptable as a substitute for EPA Method 3A because it does not include quantitative specifications for measurement system performance, most notably the calibration procedures and instrument performance characteristics. The instrument performance characteristics that are provided are non-mandatory and also do not provide the same level of quality assurance as the EPA methods. For example, the zero and span/calibration drift is only checked weekly, whereas the EPA methods require drift checks after each run.

Two very similar voluntary consensus standards, ASTM D5835-95 (Reapproved 2001), "Standard Practice for Sampling Stationary Source Emissions for Automated Determination of Gas Concentration," and ISO 10396:1993, "Stationary Source Emissions: Sampling for the Automated Determination of Gas Concentrations," are impractical alternatives to EPA Method 3A for the purposes of this final rule because they lack in detail and quality assurance/quality control requirements. Specifically, these two standards do not include the following: (1) sensitivity of the method; (2) acceptable levels of analyzer calibration error; (3) acceptable levels of sampling system bias; (4) zero drift and calibration drift limits, time span, and required testing frequency; (5) a method to test the interference response of the analyzer; (6) procedures to determine the minimum sampling time per run and minimum measurement time; and (7) specifications for data recorders, in terms of resolution (all types) and recording intervals (digital and analog recorders, only).

The voluntary consensus standard ISO 12039:2001, "Stationary Source Emissions--Determination of Carbon Monoxide, Carbon Dioxide, and Oxygen--Automated Methods," is not acceptable as an alternative to EPA Method 3A. This ISO standard is similar to EPA Method 3A, but is missing some key features. In terms of sampling, the hardware required by ISO 12039:2001 does not include a 3-way calibration valve assembly or equivalent to block the sample gas flow while calibration gases are introduced. In its calibration procedures, ISO 12039:2001 only specifies a two-point calibration while EPA Method 3A specifies a three-point calibration. Also, ISO 12039:2001 does not specify performance criteria for calibration error,

calibration drift, or sampling system bias tests as in the EPA method, although checks of these quality control features are required by the ISO standard.

The voluntary consensus standard ASTM D6522-00, "Standard Test Method for the Determination of Nitrogen Oxides, Carbon Monoxide, and Oxygen Concentrations in Emissions from Natural Gas-Fired Reciprocating Engines, Combustion Turbines, Boilers and Process Heaters Using Portable Analyzers" is not an acceptable alternative to EPA Method 3A for measuring CO and O2 concentrations for this final rule as the method is designed for application to sources firing natural gas.

The voluntary consensus standard ASME PTC-38-80 R85 (1985), "Determination of the Concentration of Particulate Matter in Gas Streams," is not acceptable as an alternative for EPA Method 5 because ASTM PTC-38-80 is not specific about equipment requirements, and instead presents the options available and the pros and cons of each option. The key specific differences between ASME PTC-38-80 and the EPA methods are that the ASME standard: (1) allows in-stack filter placement as compared to the out-of-stack filter placement in EPA Methods 5 and 17; (2) allows many different types of nozzles, pitots, and filtering equipment; (3) does not specify a filter weighing protocol or a minimum allowable filter weight fluctuation as in the EPA methods; and (4) allows filter paper to be only 99 percent efficient, as compared to the 99.95 percent efficiency required by the EPA methods.

The voluntary consensus standard ASTM D3685/D3685M-98, "Test Methods for Sampling and Determination of Particulate Matter in Stack Gases," is similar to EPA Methods 5 and 17, but is lacking in the following areas that are needed to produce quality, representative particulate data: (1) requirement that the filter holder temperature should be between 120oC and 134oC, and not just "above the acid dew-point;" (2) detailed specifications for measuring and monitoring the filter holder temperature during sampling; (3) procedures similar to EPA Methods 1, 2, 3, and 4, that are required by EPA Method 5; (4) technical guidance for performing the Method 5 sampling procedures, e.g., maintaining and monitoring sampling train operating temperatures, specific leak check guidelines and procedures, and use of reagent blanks for determining and subtracting background contamination; and (5) detailed equipment and/or operational requirements, e.g., component exchange leak checks, use of glass cyclones for heavy particulate loading and/or water droplets, operating under a negative stack pressure, exchanging particulate loaded filters, sampling preparation and implementation guidance, sample recovery guidance, data reduction guidance, and particulate sample calculations input.

The voluntary consensus standard ISO 9096:1992, "Determination of Concentration and Mass Flow Rate of Particulate Matter in Gas Carrying Ducts - Manual Gravimetric Method," is not acceptable as an alternative for EPA Method 5. Although sections of ISO 9096 incorporate EPA Methods 1, 2, and 5 to some degree, this ISO standard is not equivalent to EPA Method 5 for collection of PM. The standard ISO 9096 does not provide applicable technical guidance for performing many of the integral procedures specified in Methods 1, 2, and 5. Major performance and operational details are lacking or nonexistent and detailed quality assurance/quality control guidance for the sampling operations required to produce quality, representative particulate data (e.g., guidance for maintaining and monitoring train operating temperatures, specific leak check guidelines and procedures, and sample preparation and recovery procedures) are not provided by the standard, as in EPA Method 5. Also, details of equipment and/or operational requirements, such as those specified in EPA Method 5, are not included in the ISO standard, e.g., stack gas moisture measurements, data reduction guidance, and particulate sample calculations.

The voluntary consensus standard CAN/CSA Z223.1-M1977, "Method for the Determination of Particulate Mass Flows in Enclosed Gas Streams," is not acceptable as an alternative for EPA Method 5. Detailed technical procedures and quality control measures that are required in EPA Methods 1, 2, 3, and 4 are not included in CAN/CSA Z223.1. Second, CAN/CSA Z223.1 does not include the EPA Method 5 filter weighing requirement to repeat weighing every 6 hours until a constant weight is achieved. Third, EPA Method 5 requires the filter weight to be reported to the nearest 0.1 milligram (mg), while CAN/CSA Z223.1 requires reporting only to the nearest 0.5 mg. Also, CAN/CSA Z223.1 allows the use of a standard pitot for velocity measurement when plugging of the tube opening is not expected to be a problem. The EPA Method 5 requires an S-shaped pitot.

The voluntary consensus standard EN 1911-1,2,3 (1998), "Stationary Source Emissions-Manual Method of Determination of HCl-Part 1: Sampling of Gases Ratified European Text-Part 2: Gaseous Compounds Absorption Ratified European Text-Part 3: Adsorption Solutions Analysis and Calculation Ratified European Text," is impractical as an alternative to EPA Methods 26 and 26A. Part 3 of this standard cannot be considered equivalent to EPA Method 26 or 26A because the sample absorbing solution (water) would be expected to capture both HCl and chlorine gas, if present, without the ability to distinguish between the two. The EPA Methods 26 and 26A use an acidified absorbing solution to first separate HCl and chlorine gas so that they can be selectively absorbed, analyzed, and reported separately. In addition, in EN 1911 the absorption

efficiency for chlorine gas would be expected to vary as the pH of the water changed during sampling.

The voluntary consensus standard EN 13211 (1998), is not acceptable as an alternative to the Hg portion of EPA Method 29 primarily because it is not validated for use with impingers, as in the EPA method, although the method describes procedures for the use of impingers. This European standard is validated for the use of fritted bubblers only and requires the use of a side (split) stream arrangement for isokinetic sampling because of the low sampling rate of the bubblers (up to 3 liters per minute, maximum). Also, only two bubblers (or impingers) are required by EN 13211, whereas EPA Method 29 require the use of six impingers. In addition, EN 13211 does not include many of the quality control procedures of EPA Method 29, especially for the use and calibration of temperature sensors and controllers, sampling train assembly and disassembly, and filter weighing.

Two of the 16 voluntary consensus standards identified in this search were not available at the time the review was conducted for the purposes of the final rule because they are under development by a voluntary consensus body: ASME/BSR MFC 13M, "Flow Measurement by Velocity Traverse," for EPA Method 2 (and possibly 1); and ASME/BSR MFC 12M, "Flow in Closed Conduits Using Multiport Averaging Pitot Primary Flowmeters," for EPA Method 2.

Finally, in addition to the three voluntary consensus standards identified as acceptable alternatives to EPA methods required in the final rule, the EPA is also specifying four voluntary consensus standards in the rule for use in sampling and analysis of liquid oil samples for moisture content. These standards are: ASTM D95-05 (Reapproved 2010), "Standard Test Method for Water in Petroleum Products and Bituminous Materials by Distillation", ASTM D4006-11, "Standard Test Method for Water in Crude Oil by Distillation", ASTM D4177-95 (Reapproved 2010), "Standard Practice for Automatic Sampling of Petroleum and Petroleum Products, and ASTM D4057-06 (Reapproved 2011), "Standard Practice for Manual Sampling of Petroleum and Petroleum Products."

Table 5, section 4.1.1.5 of appendix A, and section 3.1.2 of appendix B to subpart UUUUU, 40 CFR Part 63, list the EPA testing methods included in the final rule. Under section 63.7(f) and section 63.8(f) of subpart A of the General Provisions, a source may apply to the EPA for permission to use alternative test methods or alternative monitoring requirements in place of any of the EPA testing methods, performance specifications, or procedures specified.

7.11 Environmental Justice

7.11.1 Environmental Justice Impacts

Executive Order 12898 (59 FR 7629, February 16, 1994) establishes Federal executive policy on environmental justice. Its main provision directs Federal agencies, to the greatest extent practicable and permitted by law, to make environmental justice (EJ) part of their mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of their programs, policies, and activities on minority populations and low-income populations in the U.S.

The EPA has determined that this final rule will not have disproportionately high and adverse human health or environmental effects on minority, low income, or indigenous populations because it increases the level of environmental protection for all affected populations.

This final rule establishes national emission standards for new and existing EGUs that combust coal and oil. The EPA estimates that there are approximately 1,400 units located at 575 facilities covered by this final rule.

This final rule will reduce emissions of all the listed HAP that come from EGUs. This includes metals (Hg, As, Be, Cd, Cr, Pb, Mn, Ni, and Se), organics (POM, acetaldehyde, acrolein, benzene, dioxins, ethylene dichloride, formaldehyde, and PCB), and acid gases (HCl and HF). At sufficient levels of exposure, these pollutants can cause a range of health effects including cancer; irritation of the lungs, skin, and mucous membranes; effects on the central nervous system such as memory and IQ loss and learning disabilities; damage to the kidneys; and other acute health disorders.

The final rule will also result in substantial reductions of criteria pollutants such as CO, PM, and SO2. Sulfur dioxide is a precursor pollutant that is often transformed into fine PM ($PM_{2.5}$) in the atmosphere. Reducing direct emissions of $PM_{2.5}$ and SO2 will, as a result, reduce concentrations of $PM_{2.5}$ in the atmosphere. These reductions in $PM_{2.5}$ will provide large health benefits, such as reducing the risk of premature mortality for adults, chronic and acute bronchitis, childhood asthma attacks, and hospitalizations for other respiratory and cardiovascular diseases. (For more details on the health effects of metals, organics, and $PM_{2.5}$, please refer to Chapters 4 and 5 of this RIA.) This final rule will also have a small effect on electricity and natural gas prices but has the potential to affect the cost structure of the utility industry and could lead to shifts in how and where electricity is generated.

Today's final rule is one of a group of regulatory actions that the EPA will take over the next several years to respond to statutory and judicial mandates that will reduce exposure to HAP and $PM_{2.5}$, as well as to other pollutants, from EGUs and other sources. In addition, the EPA will pursue energy efficiency improvements throughout the economy, along with other Federal agencies, states and other groups. This will contribute to additional environmental and public health improvements while lowering the costs of realizing those improvements. Together, these rules and actions will have substantial and long-term effects on both the U.S. power industry and on communities currently breathing dirty air. Therefore, we anticipate significant interest in these actions from EJ communities, as well as many others.

7.11.1.1 Key EJ Aspects of the Rule

This is an air toxics rule; therefore, it does not permit emissions trading among sources. Instead, this final rule will place a limit on the rates of Hg and other HAP emitted from each affected EGU. As a result, emissions of Hg and other HAP such as HCl will be substantially reduced in the vast majority of states. In some states, however, there may be small increases in Hg and other HAP emissions due to shifts in electricity generation from EGUs with higher emission rates to EGUs with already low emission rates. Hydrogen chloride emissions are projected to increase at a small number of sources but that does not lead to any increased emissions at the state level.

The primary risk analysis to support the finding that this final rule is both appropriate and necessary includes an analysis of the effects of Hg from EGUs on people who rely on freshwater fish they catch as a regular and frequent part of their diet. These groups are characterized as subsistence level fishing populations or fishers. A significant portion of the data in this analysis came from published studies of EJ communities where people frequently consume locally-caught freshwater fish. These communities included: (1) White and black populations (including female and poor strata) surveyed in South Carolina; (2) Hispanic, Vietnamese and Laotian populations surveyed in California; and (3) Great Lakes tribal populations (Chippewa and Ojibwe) active on ceded territories around the Great Lakes. These data were used to help estimate risks to similar populations beyond the areas where the study data was collected. For example, while the Vietnamese and Laotian survey data were collected in California, given the ethnic (heritage) nature of these high fish consumption rates, we assumed that they could also be associated with members of these ethnic groups living elsewhere in the U.S. Therefore, the high-end consumption rates referenced in the California study for these ethnic groups were used to model risk at watersheds elsewhere in the U.S. As a result of this approach, the specific fish consumption patterns of several different EJ groups are

fundamental to the EPA's assessment of both the underlying risks that make this final rule appropriate and necessary, and of the analysis of the benefits of reducing exposure to Hg and the other hazardous air pollutants.

The EPA's analysis of risks from consumption of Hg-contaminated fish is contained in Chapter 4 of this RIA. The effects of this final rule on the health risks from Hg and other HAP are presented in the preamble and in the RIA for this rule.

7.11.1.2 Potential Environmental and Public Health Impacts to Vulnerable Populations

The EPA has conducted several analyses that provide additional insight on the potential effects of this rule on EJ communities. These include: (1) The socio-economic distribution of people living close to affected EGUs who may be exposed to pollution from these sources; and (2) an analysis of the distribution of health effects expected from the reductions in $PM_{2.5}$ that will result from implementation of this final rule (''co-benefits'').

Socio-economic distribution. As part of the analysis for this final rule, the EPA reviewed the aggregate demographic makeup of the communities near EGUs covered by this final rule. Although this analysis gives some indication of populations that may be exposed to levels of pollution that cause concern, it does NOT identify the demographic characteristics of the most highly affected individuals or communities. EGUs usually have very tall emission stacks; this tends to disperse the pollutants emitted from these stacks fairly far from the source. In addition, several of the pollutants emitted by these sources, such as a common form of mercury and SO2, are known to travel long distances and contribute to adverse impacts on the environment and human health hundreds or even thousands of miles from where they were emitted (in the case of elemental mercury, globally).

The proximity-to-the-source review is included in the analysis for this final rule because some EGUs emit enough hazardous air pollutants such as Nickel or Chromium (VI) to cause elevated lifetime cancer risks greater than 1 in a million in nearby communities. In addition, the EPA's analysis indicates that there are localized areas with elevated levels of Hg deposition around most U.S. EGUs.[4]

The analysis of demographic data used proximity-to-the-source as a surrogate for exposure to identify those populations considered to be living near affected sources, such that they have notable exposures to current hazardous air pollutant emissions from these sources. The demographic data for this analysis were extracted from the 2000 census data which were

[4] See Excess Local Deposition TSD for more detail.

provided to the EPA by the US Census Bureau. Distributions by race are based on demographic information at the census block level, and all other demographic groups are based on the extrapolation of census block group level data to the census block level. The socio-demographic parameters used in the analysis included the following categories: Racial (White, African American, Native American, Other or Multiracial, and All Other Races); Ethnicity (Hispanic); and Other (Number of people below the poverty line, Number of people with ages between 0 and 18, Number of people greater than or equal to 65, Number of people with no high school diploma).

In determining the aggregate demographic makeup of the communities near affected sources, the EPA focused on those census blocks within three miles of affected sources and determined the demographic composition (e.g., race, income, etc.) of these census blocks and compared them to the corresponding compositions nationally. The radius of three miles (or approximately 5 kilometers) is consistent with other demographic analyses focused on areas around potential sources. In addition, air quality modeling experience has shown that the area within three miles of an individual source of emissions can generally be considered the area with the highest ambient air levels of the primary pollutants being emitted for most sources, both in absolute terms and relative to the contribution of other sources (assuming there are other sources in the area, as is typical in urban areas). While facility processes and fugitive emissions may have more localized impacts, the EPA acknowledges that because of various stack heights there is the potential for dispersion beyond 3 miles. To the extent that any minority, low income, or indigenous subpopulation is disproportionately impacted by the current emissions as a result of the proximity of their homes to these sources, that subpopulation also stands to see increased environmental and health benefit from the emissions reductions called for by this rule.

The results of EPA's demographic analysis for coal fired EGUs are shown in Table 7-5.

The data indicate that affected sources are located in areas where the minority share of the population living within a three mile buffer is higher than the national average by 12 percentage points or 48%. For these same areas, the percent of the population below the poverty line is also higher than the national average by 4 percentage points or 31%. These results are presented in more detail in the ''Review of Proximity Analysis,'' February 2011, a copy of which is available in the docket.

$PM_{2.5}$ (co-benefits) analysis. As mentioned above, many of the steps EGUs take to reduce their emissions of air toxics as required by this final rule will also reduce emissions of

PM and SO2. As a result, this final rule will reduce concentrations of $PM_{2.5}$ in the atmosphere. Exposure to $PM_{2.5}$ can cause or contribute to adverse health effects, such as asthma and heart disease, that significantly affect many minority, low-income, and tribal individuals and their communities. Fine PM ($PM_{2.5}$) is particularly (but not exclusively) harmful to children, the elderly, and people with existing heart and lung diseases, including asthma. Exposure can cause premature death and trigger heart attacks, asthma attacks in children and adults with asthma, chronic and acute bronchitis, and emergency room visits and hospitalizations, as well as milder illnesses that keep children home from school and adults home from work. Missing work due to illness or the illness of a child is a particular problem for people who have jobs that do not provide paid sick days. Low-wage employees also risk losing their jobs if they are absent too often, even if it is due to their own illness or the illness of a child or other relative. Finally, many individuals in these communities lack access to high quality health care to treat these types of illnesses. Due to all these factors, many minority and low-income communities are particularly susceptible to the health effects of $PM_{2.5}$ and receive a variety of benefits from reducing it.

We estimate that in 2016 the annual PM related benefits of the final rule for adults include approximately 4,200 to 11,000 fewer premature mortalities, 2,800 fewer cases of chronic bronchitis, 4,800 fewer non-fatal heart attacks, 2,600 fewer hospitalizations (for respiratory and cardiovascular disease combined), 3.2 million fewer days of restricted activity due to respiratory illness and approximately 540,000 fewer lost work days. We also estimate substantial health improvements for children in the form of 130,000 fewer asthma attacks, 3,100 fewer emergency room visits due to asthma, 6,300 fewer cases of acute bronchitis, and approximately 140,000 fewer cases of upper and lower respiratory illness.

We also examined the level of $PM_{2.5}$ mortality risks prior to the implementation of the rule according to race, income, and educational attainment. We then estimated the change in $PM_{2.5}$ mortality risk as a result of this final rule among people living in the counties with the highest (top 5 percent) $PM_{2.5}$ mortality risk in 2005. We then compared the change in risk among the people living in these ‘‘high-risk’’ counties with people living in all other counties.

In 2005, people living in the highest risk counties and in the poorest counties were estimated to be at substantially higher risk of $PM_{2.5}$- related death than people living in the other 95 percent of counties. This was true regardless of race; the difference between the groups of counties for each race was large while the differences among races in both groups of counties were very small. In contrast, the analysis found that people with less than high school education were predicted to have significantly greater risk from $PM_{2.5}$ mortality than people with a greater than high school education. This was true both for the highest-risk counties and

for the other counties. In summary, the analysis indicates that in 2005, educational status, living in one of the poorest counties, and living in a high-risk county are associated with higher estimated $PM_{2.5}$ mortality risk while race is not.

Our analysis predicts that this final rule will likely significantly reduce the risk of $PM_{2.5}$-related premature mortality among all populations of different races living throughout the U.S. compared to both 2005 and 2016 pre-rule (i.e., base case) levels. The analysis indicates that people living in counties with the highest rates (top 5 percent) of $PM_{2.5}$ mortality risk in 2005 receive the largest reduction in mortality risk after this rule takes effect. We also estimate that people living in the poorest 5 percent of the counties will experience a larger reduction in $PM_{2.5}$ mortality risk when compared to all other counties. More information can be found below in section 7.11.3.

The EPA estimates that the benefits of the final rule are likely distributed among races, income levels, and levels of education fairly evenly, although there is insufficient data to generate different concentration response functions for each demographic group. However, the analysis does indicate that this final rule in conjunction with the implementation of existing or final rules (e.g., the Cross-State Air Pollution Rule) may help reduce the disparity in risk between those in the highest-risk counties and the other 95 percent of counties for all races and educational levels.

Table 7-5. Comparative Summary of the Demographics within 5 Kilometers (3 Miles) of the Affected Sources (population in millions)[a]

	Population	White	African American	Native American	Other or Multiracial	Minority[b]	Hispanic or Latino[c]	Age 0–17	Age 65+	No High School Diploma	Below Poverty Line
Near source total (3 mi)	13.9	8.78	2.51	0.10	2.52	5.13	2.86	3.37	1.65	2.20	2.43
% of near source total		63%	18%	1%	18%	37%	21%	24%	12%	16%	17%
National total	285	215	35.0	2.49	33.3	70.8	39.1	77.4	35.4	36.7	37.1
% of national total		75%	12%	1%	12%	25%	14%	27%	12%	13%	13%

Sources: The demographics are from the U.S. Census Bureau, 2000. Information on the facilities is from U.S. EPA.

a Racial and ethnic categories overlap and cannot be summed.

b The "Minority" population is the overall population (in the first row) minus white population (in the second row).

c The Census Bureau defines "Hispanic or Latino" as an ethnicity rather than a racial category, Hispanics or Latinos may belong to any race.

7.11.1.3 Meaningful Public Participation

The EPA defines ‘‘environmental justice’’ to include meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies. To promote meaningful involvement, the EPA publicized the rulemaking via newsletters, EJ listserves, and the internet, including the Office of Policy's (OP) Rulemaking Gateway Web site (http://yosemite.epa.gov/opei/RuleGate.nsf/). During the comment period, the EPA discussed the proposed rule via a conference call with communities, conducted a community-oriented webinar on the proposed rule, and posted the webinar presentation on- line. The EPA also held three public hearings to receive additional input on the proposal.

Once this rule is finalized, affected EGUs will need to update their Title V operating permits to reflect their new emission limits, any other new applicable requirements, and the associated monitoring and recordkeeping from this rule. The Title V permitting process provides that when most permits are reopened (for example, to incorporate new applicable requirements) or renewed, there must be opportunity for public review and comments. In addition, after the public review process, the EPA has an opportunity to review the proposed permit and object to its issuance if it does not meet CAA requirements.

7.11.1.4 Summary

This final rule strictly limits the emissions rate of Hg and other HAP from every affected EGU. The EPA's analysis indicates substantial health benefits, including for vulnerable populations, from reductions in $PM_{2.5}$.

The EPA's analysis also indicates reductions in risks for individuals, including for members of minority populations, who eat fish frequently from U.S. lakes and rivers and who live near affected sources. Based on all the available information, the EPA has determined that this final rule will not have disproportionately high and adverse human health or environmental effects on minority, low income, or indigenous populations. The EPA is providing multiple opportunities for EJ communities to both learn about and comment on this rule and welcomes their participation.

7.11.2 Analysis of High Risk Sub-Populations

In addition to the previously described assessment of EJ impacts, EPA is providing a qualitative assessment of sub-populations with particularly high potential risks of mercury exposure due to high rates of fish consumption. These populations overlap in many cases with

traditional EJ populations and would benefit from mercury reductions resulting from this rule. This section describes the available information on consumption rates for subpopulations with high fish consumption, and shows their locations in the U.S. Because of their high rates of fish consumption, reductions in mercury occurring in waterbodies where these populations catch fish will have a larger IQ benefit for these populations relative to the general fish consuming population.

Based on a detailed review of the literature, EPA identified several high-risk sub-populations (Moya, 2004; Burger, 2002, Shilling et al., 2010, Dellinger, 2004). The analysis of potentially high-risk groups focuses on six subpopulations:

- low-income African-American recreational/subsistence fishers in the Southeast region[5]
- low-income white recreational/subsistence fishers in the Southeast region
- low-income female recreational/subsistence fishers
- Hispanic subsistence fishers
- Laotian subsistence fishers
- Chippewa/Ojibwe Tribe members in the Great Lakes area

These specific subpopulations were selected based on published empirical evidence of particularly high self-caught freshwater fish consumption rates among these groups. Evidence for the first three groups is based on a study by Burger (2002), which collected survey data from a random sample of participants in the Palmetto Sportsmen's Classic in Columbia, SC. Of 458 respondents, 39 were black, 415 were white, and 149 were female. The sample size for the black population is relatively small, which increases uncertainty, particularly in higher percentile consumption rate values provided for this group. In this study, results are also split out for poor respondents (0–20K$ annual income). These consumption rates are relatively high, particularly for the higher percentiles. This observation forms the basis for our decision to assess a number of the subsistence populations only for watersheds located in US Census tracts containing members of source populations below the poverty line for the white and black populations.

[5]The low-income designation is based on Census 2000 estimates of populations living in poverty. The Southeast for purposes of this analysis comprises Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, Virginia, and West Virginia.

Evidence for the Hispanic and Laotian groups is based on a study by Shilling et al. (2010). This study looks at subsistence fishing activity among ethnic groups associated with more urbanized areas near the Sacramento and San Joaquin rivers in the Central Valley in CA. The authors note that many of these ethnic groups relied on fishing in origin countries and bring that practice here (e.g., Cambodian, Vietnamese and Mexican). The authors also note that fish consumption rates reported here for specific ethnic groups (specifically Southeast Asian) are generally in-line with rates seen in WA and OR studies. For the Chippewa population, we use results from a study by Dellinger (2004), which gathered data on self-reported fish consumption rates by Tribes in the Great Lakes area. Because fishing activity is highly variable across Tribes (and closely associated with heritage cultural practices) we have not extrapolated fishing behavior outside of the areas ceded to the Tribes covered in the study (regions in the vicinity of the Great Lakes). The terms "subsistence" and "recreational" fishing are based on the terminology used in these published studies to describe the population of interest. In general, subsistence fishers are individuals whose primary objective in fishing is to acquire food for household consumption. For recreational fishers, the primary objective is to enjoy the outdoor activity; however, fish consumption is also often an objective.

Table 7-6. Reported Distributions of Self-Caught Freshwater Fish Consumption Rates Among Selected Potentially High-Risk Subpopulations

Population	Self-Caught Freshwater Fish Consumption Rate (g/day)			Study
	Sample Size	Mean (Median)	90th (95th) Percentile	
Low-income African-American recreational/subsistence fishers in Southeast	39	171 (137)	446 (557)	Burger (2002)
Low-income white recreational/ subsistence fishers in Southeast	415	38.8 (15.3)	93 (129)	Burger (2002)
Low-income female recreational/ subsistence fishers	149	39.1 (11.6)	123 (173)	Burger (2002)
Hispanic subsistence fishers	45	25.8 (19.1)	98[a] (155.9)	Shilling et al. (2010)
Laotian subsistence fishers	54	47.2 (17)	144.8[a] (265.8)	Shilling et al. (2010)
Great Lakes tribal groups	822	60 (113[b])	136.2[a] (213.1)[a]	Dellinger (2004)

[a] Derived values using a log-normal distribution, based on the median and the 95th percentile or standard deviation reported in study.

[b] Standard deviation in parentheses, rather than median.

Using county-level growth projections, there were an estimated 3.09 million low-income African Americans in census tracts that have (1) at least one HUC-12 within 20 miles with a mercury fish tissue concentration estimate and (2) at least 25 African-American inhabitants living below the poverty level, and 3.56 million are projected to reside in these areas in 2016. The geographic distribution of the expected 2016 population is shown in Figure 7-1. The total low-income (below the poverty level) White population in the southeastern states was 3.26 million for 2005 and is projected to be 3.58 million in 2016. The geographic distribution of this population for 2016 is shown in Figure 7-2. The total modeled low-income female population was 18.4 million for 2005 and is projected to be 20.1 million for 2016. The geographic distribution of the population modeled for 2016 is shown in Figure 7-3. The total modeled Hispanic population was 19.6 million for 2005 and is projected to be 27.2 million in 2016. The geographic distribution of the population modeled for 2016 is shown in Figure 7-4. The total modeled Laotian population was 80,000 for 2005 and projected to be 137,500 in 2016. The geographic distribution of the population modeled for 2016 is shown in Figure 7-5. The total modeled Chippewa population used to simulate the distribution of IQ loss was 23,900 for 2005 and is projected to be 29,500 for 2016. The geographic distribution of the population modeled for 2016 is shown in Figure 7-6.

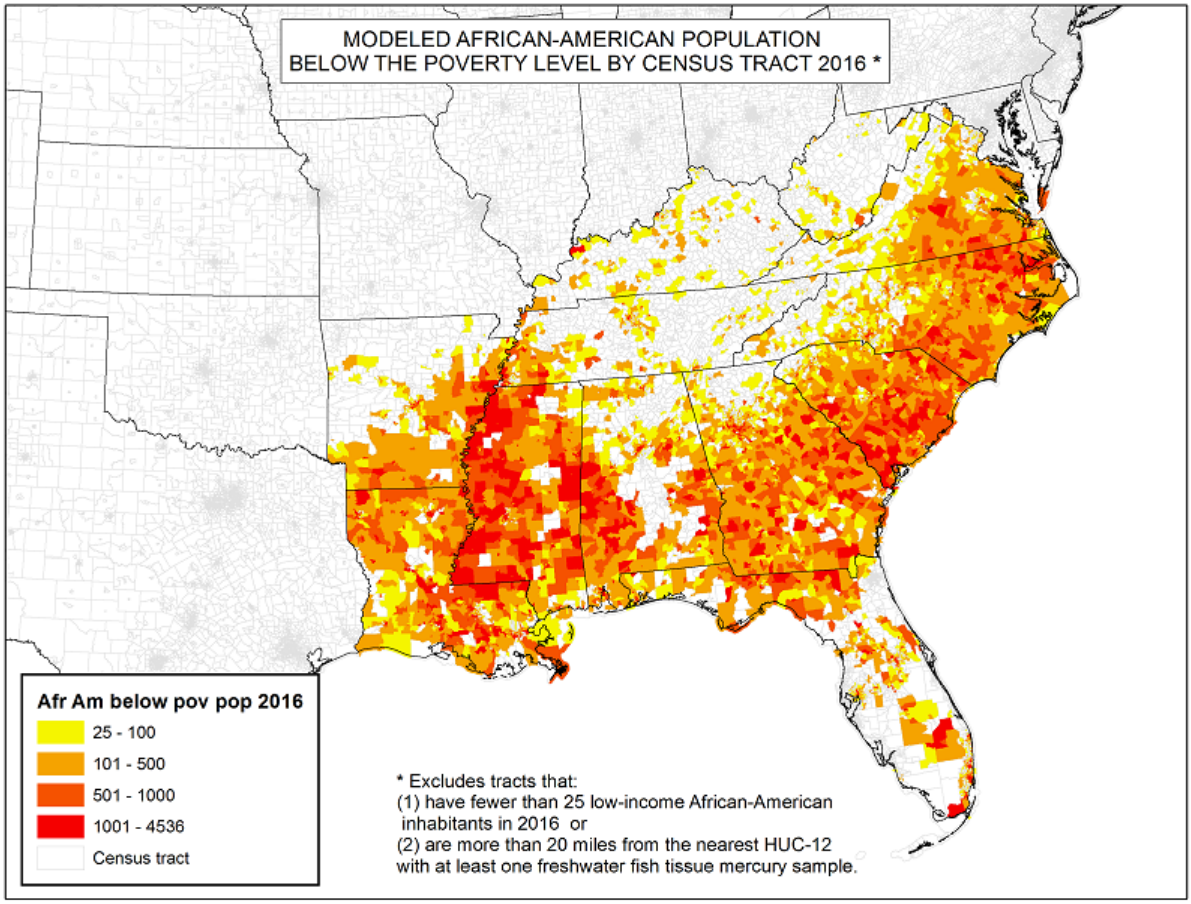

Figure 7-1. Projected African-American Population Below the Poverty Level by Census Tract in the Southeast for 2016

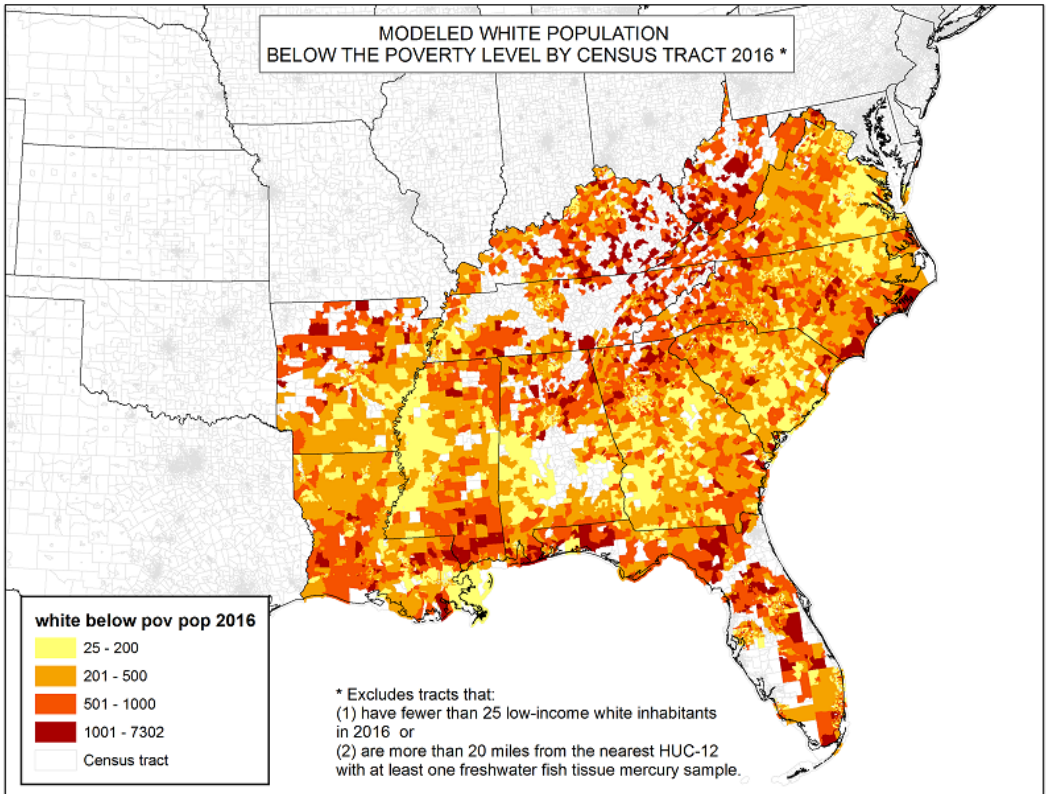

Figure 7-2. Projected White Population Below the Poverty Level by Census Tract in the Southeast for 2016

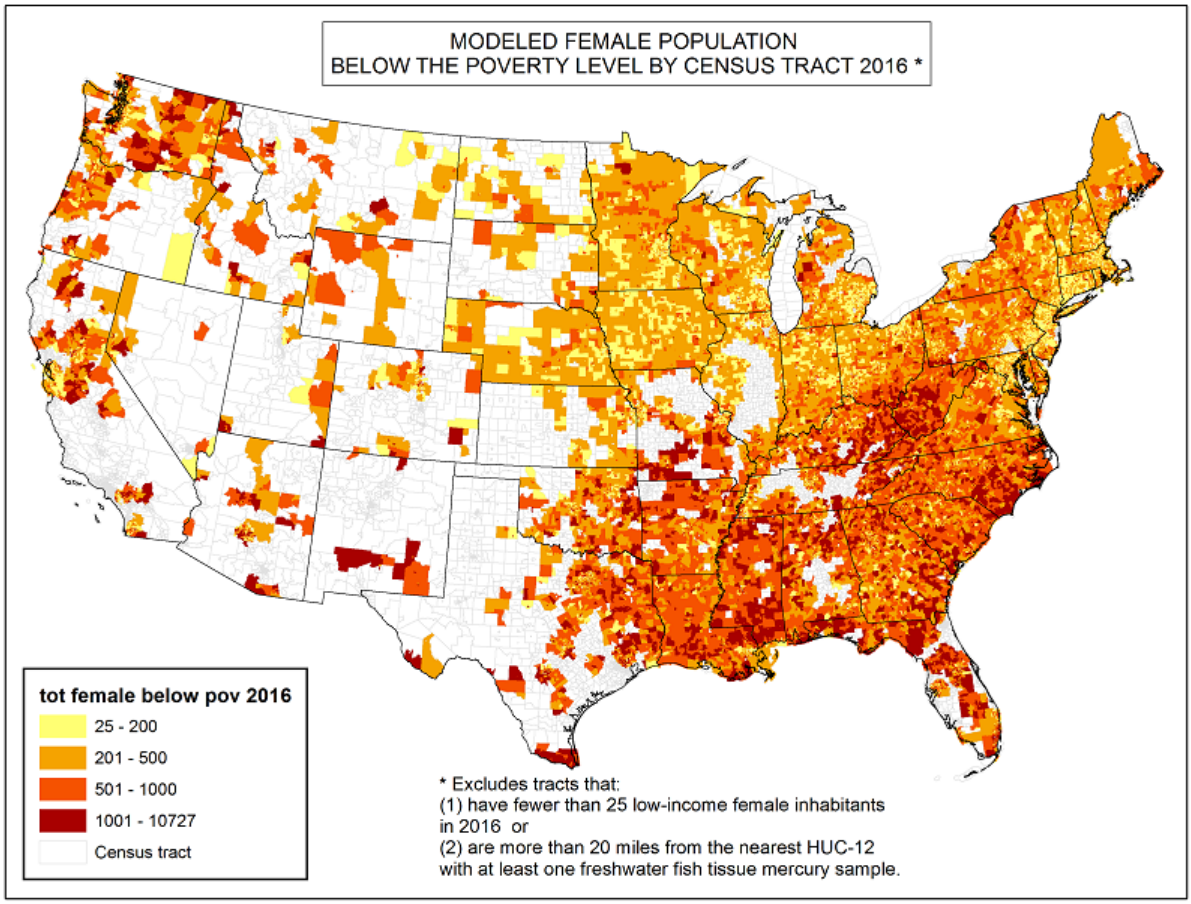

Figure 7-3. Projected Female Population Below the Poverty Level by Census Tract for 2016

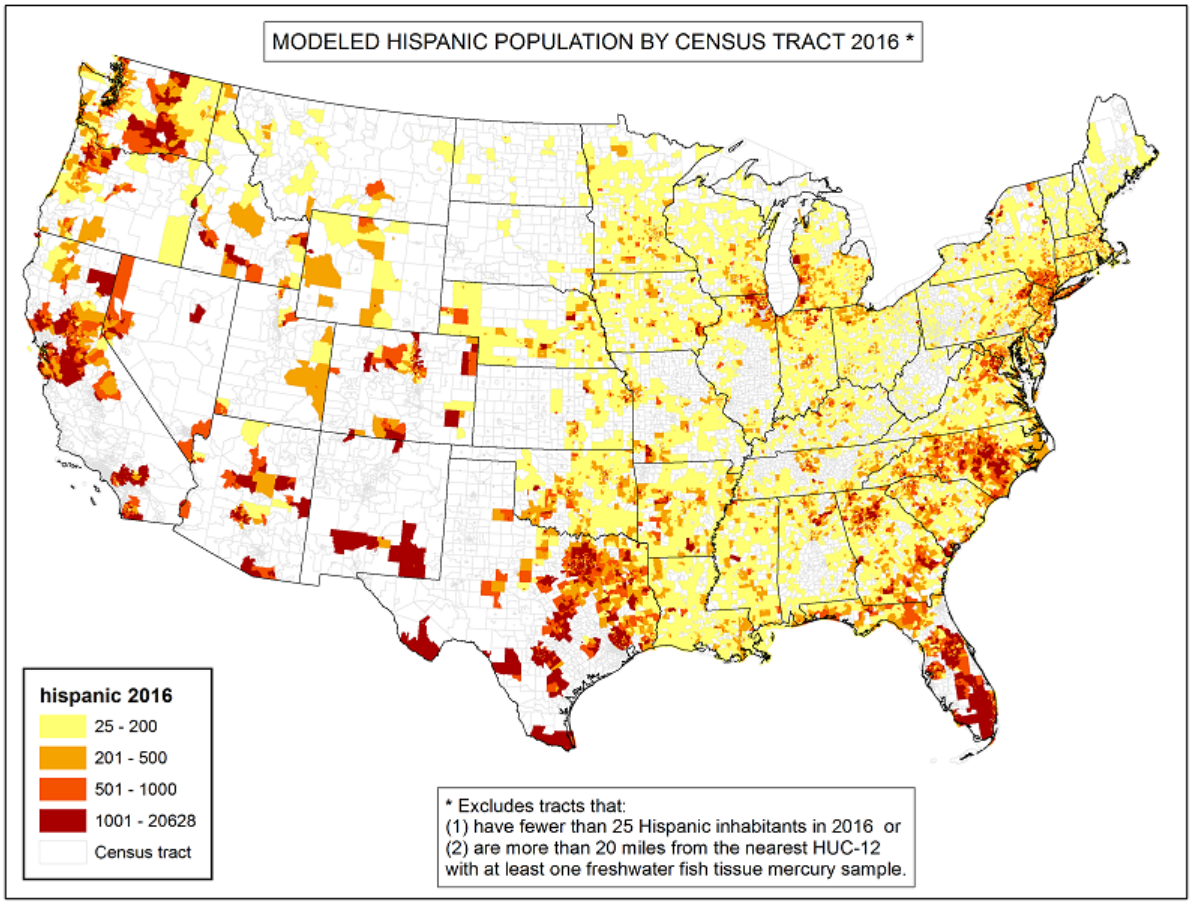

Figure 7-4. Modeled Hispanic Population by Census Tract for 2016

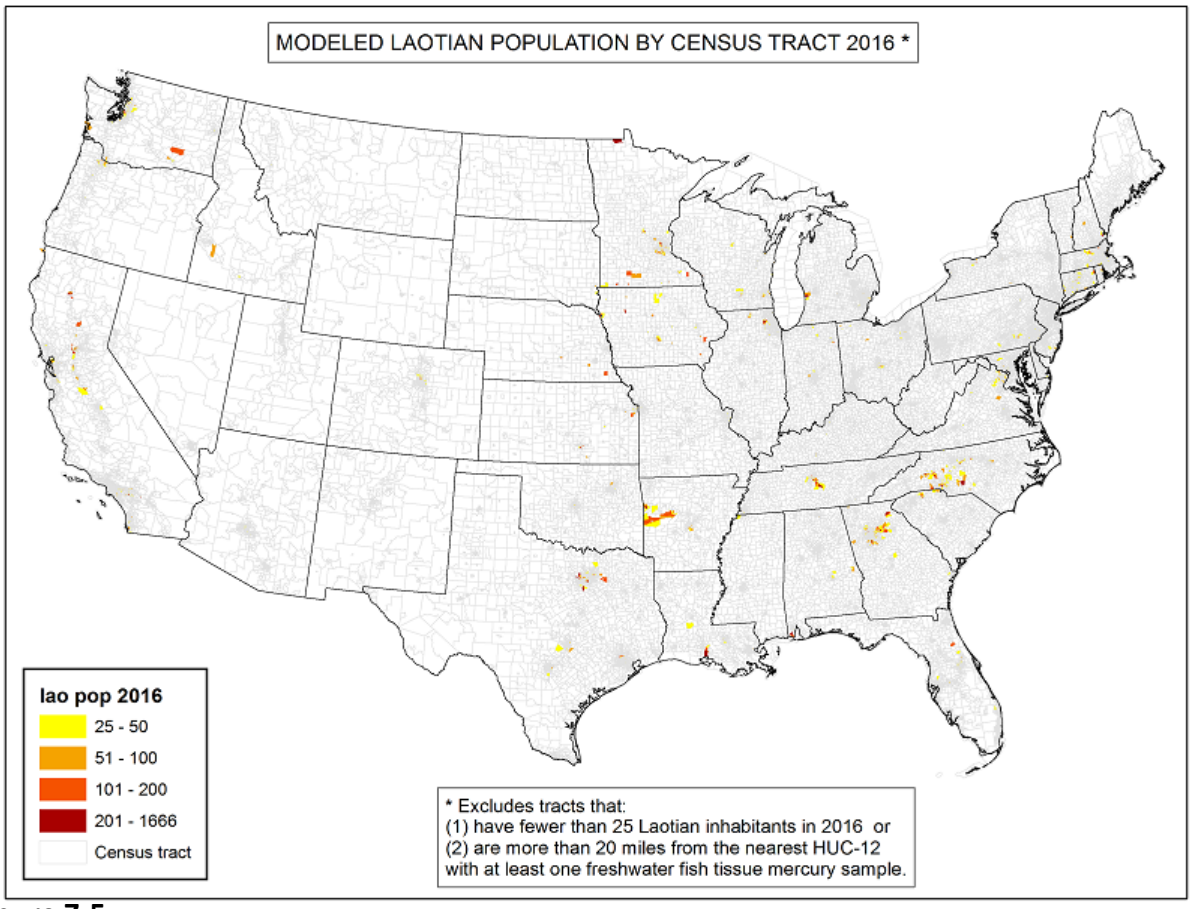
MODELED LAOTIAN POPULATION BY CENSUS TRACT 2016 *
lao pop 2016
25 - 50
51 - 100
101 - 200
201 - 1666
Census tract
* Excludes tracts that:
(1) have fewer than 25 Laotian inhabitants in 2016 or
(2) are more than 20 miles from the nearest HUC-12
with at least one freshwater fish tissue mercury sample.

Figure 7-5.

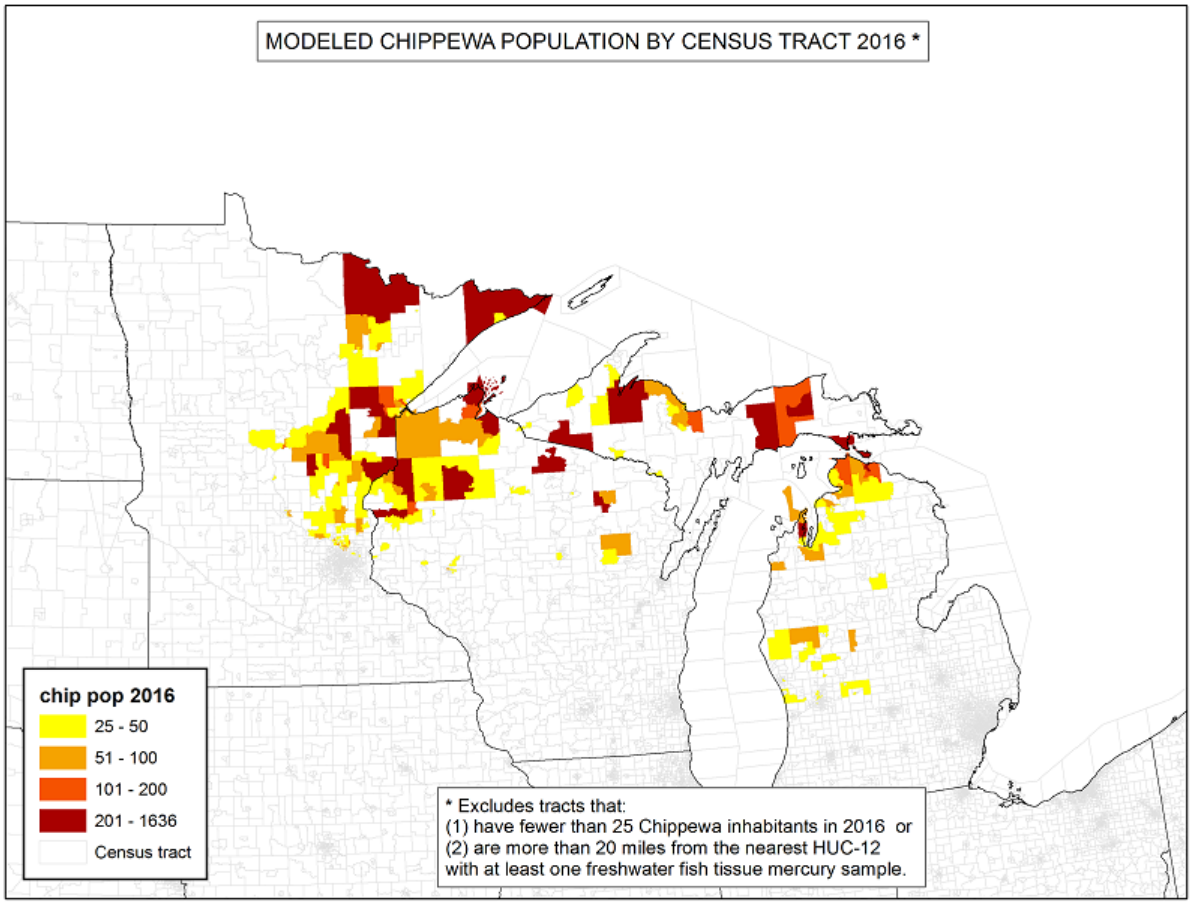

Figure 7-6. Modeled Chippewa Population by Census Tract in the Great Lakes Area for 2016.

7.11.3 Characterizing the Distribution of Health Impacts across Populations

EPA is developing new approaches and metrics to improve its characterization of the impacts of EPA rules on different populations. This analysis reflects one such approach, which explores two principal questions regarding the distribution of $PM_{2.5}$-related benefits resulting from the implementation of MATS:

1. What is the baseline distribution of $PM_{2.5}$-related mortality risk for adults according to the race, income and education of the population?

2. How does MATS change the distribution of $PM_{2.5}$ mortality risk among populations of different races—particularly among those populations at greatest risk in the baseline?[6]

In this analysis we estimated that $PM_{2.5}$ mortality risk from the modeled scenarios is not distributed equally throughout the U.S., or among populations of different levels of educational attainment—though the level of $PM_{2.5}$ mortality risk appears to be shared fairly equally among populations of different races. We estimate that the air quality and $PM_{2.5}$-related mortality risk improvements achieved by MATS are relatively equally distributed among minority populations, and that the rule reduces $PM_{2.5}$ mortality risk the most among those populations at greatest risk in the 2005 baseline we selected for this analysis. We note that while the methods used for this analysis have been employed in recent EPA Regulatory Impact Assessments (EPA, 2011) and are drawn from techniques described in the peer reviewed literature (Fann et al. 2011b) EPA will continue to modify these approaches based on evaluation of the methods.

7.11.3.1 Methodology

The methods used here to describe the distribution of $PM_{2.5}$ mortality impacts are consistent with the approach used in the proposed MATS RIA (U.S. EPA, 2011a) and the final CSAPR RIA (U.S. EPA, 2011b). As a first step, we estimate the level of $PM_{2.5}$-related mortality risk in each county in the continental U.S. based on 2005 air quality levels, which provides a baseline distribution of risk which we use to identify populations with initial higher and lower baseline $PM_{2.5}$-related mortality risk. This portion of the analysis follows an approach described elsewhere (Fann et al. 2011a, Fann et al. 2011b), wherein modeled 2005 $PM_{2.5}$ levels are used to calculate the proportion of all-cause mortality risk attributable to total $PM_{2.5}$ levels in each county in the Continental U.S. Within each county we estimate the level of all-cause $PM_{2.5}$ mortality risks for adult populations as well as the level of $PM_{2.5}$ mortality risk according to the race, income and educational attainment of the population.

Our approach to calculating the distribution of $PM_{2.5}$ mortality risk across the population is generally consistent with the benefits analysis conducted for the modeled scenario described in Appendix 5C with two exceptions: the $PM_{2.5}$ mortality risk coefficients used to quantify impacts and the baseline mortality rates used to calculate mortality impacts (a detailed discussion of how both the mortality risk coefficients and baseline incidence rates are used to estimate the incidence of $PM_{2.5}$-related deaths may be found in the Chapter 5 of the RIA). We

[6] In this analysis we assess the change in risk among populations of different race, income and educational attainment. As we discuss further in the methodology, we consider this last variable because of the availability of education-modified $PM_{2.5}$ mortality risk estimates.

substitute risk estimates drawn from the Krewski et al. (2009) extended analysis of the ACS cohort. In particular, we applied the all-cause mortality risk estimate random effects Cox model that controls for 44 individual and 7 ecological covariates, using average exposure levels for 1999-2000 over 116 U.S. cities (Krewski et al. 2009) (RR=1.06, 95% confidence intervals 1.04—1.08 per 10μg/m^3 increase in $PM_{2.5}$). This mean relative risk estimate is identical to the Pope et al. (2002) risk estimate applied for the benefits analysis (though the standard error around the mean RR estimate is slightly narrower).

Within both this and other analyses of the ACS cohort (see Krewski et al. 2000), educational attainment has been found to be inversely related to the risk of all-cause mortality. That is, populations with lower levels of education (in particular, < grade 12) are more vulnerable to $PM_{2.5}$-related mortality. Krewski and colleagues note that "…the level of education attainment may likely indicate the effects of complex and multifactorial socioeconomic processes on mortality…," factors that we would like to account for in this EJ assessment. When estimating PM mortality impacts among populations according to level of education, we applied $PM_{2.5}$ mortality risk coefficients modified by educational attainment: less than grade 12 (RR = 1.082, 95% confidence intervals 1.024—1.144 per 10 μg/m^3 change), grade 12 (RR = 1.072, 95% confidence intervals 1.020—1.127 per 10 μg/m^3 change), and greater than grade 12 (RR = 1.055, 95% confidence intervals 1.018—1.094 per 10 μg/m^3 change). The Pope et al. (2002) study does not provide education-stratified RR estimates. The principal reason we applied risk estimates from the Krewski et al. (2009) study was to ensure that the risk coefficients used to estimate all-cause mortality risk and education-modified mortality risk were drawn from a consistent modeling framework.

The other key difference between this distributional analysis and the benefits analysis for this rule relates to the baseline mortality rates. As described in Chapter 5 of this RIA, we calculate $PM_{2.5}$-related mortality risk relative to baseline mortality rates in each county. Traditionally, for benefits analysis, we have applied county-level age- and sex-stratified baseline mortality rates when calculating mortality impacts (Abt, 2010). To calculate $PM_{2.5}$ impacts by race, we incorporated race-specific (stratified by White/Black/Asian/Native American) baseline mortality rates. This approach improves our ability to characterize the relationship between race and $PM_{2.5}$-related mortality however, we do not have a differential concentration-response function as we do for education, and as a result, we are not able to capture the full impacts of race in modifying the benefits associated with reductions in PM2.5

The result of this analysis is a distribution of $PM_{2.5}$ mortality risk estimates by county, stratified by each of the three population variables (race, income and educational attainment).

We have less confidence in county-level estimates of mortality than the national or even state estimates, however, the modeling down to the county level can be considered reasonable because the estimates are based on 12km air quality modeling estimates of $PM_{2.5}$, county level baseline mortality rates, and a concentration-response function that is derived from county level data. We next identified the counties at or above the median and upper 95th percentile of the $PM_{2.5}$ mortality risk distribution. We selected this percentile cut-off to capture the very highest levels of $PM_{2.5}$ mortality risk. The second step of the analysis was to repeat the sequence above by estimating $PM_{2.5}$ mortality risk in 2016 prior to, and after, the implementation of MATS.

7.11.3.2 Results

We estimated the change in $PM_{2.5}$ mortality risk in 2016 among populations living in those counties at the upper 95th percentile of the mortality risk in 2005. We then compared the change in risk among these populations living in high-risk counties with populations living in all other counties (Tables 7-17 through 7-9).

Table 7-17. Estimated Change in the Percentage of All Deaths Attributable to $PM_{2.5}$ Before and After Implementation of MATS by 2016 for Each Populations, Stratified by Race

	Race			
Year	Asian	Black	Native American	White
Among populations at greater risk				
2016 (pre-MATS Rule)	4.3%	4.4%	4.4%	4.5%
2016 (post-MATS Rule)	4.1%	4.1%	4.2%	4.3%
Among all other populations				
2016 (pre-MATS Rule)	3.2%	3.1%	3.1%	3.3%
2016 (post-MATS Rule)	3%	2.9%	2.9%	3.1%

Table 7-8. Estimated Change in the Percentage of All Deaths Attributable to $PM_{2.5}$ Before and After Implementation of MATS by 2016 for Each Population, Stratified by Race and Poverty Level

	Race			
Year	Asian	Black	Native American	White
Among populations living in counties with the largest number of individuals living below the poverty line				
2016 (pre-MATS)	3.6%	3.5%	3.6%	3.6%
2016 (post-MATS)	3%	3.4%	3%	3.5%
Among all other populations				
2016 (pre-MATS)	3.2%	3.2%	3.2%	3.3%
2016 (post-MATS)	3%	2.9%	3%	3.1%

Table 7-9. Estimated Change in the Percentage of All Deaths Attributable to $PM_{2.5}$ Before and After the Implementation of MATS by 2016 for Each Population, Stratified by Educational Attainment

	Race		
Year	< Grade 12	= Grade 12	> Grade 12
Among populations at greater risk			
2016 (pre-MATS)	6.2%	5.5%	4.3%
2016 (post-MATS)	5.9%	5.3%	4.1%
Among all other populations			
2016 (pre-MATS)	4.5%	4%	3.1%
2016 (post-MATS)	4.2%	3.8%	2.9%

Table 7-7, shows the estimated level of $PM_{2.5}$ mortality risk among populations of different races according to whether those populations live in counties identified as "greater risk" counties or "all other counties." As described above, we define "greater risk" counties as those at or above the 95^{th} percentile of the estimated $PM_{2.5}$ mortality risk in 2005, and "all other counties" as those with estimated $PM_{2.5}$ mortality risk below this level. The results of this

analysis suggest that the $PM_{2.5}$ mortality risk among these populations at "greater risk" falls with implementation of the 2016 MATS. These results also suggest that all populations, irrespective of race, may receive an estimated reduction in $PM_{2.5}$ mortality risk. However, limits to data resolution prevent us from delineating the $PM_{2.5}$ mortality risk according to population race with confidence.

Table 7-8 illustrates the estimated change in the level of $PM_{2.5}$ mortality risk among populations living in those counties that meet two criteria: (1) the county is at the upper 95th percentile of mortality risk in 2005; (2) the county is at the upper 95th percentile in terms of the number of individuals living below the poverty line. We also estimate the change in $PM_{2.5}$ risk among all other counties. The analysis suggests that people living in the highest mortality risk and poorest counties may experience a larger improvement in $PM_{2.5}$ mortality risk than those living in lower risk counties containing a smaller number of individuals living below the poverty line.

Table 7-9 summarizes the estimated change in $PM_{2.5}$ mortality risk among populations who have attained three alternate levels of education—less than high school, high school and greater than high school. As described above, we apply education-stratified $PM_{2.5}$ mortality risk coefficients for this analysis. These results indicate that populations with less than a high school education are at higher risk of $PM_{2.5}$ mortality, irrespective of whether these populations live in "greater risk" counties, according to the definition described above. We estimate that with the implementation of MATS, all populations see their $PM_{2.5}$ mortality risk fall, regardless of educational attainment.

7.12 Congressional Review Act

The Congressional Review Act, 5 U.S.C. 801 et seq., as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of the rule in the Federal Register. A major rule cannot take effect until 60 days after it is published in the Federal Register. This action is a ''major rule'' as defined by 5 U.S.C. 804(2). This rule will be effective 60 days after publication.

7.13 References

Abt Associates, Inc. 2010. Environmental Benefits and Mapping Program (Version 4.0). Bethesda, MD. Prepared for U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at <http://www.epa.gov/air/benmap>.

Bullard RD, Mohai P, Wright B, Saha R, et al. Toxic Waste and Race at Twenty 1987-2007. United Church of Christ. March, 2007.

Burger, J. (2002). Daily consumption of wild fish and game: Exposures of high end recreationalists, *International Journal of Environmental Health Research*, 12:4, p. 343-354.

Dellinger, JA (2004). Exposure assessment and initial intervention regarding fish consumption of tribal members in the Upper Great Lakes Region in the United States. *Environmental Research* 95 (2004) p. 325-340.

Fann N, Lamson AD, Anenberg SC, Wesson K, Risley D, Hubbell B. 2011a. Estimating the national public health burden associated with exposure to ambient $PM_{2.5}$ and ozone. Risk Analysis, in press.

Fann N, Roman HA, Fulcher CM, Gentile MA, Hubbell BJ, Wesson K, et al. 2011b. Maximizing Health Benefits and Minimizing Inequality: Incorporating Local-Scale Data in the Design and Evaluation of Air Quality Policies. Risk Analysis 31:908-922; doi:10.1111/j.1539-6924.2011.01629.x.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi, Y, et al. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. HEI Research Report, 140, Health Effects Institute, Boston, MA.

Krewski, D., R.T. Burnett, M.S. Goldbert, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz, and W.H. White. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute. Cambridge MA. July.

Mennis J. "Using Geographic Information Systems to Create and Analyze Statistical Surfaces of Populations and Risk for Environmental Justice Analysis." Social Science Quarterly, 2002;83(1):281-297.

Mohai P, Saha R. "Reassessing Racial and Socio-economic Disparities in Environmental Justice Research." Demography. 2006;43(2): 383–399.

Moya, J. 2004. Overview of fish consumption rates in the United States. *Human and Ecological Risk Assessment: An International Journal* 10, no. 6: 1195–1211.

Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association 287:1132-1141.

Shilling, Fraser, Aubrey White, Lucas Lippert, Mark Lubell (2010). Contaminated fish consumption in California's Central Valley Delta. *Environmental Research* 110, p. 334-344.

Woods & Poole Economics, Inc. 2008. Population by Single Year of Age CD. CD-ROM. Woods & Poole Economics, Inc.

U.S. GAO (Government Accountability Office). "Demographics of People Living Near Waste Facilities." Washington DC: Government Printing Office; 1995.

U.S. Environmental Protection Agency (U.S. EPA). 2011a. Proposed Regulatory Impact Analysis (RIA) for the Mercury and Air Toxics Rule. Office of Air Quality Planning and Standards, Research Triangle Park, NC. January.

U.S. Environmental Protection Agency (U.S. EPA). 2011b. Final Regulatory Impact Analysis (RIA) for the Transport Rule. Office of Air Quality Planning and Standards, Research Triangle Park, NC. June.

CHAPTER 8
COMPARISON OF BENEFITS AND COSTS

8.1 Comparison of Benefits and Costs

The estimated costs to implement the final MATS Rule, as described earlier in this document, are approximately $9.6 billion annually for 2016 (2007 dollars). Thus, the net benefits (benefits minus costs) of the program in 2016 are approximately $27 to 80 +B billion or $24 to 71 +B billion annually (2007 dollars, based on a discount rate of 3 percent and 7 percent for the benefits, respectively and rounded to two significant figures). (B represents the sum of all unquantified benefits and disbenefits of the regulation.) Therefore, implementation of this rule is expected, based purely on economic efficiency criteria, to provide society with a significant net gain in social welfare, even given the limited set of health and environmental effects we were able to quantify. Addition of health endpoints other than IQ loss to children exposed to mercury from recreationally caught freshwater fish and acidification-, and eutrophication-related impacts would likely increase the net benefits of the rule. Table 8-1 presents a summary of the benefits, costs, and net benefits of the final MATS Rule.

As with any complex analysis of this scope, there are several uncertainties inherent in the final estimate of benefits and costs that are described fully in Chapters 3, 4 and 5.

Table 8-1. Summary of EPA's Estimates of Annualized[a] Benefits, Costs, and Net Benefits of the Final MATS in 2016[b] (billions of 2007$)

Description	Estimate (3% Discount Rate)	Estimate (7% Discount Rate)
Costs[c]	$9.6	$9.6
Benefits[d,e,f]	$37 to $90 + B	$33 to $81 + B
Net benefits (benefits-costs)[g]	$27 to $80 + B	$24 to $71 + B

[a] All estimates presented in this report represent annualized estimates of the benefits and costs of the final MATS in 2016 rather than the net present value of a stream of benefits and costs in these particular years of analysis.

[b] Estimates rounded to two significant figures and represent annualized benefits and costs anticipated for the year 2016.

[c] Total social costs are approximated by the compliance costs. Compliance costs consist of IPM projections, monitoring/reporting/record-keeping costs, and oil-fired fleet analysis costs. For a complete discussion of these costs refer to Chapter 3. Costs are estimated using a 6.15% discount rate

[d] Total benefits are composed primarily of monetized PM-related health benefits. The reduction in premature fatalities each year accounts for over 90% of total monetized benefits. Benefits in this table are nationwide and are associated with directly emitted $PM_{2.5}$ and SO_2 reductions. The estimate of social benefits also includes CO_2-related benefits calculated using the social cost of carbon, discussed further in chapter 5.

[e] Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Data limitations prevented us from quantifying these endpoints, and as such, these benefits are inherently more uncertain than those benefits that we were able to quantify. Estimates here are subject to uncertainties discussed further in the body of the document. Potential benefit categories that have not been quantified and monetized are listed in Table ES-4.

[f] Mortality risk valuation assumes discounting over the SAB-recommended 20-year segmented lag structure. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

[g] Net benefits are rounded to two significant figures. Columnar totals may not sum due to rounding

8.2 References

Laden, F., J. Schwartz, F.E. Speizer, and D.W. Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality. American Journal of Respiratory and Critical Care Medicine 173:667-672.

Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.

U.S. Environmental Protection Agency (EPA). December 2010. *Guidelines for Preparing Economic Analyses.* EPA 240-R-10-001.

U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance to Federal Agencies on Preparation of Regulatory Analysis.

Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.

United States Environmental Protection Agency

Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

Publication No. EPA-452/R-11-011
[December 2011]